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# TAYLOR'S PRACTICE OF MEDICINE

THIRTEENTH EDITION

BY


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*WITH 45 PLATES (8 COLOURED) AND 93 TEXT-FIGURES*

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# PREFACE TO THE THIRTEENTH EDITION

THE kind way in which the twelfth edition of Taylor's "Practice of Medicine" has been received has made it necessary to bring out another edition in a little over three years. My thanks are due to Dr. C. P. Symonds and Dr. H. W. Barber, who have again made themselves responsible for the sections on Nervous Diseases and Diseases of the Skin. Alterations and additions have been made all through the book, and some new figures have been introduced into the text.

The following may be mentioned among the new articles: Diseases of the Gonads, Pluriglandular Syndromes, Melioidosis, Tularæmia, Ectoscopy, Subphrenic Abscess, Chronic Manganese Poisoning, Thrombo-angiitis Obliterans, Mönckeberg's Degeneration, Price Jones' Blood Curves, Relation of Body Weight to Body Length, Nomogram relating Age, Weight and Basal Metabolism, Ætiology of Malignant [illegible]plasms (in Appendix), Lipiodol Injections and Peroneal Muscular Atrophy. The following, among other articles, have been added to or more or less completely rewritten: Anaphylaxis, Diagnosis of Small Pox, Treatment of Syphilis, Leprosy, Pathology of Plague, Asthma, Chronic Dyspepsia, Peptic Ulcer, Gastroptosis, Diarrhœa, Jaundice, Congenital Malformations of the Heart, Examination of Heart by X-rays, Treatment of Chronic Heart Disease, Angina Pectoris, Life Histories of Various Worms, Filariasis, Pernicious Anæmia, Functions of the Spleen, Purpura, Aplastic Anæmia, Diabetes Mellitus, Tetany, Status Thymico-lymphaticus, Examination of the Cerebro-spinal Fluid, Neuritis, Compression of the Spinal Cord, with the Differential Diagnosis and Treatment of Transverse Lesions, Cerebral Diplegia, Epilepsy, Migraine and the Psychoneuroses. The term "neuralgia" has been eliminated from the section on Nervous Diseases (except in the case of Trigeminal Neuralgia), and the conditions formerly described under this head have been transferred to the sections on Neuritis and Fibrositis.

In the section on Diseases of the Skin, which has been largely rewritten, the new classification adopted is based on that employed by Dr. Darier in his *Précis de Dermatologie*, and this admirable work has been freely used for reference.

There are three features of the book on which I should like to lay special emphasis. Two of these are new.

In the first place, instead of cumbering up the text with the names of authors in brackets, these have so far as possible been removed and collected together to form a list of references at the end of each section. In a book that is meant to appeal to qualified men as well as to students, as giving in a compact form an up-to-date account of the present state of medical knowledge and practice some such step is obviously desirable. The references have been chosen from two points of view: as providing means of verifying statements in the text about which the reader may be sceptical, and as indicating the publication where a fuller review of the subject with more copious references may be obtained. Hence, no attempt has been made to refer to the earliest work on any subject, and most references are to medical journals, well-known and widely read in this country.

In the second place, at a time when X-ray examination is becoming more and more an essential part of medical diagnosis, it is a practical necessity to supply the student with as complete a series of radiograms as possible. Consequently, the number of plates, showing X-ray appearances, has been increased from nineteen to thirty-six, and in this connection my special thanks are due to Mr. W. Lindsay Locke for his kind help. I am also indebted to Dr. George Graham and Mr. H. P. Winsbury White. Miss H. Chick and the Medical Research Council have kindly allowed me to reproduce radiograms showing the healing of the limb bones in rickets.

In the third place, eight coloured plates have been added. I should like to thank Mr. Foster Moore and Mr. Holmes Spicer for allowing me to use their illustrations of the retina, and Dr. Piney for his beautiful plate showing nucleated red cells. Through the kindness of Dr. Passey material for illustrating two more plates of blood cells has been collected and Mr. Ford has painted them. It is hoped that these plates of blood cells will be of real use to students in diagnosing diseases of the blood, and for this reason both Jenner's and Leishman's stains have been used. The four coloured plates illustrating sixteen of the commoner skin diseases are the work of Mr. Shiells, and his accurate and artistic work will, it is hoped, increase the value of this section. In their preparation assistance was obtained from Jacobi's *Atlas der Hautkrankheiten* and the *Hautkrankheiten und Syphilis im Säuglings und Kindesalter* by Finkelstein, Galewsky and Halberstaedter.

Finally, I should like to thank on behalf of my two colleagues and myself the many kind friends who have helped us in different parts of the book. I can hardly express sufficient thanks to Professor T. Wardrop Griffith for reading through the section on Diseases of the Circulation and for his valuable comments, which will, I hope, bear fruit in making this section clearer than it was before. I owe a like debt to Dr. P. Manson Bahr for similar help in the parts of the book dealing with Tropical Diseases, and to Dr. A. F. Hurst for reading through the section on Nervous Diseases and for contributing some valuable suggestions, and to Mr. T. B. Layton for similar help in Diseases of the Nose and Throat. Other friends whose help I should like to acknowledge are: Professor Eyre and Mr. J. H. Ryffel in the section on Skin Diseases, and Dr. Carey Coombs and Mr. W. W. Payne. Mr. J. Rivers has again very kindly undertaken the preparation of the index.

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# PREFACE TO THE FIRST EDITION

I HAVE attempted in this work to offer a short yet complete account of the present state of medical practice, which may be useful both to students and practitioners. I have devoted most attention to the description of Symptoms, to Diagnosis, to Prognosis, and to Treatment, feeling that they are the divisions of the subject which most answer to the idea of practice. Ætiology and Pathology are also, of course, considered, but the latter could not be so fully dealt with as in works devoted especially to it.

In the arrangement of the diseases, I fear this work may be open to some criticism. Every fresh discovery, every change of opinion as to the pathology of a disease, is likely to call for an alteration in a classification which has essentially a pathological basis. More modern study tends to show that diseases formerly regarded as having a local origin in viscus or joint are of a much more general character. Thus, it is doubtful whether pneumonia, chronic Bright's disease, and gout should not be looked upon as general disorders, rather than as diseases of the lungs, kidneys, and joints respectively. Diabetes mellitus, diabetes insipidus, and hæmoglobinuria, although disorders of the urine, are not due to disease of the kidney; but their true position in classification is still very uncertain, and provisionally they may remain where I have placed them in this book. Similarly, rheumatism and rickets, classified with diseases of bones and joints, are obviously disorders involving a much wider area, but too obscure in their origin to demand a readjustment as yet.

By consulting the most recent works, especially those of Fagge, Strümpell, Payne, Ziegler, Gowers, M. Mackenzie, Douglas Powell, Ralfe, H. Morris, and Crocker, to whom I must express my indebtedness, I have sought to bring this book fully up to the modern state of knowledge. I have not, however, devoted much space to the discussion of theories, finding that the facts of medicine are amply sufficient to fill, and more than fill, a volume such as this, and being convinced that these facts require to be seized and held fast by the beginners in medicine, not only for the sake of diagnosis and treatment, but also for the right estimation of the various theories which are advanced. With a brief statement, therefore, of such views I have in most cases been content.

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# CONTENTS

PAGE

INTRODUCTION . . . . . 1

INFECTIOUS DISEASES . . . . . 9—186

Nature of infection, 9; immunity, 14; anaphylaxis, 17; pyrexia, 18; heat stroke, 26; scarlet fever, 28; measles, 33; rubella, 36; small-pox, 37; vaccination, 42; chicken-pox, 44; mumps, 46; whooping-cough, 47; glandular fever, 49; septicæmia, 50; sapræmia, 51; pyæmia, 51; erysipelas, 52; rheumatic fever, 55; influenza, 61; trench fever, 64; diphtheria, 66; pneumococcal infections, 72; gonococcal infections, 73; typhus fever, 74; typhoid fever, 80; paratyphoid fevers, 92; psittacosis, 93; cerebro-spinal fever, 93; acute poliomyelitis, 97; hydrophobia, 102; tetanus, 104; gas gangrene, 108; syphilis, 109; congenital syphilis, 120; poisoning by arseno-benzol, 123; framboesia, 124; tuberculosis, 125; leprosy, 133; glanders, 136; melioidosis, 137; anthrax, 137; foot-and-mouth disease, 140; actinomycosis, 140; madura foot, 143; sporotrichosis, 143; aspergillosis, 144; rat-bite fever, 144; relapsing fever, 144; sand-fly fever, 148; malarial fevers, 149; black-water fever, 157; trypanosomiasis, 158; leishmaniasis, 160; kala-azar, 161; dysentery, 162; amœbic infection of the liver, 169; Mediterranean fever, 171; spirochætosis ictero-hæmorrhagica, 173; yellow fever, 175; dengue, 178; cholera, 179; plague, 183; tularæmia, 185

DISEASES OF THE ORGANS OF RESPIRATION . . . . . 187—283

EXAMINATION OF THE CHEST . . . . . 187

DISEASES OF THE NASAL PASSAGES . . . . . 196

Acute rhinitis, 196; chronic rhinitis, 198; epistaxis, 198

DISEASES OF THE LARYNX . . . . . 199

Laryngitis, 199; tubercle, 202; syphilis, 204; perichondritis, 205; tumours, 206; foreign bodies, 207; paralysis of laryngeal muscles, 207; spasm of the glottis, 210; laryngismus stridulus, 211; congenital laryngeal stridor, 212; anæsthesia, 212

DISEASES OF THE TRACHEA . . . . . 213

Tracheitis and specific infections, 213; new growths, 213; obstruction, 214

DISEASES OF THE BRONCHI . . . . . 215

Bronchitis, 215; acute bronchitis, 217; chronic bronchitis, 218; fœtid bronchitis, 221; plastic bronchitis, 221; bronchiectasis, 222; hay fever and asthma, 224; obstruction, 229

DISEASES OF THE LUNGS . . . . . 231

Emphysema, 231; collapse, 234; œdema, 236; pneumonia, 237; Friedländer pneumonia, 243; broncho-pneumonia, 243; abscess, 245; chronic pneumonia, 245; gangrene, 247; pulmonary tuberculosis, 248; syphilis, 264; other infections, 265; poisoning by irritant gases, 265; neoplasms, 266; hydatid, 268; pulmonary embolism and thrombosis, 269

DISEASES OF THE PLEURA . . . . . 271

Pleurisy and empyema, 271; hydrothorax, 278; hæmothorax, 278; chylothorax, 279; pneumothorax, 279; diaphragmatic hernia, 282

PAGE

DISEASES OF THE ORGANS OF CIRCULATION . . . . . 284—388

EXAMINATION OF THE HEART . . . . . 285
Capacity for effort, 294 ; X-rays, 295

EXAMINATION OF THE BLOOD VESSELS . . . . . 297
Radial pulse, 297 ; venous pulse, 301

ABNORMALITIES OF CARDIAC ACTION . . . . . 303
Sinus irregularity, 304 ; heart block and Adams-Stokes syndrome, 304 ; premature beats, 307 ; frequent action, 310 ; paroxysmal tachycardia, 310 ; auricular flutter, 312 ; pulsus alternans, 313 ; auricular fibrillation, 313 ; infrequent action, 317 ; ventricular fibrillation, 318

DISEASES OF THE HEART . . . . . 318
Hypertrophy, 318 ; dilatation, 320 ; myocarditis, 322 ; pigmentary degeneration, 323 ; fatty degeneration, 323 ; fibroid degeneration, 324 ; symptoms of myocardial degeneration, 324 ; rupture, 325 ; aneurysm of the heart, 326 ; fatty overgrowth, 326 ; new growths and parasites, 327 ; endocarditis, 328 ; malignant endocarditis, 330 ; chronic endocarditis and diseases of the valves, 334 ; aortic stenosis, 339 ; aortic regurgitation, 339 ; mitral regurgitation, 341 ; mitral stenosis, 342 ; right-sided valvular disease, 343 ; diagnosis, prognosis, and prevention of valvular disease, 344 ; treatment of chronic cardiac disease, 347 ; effort syndrome, 350 ; congenital malformations, 353 ; pericarditis, 355 ; adherent pericardium, 359 ; hydropericardium, 360 ; pneumopericardium, 360 ; hæmopericardium, 361 ; angina pectoris, 361

DISEASES OF THE BLOOD VESSELS . . . . . 365
Arteritis, 365 ; arterio-sclerosis, 367 ; high arterial pressure, 369 ; low arterial pressure, 371 ; aneurysm, 371 ; congenital coarctation of the aorta, 377 ; phlebitis, 378 ; thrombosis and embolism, 378 ; Raynaud's disease, 381 ; intermittent claudication, 382 ; erythromelalgia, 383 ; angeio-neurotic œdema, 383

DISEASES OF THE MEDIASTINUM . . . . . 384
Mediastinitis, 384 ; tuberculosis of the bronchial glands, 385 ; mediastinal new growths, 386

DISEASES OF THE ORGANS OF DIGESTION . . . . . 389—518

EXAMINATION OF THE ABDOMEN . . . . . 389

DISEASES OF THE MOUTH, TONSILS, AND PHARYNX . . . . . 392
Stomatitis, 392 ; oral sepsis, 392 ; Ludwig's angina, 395 ; catarrhal sore throat, 395 ; tonsillitis, 395 ; peritonsillar abscess, 396 ; Vincent's angina, 397 ; chronic enlargement of the tonsils, 397 ; new growths, 398 ; lingual tonsils, 398 ; pharyngeal tonsils, 399 ; chronic pharyngitis, 399 ; retropharyngeal abscess, 400 ; parotitis, 400 ; chronic enlargements of salivary glands, 400

DISEASES OF THE ŒSOPHAGUS . . . . . 401
Œsophagitis, 401 ; obstruction, 401 ; achalasia, 403 ; diverticula, 404

DISEASES OF THE STOMACH AND DUODENUM . . . . . 404
Examination of the stomach, 404 ; X-rays, 405 ; test meals, 406 ; dyspepsia, 408 ; ærophagy, 415 ; gastroptosis, 416 ; hysterical vomiting, 418 ; cyclical vomiting, 418 ; achylia gastrica, 418 ; acute dilatation of the stomach and duodenum, 419 ; acute gastritis, 420 ; peptic ulcer, 421 ; gastro-jejunal and jejunal ulcers, 428 ; chronic dilatation, 428 ; hour-glass contraction, 429 ; congenital hypertrophic stenosis, 430 ; carcinoma, 430 ; benign tumours, 434

PAGE

DISEASES OF THE ORGANS OF DIGESTION—*continued.*

DISEASES OF THE INTESTINE . . . . . 434

Constipation, 434; alimentary toxæmia, 438; diarrhœa, 439; hæmorrhage, 440; colic, 441; enteritis, 442; food-poisoning, 447; cœliac disease, 448; sprue, 448; phlegmonous enteritis, 449; colitis, 449; appendicitis, 451; tubercle, new growths, syphilis and diverticulitis, 456; obstruction, 457; intussusception, 464; Hirschsprung's disease, 466; intestinal worms, 466

DISEASES OF THE LIVER . . . . . 472

Test of hepatic efficiency, 473; jaundice, 474; ascites, 477; circulatory changes, 479; abscess, 479; catarrhal jaundice, 480; acute yellow atrophy, 481; cirrhosis, 483; fatty liver, 488; lardaceous disease, 489; syphilis, 490; tubercle, 491; new growths, 491; cystic disease, 492; hydatid, 493; suppurative cholangitis, 495; cholecystitis, 496; gall stones, 497; perihepatitis, 501; pylephlebitis, 502

DISEASES OF THE PANCREAS . . . . . 503

Functional tests of the pancreas, 503; acute pancreatitis, 505; chronic pancreatitis, 506; concretions, 507; tumours, 507

DISEASES OF THE PERITONEUM . . . . . 508

Peritonitis, 508; tuberculous peritonitis, 514; polyorrhomenitis, 516; peritoneal adhesions, 516; peritoneal effusions, 516; new growths, 517

DISEASES OF THE BLOOD, SPLEEN, AND LYMPHATIC SYSTEM . 519—557

DISEASES OF THE BLOOD . . . . . 519

Examination of the blood, 519; anæmia, 523; chlorosis, 525; pernicious anæmia, 526; allied forms, septic and aplastic, 529; splenic anæmia, 529; anæmia infantum pseudo-leukæmica, 530; secondary anæmia, 531; acholuric jaundice, 531; leukæmia, 532; chloroma, 537; leucocytosis, 537; leucopenia, 537; polycythæmia rubra, 537; erythræmia, 538; erythrocytosis, 539; hæmoglobinæmia, 539; paroxysmal hæmoglobinuria, 540; methæmoglobinæmia and sulphæmoglobinæmia, 541; purpura, 542; hæmophilia, 544; blood transfusion, 545; reaction of the blood and acidosis, 547

DISEASES OF THE SPLEEN . . . . . 549

Congestion, 550; splenitis, 550; chronic enlargement, 550; infarcts, 550; lardaceous degeneration, 550; tubercle, 551; parasites, 551

DISEASES OF THE LYMPHATIC SYSTEM . . . . . 551

Hodgkin's disease, 551; filariasis, 554; chyluria, 555; lymph scrotum, 556; elephantiasis, 556; other forms, 557

DISEASES OF METABOLISM INCLUDING DISEASES OF THE ENDOCRINE ORGANS . . . . . 558—602

THE BASAL METABOLISM . . . . . 558

DIABETES MELLITUS . . . . . 562

DISEASES OF THE THYROID GLAND . . . . . 576

Goitre, 576; exophthalmic goitre and toxic adenoma, 578; myxœdema, 581; cretinism, 583

DISEASES OF THE PARATHYROID GLANDS . . . . . 584

Tetany, 584

PAGE

DISEASES OF METABOLISM INCLUDING DISEASES OF THE ENDOCRINE ORGANS—*continued.*

DISEASES OF THE THYMUS GLAND . . . . . 586
Status thymico-lymphaticus, 587

DISEASES OF THE SUPRARENAL CAPSULES . . . . . 588
Acute inadequacy, 589; Addison's disease, 589; tumours, 592

DISEASES OF THE PITUITARY GLAND . . . . . 592
Acromegaly, 594; hypophyseal dystrophia adiposo-genitalis, 595; diabetes insipidus, 595

DISEASES OF THE PINEAL GLAND . . . . . 597

DISEASES OF THE GONADS . . . . . 597
Eunuchism, eunuchoidism, hyper- and hypogenitalism, 598

VARIED ENDOCRINOPATHIC SYNDROMES . . . . . 598
Obesity, 598; infantilism, 600; pluri-glandular syndromes, 601

DISEASES OF THE URINARY ORGANS . . . . . 603—662

EXAMINATION OF THE KIDNEY AND BLADDER . . . . . 603

EXAMINATION OF THE URINE . . . . . 603
Quantity, 603; specific gravity, 604; solids, 604; reaction, 609

ESTIMATION OF THE RENAL FUNCTIONS . . . . . 609

DISEASES OF THE KIDNEYS . . . . . 611
Nephritis and Bright's disease, 611; albuminuria, 613; hæmaturia, 616; casts, 617; acute nephritis, 618; chronic parenchymatous nephritis, 624; secondary contracted kidney, 627; primary contracted kidney, 630; senile arterio-sclerotic kidney, 633; bacilluria, 634; pyelitis, 635; suppurative pyelo-nephritis, 637; metastatic nephritis, 639; perinephritis and perinephric abscess, 639; tuberculosis, 640; hydronephrosis, 643; lardaceous disease, 645; renal hæmorrhage, 647; hydatid, 648; bilharziasis, 649; new growths, 651; cysts and cystic disease, 653; movable kidney, 654; calculus, 656; functional albuminuria, 660

DISEASES INVOLVING BONES AND JOINTS . . . . . 663—676
Gout, 663; infective arthritis, 667; rheumatoid arthritis and osteo-arthritis, 668; intermittent hydrarthrosis, 672; hypertrophic pulmonary osteo-arthropathy, 672; osteomalacia, 673; achondroplasia, 674; osteogenesis imperfecta, 675; osteitis deformans, 675

SOME CHRONIC INTOXICATIONS . . . . . 677—684
Alcoholism, 677; delirium tremens, 677; lead poisoning, 680; mercurial poisoning, 683; arsenical poisoning, 683; manganese poisoning, 684

DISEASES DUE TO CERTAIN DEFICIENCIES IN DIET . . . 685—697
Accessory food factors, 685; beri-beri, 686; scurvy, 688; rickets, 691; hunger osteomalacia, 695; pellagra, 695; hunger œdema, 697

DISEASES OF THE NERVOUS SYSTEM . . . . . 698—909
Anatomy and physiology, 699; involuntary nervous system, 720; clinical examination of the nervous system, 722

PAGE

DISEASES OF THE NERVOUS SYSTEM—*continued.*

DISEASES OF THE NERVES . . . . . 738
Neuritis, 738; neuroma, 743

LESIONS OF CRANIAL NERVES . . . . . 743
Olfactory, 743; optic, 744; third, fourth and sixth, 746; fifth, 747; facial, 748; auditory and vestibular, 750; glosso-pharyngeal, 752; vagus, 752; spinal accessory, 753; hypoglossal, 753

LESIONS OF SPINAL NERVES . . . . . 754
Upper cervical, 754; phrenic, 755; posterior thoracic, 755; circumflex, 755; musculo-spiral, 756; ulnar, 756; median, 757; brachial plexus, 757; sciatic, 760; external popliteal, 760; internal popliteal, 760; sciatic neuritis, 761; meralgia paræsthetica, 762

LOCALISED MUSCULAR SPASM . . . . . 763

DISEASES OF THE SPINAL CORD . . . . . 764
Transverse lesions, 764; compression, 766; tumours, 772; myelitis, 772; hæmorrhage, 777; caisson disease, 778; disseminated sclerosis, 780; syringomyelia, 784; tabes dorsalis, 786; primary lateral sclerosis, 793; subacute combined degeneration, 794; Friedreich's ataxia, 797; peroneal muscular atrophy, 798; progressive muscular atrophy, 799; amyotrophic lateral sclerosis, 802; Landry's paralysis, 803; senile paraplegia, 804; spinal meningitis, 804; meningeal hæmorrhage, 808

DISEASES OF THE MEDULLA OBLONGATA . . . . . 808
Progressive bulbar paralysis, 809; acute bulbar paralysis, 810; pseudo-bulbar paralysis, 811; compression and tumours, 812

DISEASES OF THE BRAIN . . . . . 812
Localisation of functions and effects of lesions, 813; arteries of the brain, 816; hemiplegia, 817; aphasia, 820; cerebral hæmorrhage, 821; embolism and thrombosis of cerebral arteries, 825; meningeal hæmorrhage, 828; hæmorrhage into the pons, 828; cerebellar hæmorrhage, 829; encephalitis, 829; encephalitis lethargica, 831; abscess, 836; infantile cerebral diplegia, 838; hereditary cerebellar ataxy, 839; progressive lenticular degeneration, 840; paralysis agitans, 840; meningitis, 843; pachymeningitis, 847; thrombosis of cerebral sinuses, 848; tumours, 849; chronic hydrocephalus, 858; general paralysis of the insane, 861; chorea, 864; Huntington's chorea, 867

NERVOUS DISEASES OF UNCERTAIN PATHOLOGY . . . . . 868
Epilepsy, 868; infantile convulsions, 875; migraine, 876; myoclonus, 880; the tics, 880; general convulsive tic, 881; spasmodic torticollis, 881; writers' cramp and allied neuroses, 883; neuralgia, 884; trigeminal neuralgia, 885; neurasthenia, 886

THE PSYCHONEUROSES . . . . . 889
Psychology, 889; psychopathology, 891; anxiety neurosis, 894; hysteria, 902

DISEASES OF THE MUSCLES . . . . . 910—922
Fibrositis, 910; myositis, 911; parasitic diseases of muscle, 912; new growths, 914; muscular atrophy, 914; progressive muscular dystrophy, 914; Thomsen's disease, 917; myotonia atrophica, 918; amyotonia congenita, 918; family periodic paralysis, 919; myasthenia gravis, 919; other disorders of muscle, 920

PAGE

DISEASES OF THE SKIN . . . . . 923—1019

PRIMARY LESIONS . . . . . 924

*Group I.—Diseases caused by External Infection of the Skin and its Appendages :—*

STREPTOCOCCAL INFECTIONS . . . . . 926

Impetigo contagiosa, 927 ; ecthyma, 928 ; dermatitis gangrenosa infantum, 929 ; intertrigo, 929 ; napkin-rash, 930 ; streptococcal fissure, 930 ; relapsing streptococcal lymphangitis, 931

STAPHYLOCOCCAL AND OTHER INFECTIONS ASSOCIATED WITH THE SEBORRHŒIC STATE . . . . . 931

Pityriasis simplex, 934 ; comedo, 935 ; acne vulgaris, 936 ; impetigo of Bockhart, 938 ; seborrhœic dermatitis, 939 ; sycosis coccogenica, 941 ; sycosis nuchæ, 942 ; boils, 943 ; carbuncle, 943 ; dermatitis infectiosa eczematoides, 944 ; acne necrotica, 944 ; multiple abscesses of infants, 945 ; miliaria rubra, 945 ; hidradenitis, 946 ; granuloma pyogenicum, 946

BACILLARY INFECTIONS . . . . . 947

Tuberculosis, 947 ; lupus vulgaris, 947 ; lupus verrucosus, 951 ; the tuberculides, 951 ; lichen scrofulosorum, 953 ; erythema induratum, 953 ; folliclis, 953 ; soft sore, 954 ; rhinoscleroma, 955

INFECTION WITH HIGHER FUNGI . . . . . 955

Favus, 956 ; ringworm, 956 ; tinea versicolor, 961 ; erythrasma, 961 ; blastomycosis, 961

INFECTION WITH PROTOZOA . . . . . 962

Venereal warts, 962

INFECTION WITH FILTER-PASSING ORGANISMS . . . . . 962

Molluscum contagiosum, 963 ; warts, 963

INFECTION WITH ANIMAL PARASITES . . . . . 964

Pediculus capitis, 964 ; pediculus vestimentorum, 965 ; pediculus pubis, 965 ; scabies, 966

*Group II.—Diseases classified according to their Morphology :—*

THE ERYTHEMAS . . . . . 967

Erythema multiforme, 968 ; erythema nodosum, 969 ; erythema pernio, 970 ; lupus erythematosus, 971 ; erythema ab igne, 973 ; rosacea, 974

URTICARIA . . . . . 975

Urticaria papulosa, 977 ; urticaria pigmentosa, 978

DERMATITIS VENENATA AND TOXIC ERUPTIONS . . . . . 979

Eruptions caused by drugs ingested, 979 ; eruptions caused by drugs applied externally, 980

TRAUMATIC AND SOLAR DERMATITIS . . . . . 981

ECZEMA . . . . . 981

ERYTHEMATO-SQUAMOUS ERUPTIONS . . . . . 988

Pityriasis rosea, 988 ; psoriasis, 989

PAGE

DISEASES OF THE SKIN—*continued.*

THE ERYTHRODERMIAS . . . . . 992
Pityriasis rubra, 992; premycosic erythrodermia, 993; leukæmic erythrodermia, 994; pityriasis rubra pilaris, 994

PAPULAR ERUPTIONS . . . . . 995
Lichen planus, 995; prurigo, 996; pruritus, 997; parakeratosis variegata, 997; granuloma annulare, 998

VESICULAR ERUPTIONS . . . . . 998
Herpes zoster, 998; herpes facialis, 1000; herpes genitalis, 1000; cheiropompholyx, 1001

BULLOUS ERUPTIONS . . . . . 1002
Pemphigus, 1002; pemphigus acutus malignus, 1002; pemphigus vulgaris, 1003; pemphigus foliaceus, 1004; pemphigus vegetans, 1004; epidermolysis bullosa, 1004; dermatitis herpetiformis, 1005

HYPERKERATOSES OF THE SKIN . . . . . 1005
Callosites and corns, 1005; keratosis, 1006; keratosis pilaris, 1006; keratosis follicularis, 1006; cornu cutaneum, 1006; ichthyosis, 1007

ATROPHIC AND DYSTROPHIC CONDITIONS OF THE SKIN . . . . . 1007
Scleroderma, 1007; Sclerema neonatorum, 1008; atrophoderma neuriticа, 1009; striæ et maculæ atrophicæ, 1009; xerodermia pigmentosa, 1009

ALTERATIONS OF PIGMENT . . . . . 1010
Lentigo, 1010; chloasma uterinum, 1011; ochronosis, 1011; albinism, 1011; vitiligo, 1012

AFFECTIONS OF THE SWEAT GLANDS . . . . . 1012
Anidrosis, 1012; hyperidrosis, 1012; bromidrosis, 1013; chromidrosis, 1013; hæmatidrosis, 1013; uridrosis, 1013; miliaria and sudamina, 1013

AFFECTIONS OF THE SEBACEOUS GLANDS . . . . . 1014
Milium, 1014; adenoma sebaceum, 1014; sebaceous cysts, 1014

AFFECTIONS OF THE HAIR AND HAIR FOLLICLES . . . . . 1014
Alopecia, 1015; alopecia areata, 1015; universal alopecia and congenital alopecia, 1017

NEW GROWTHS IN THE SKIN . . . . . 1017
Fibroma molluscum, 1017; cheloid, 1017; myoma, neuroma, lymphangeioma, 1018; xanthoma, 1018; mycosis fungoides, 1019

## APPENDIX

THE ÆTIOLOGY OF MALIGNANT NEOPLASMS . . . . . 1020

INDEX . . . . . 1021

# PLATES


| PLATE. | | FACING PAGE |
|---|---|---|
| I. | Radiogram of Normal Chest | 257 |
| II. | Radiogram of Chest showing Advanced Phthisis with Cavity Formation | 257 |
| III. | Radiogram of Chest showing Advanced Active Tuberculous Infiltration of the Lung | 257 |
| IV. | Radiogram showing Secondary Deposits in the Left Lung | 268 |
| V. | Fig. 1.—Radiogram of Chest in case of Pleural Effusion | 275 |
| | Fig. 2.—Radiogram of Chest in case of Pneumothorax | 275 |
| VI. | Fig. 1.—Radiogram of Chest in case of Pyo-pneumothorax | 281 |
| | Fig. 2.—Radiogram of Chest in case of Pyo-pneumothorax | 281 |
| VII. | Fig. 1.—Radiogram of Chest with Pericardial Effusion | 358 |
| | Fig. 2.—Skiagram of Aneurysm of Innominate Artery | 358 |
| VIII. | Coloured Illustration of the Retina in Arteriosclerosis and Parenchymatous Nephritis | 368 |
| IX. | Radiogram of Thoracic Aneurysm | 375 |
| X. | Radiogram of Thoracic Aneurysm | 375 |
| XI. | Radiogram showing Mediastinal Growth | 387 |
| XII. | Figs. 1, 2, 3.—Radiograms of Normal Teeth | 392 |
| | Figs. 4, 5, 6.—Radiograms of Teeth showing Early Pyorrhœa Alveolaris | 392 |
| XIII. | Figs. 1, 2, 3.—Radiograms of Teeth showing Advanced Pyorrhœa Alveolaris | 392 |
| | Figs. 4, 5, 6.—Radiograms of Teeth showing Apical Root Abscesses | 392 |
| XIV. | Radiogram of Œsophagus showing Carcinomatous Stricture | 402 |
| XV. | Radiogram showing Dilatation of the Œsophagus with Spasm in Heartburn | 402 |
| XVI. | Radiogram showing Dilatation of the Œsophagus with Achalasia of the Cardia | 403 |
| XVII. | Radiogram showing Normal Œsophagus filled with Barium | 403 |
| XVIII. | Radiogram showing Normal Stomach | 405 |
| XIX. | Radiogram of Hypertonic Stomach | 405 |
| XX. | Radiogram of Dropped Stomach | 405 |
| XXI. | Radiogram of Stomach with Ulcer on Lesser Curvature | 425 |
| XXII. & XXIII. | Radiograms of Duodenal Cap with Ulceration | 426 |
| XXIV. | Radiogram of Dilated Stomach from Pyloric Stenosis | 429 |
| XXV. | Radiogram of Stomach with Hour-glass Contraction and Ulceration | 430 |
| XXVI. | Radiogram of Stomach with Carcinoma | 434 |
| XXVII. | Radiogram of Colon with Carcinoma | 457 |

| PLATE. | | FACING PAGE |
|---|---|---|
| XXVIII. | Radiogram of Gall-stones | 500 |
| XXIX. | Coloured Illustration of Nucleated Red Cells | 520 |
| XXX. | Coloured Illustration of Normal Leucocytes | 521 |
| XXXI. | Coloured Illustration of Abnormal Blood Cells | 521 |
| XXXII. | Radiograms of Renal Pelvis in Hydronephrosis | 644 |
| XXXIII. | Radiogram of Renal Calculi | 659 |
| XXXIV. | Radiograms of the Hands in Gout | 666 |
| XXXV. | Radiogram of Hand in Rheumatoid Arthritis | 671 |
| XXXVI., XXXVII., XXXVIII., XXXIX. | Radiograms of the Arms and Legs in Rickets | 694 |
| XL. | Sections of Spinal Cord showing Degeneration of Fibre Tracts in Complete Transverse Lesion | 767 |
| XLI. | Radiograms after Lipiodol Injection | 769 |
| XLII. | Sections of Spinal Cord in Tabes Dorsalis | 787 |
| XLIII. | Sections of Spinal Cord showing conditions found in Amyotrophic Lateral Sclerosis, Subacute Combined Degeneration and Friedreich's Ataxia. | 795 |
| XLIV. | Sections of Spinal Cord in Disseminated Sclerosis | 781 |
| XLV. | (Coloured) A. Impetigo Contagiosa; B. Acne Vulgaris; C. Pemphigus Neonatorum; D. Molluscum Contagiosum | 927 |
| XLVI. | (Coloured) A. Seborrhœic Dermatitis; B. Pityriasis Rosea; C. Psoriasis; D. Infantile Eczema | 939 |
| XLVII. | (Coloured) A. Erythema Multiforme; B. Lupus Vulgaris; C. Urticaria Papulosa; D. Lupus Erythematosus | 969 |
| XLVIII. | (Coloured) A. Scabies; B. Pediculosis Corporis; C. Alopecia Areata; D. Tinea Tonsurans. | 1015 |

# THE PRACTICE OF MEDICINE

## INTRODUCTION

A WORK on the PRACTICE OF MEDICINE should deal with Diseases, their Nature, Course, Prevention and Treatment.

It has never been very satisfactorily determined what is to be called disease, or what are to be called diseases. To say that disease is any divergence from health or anything that is the opposite of health opens up the question, What is health? To this the answer is: A perfect structure of all the organs or tissues, with a perfect performance of all their functions; and, in the broadest sense, any alteration of structure or function may be called disease. But some special cases have to be considered.

A distinction is commonly drawn between injury and disease; but the inflammation which so often results from injury is disease of structure and function, and although the immediate effects of injuries are not generally spoken of as diseases, remote troubles occur which are so classed.

Slight ailments, whether of a structural kind, such as mild catarrhs, or of a functional kind, like headaches, are often spoken of not as diseases, but as disorders or ailments. There is, however, no essential difference, and no broad line of distinction can be drawn between those which pass off readily, leaving no trace, and those which persist, or recur frequently, or finally shorten life.

The structural changes of the various organs and tissues of the body certainly constitute diseases, such as inflammation of the lung or pneumonia, chronic inflammation or cirrhosis of the liver, and cancerous growth of the stomach. Such changes are spoken of as *primary* disease, meaning thereby that each is the first essential lesion in the history of the patient's illness, although each owns some precedent cause, such as contagion or by micro-organisms in the case of pneumonia, alcoholic indulgence in the case of cirrhosis, and some hitherto unknown causation in the case of carcinoma of the stomach.

The name of disease must also be given to the disturbances of anatomy that may follow such primary lesions in some cases. Thus, cirrhosis of the liver leads to effusion of liquid into the peritoneal cavity, abdominal dropsy or ascites; carcinoma of the stomach, involving the pylorus, causes secondary dilatation of the walls of the stomach; valvular disease of the heart causes congestion of the liver and kidneys, and dropsy of the feet and legs.

Then, again, as a result of either of these groups of anatomical changes, there are certain disturbances of functions and abnormal subjective sensations, such as pain, vomiting, jaundice, the passage of albumin in the urine, and others. Are they also diseases? There is no difficulty in coming to a conclusion that in these also there is disease.

Lastly, we have to deal with a class of disorders in which no structural change whatever can be found: these are mostly referable to the nervous system; they consist of pain, or spasm of muscle, or loss of sensation, or loss of power, or vascular changes secondary to functional alterations in the vasomotor nerves. This change in every case is for the time being disease, and the different instances are grouped as *functional diseases*. Their number is likely to diminish, as systematic research reveals a hitherto unsuspected structural change in one case or the operation of some toxin in another.

But the subject may be approached from another point of view. Having decided that the term *disease* has a very comprehensive meaning, we may seek to determine how we shall employ the word *diseases*—that is, how we shall arrange what we know of the possible changes of structure or function into separate groups for practical purposes. These practical purposes, in relation to diseases, are their recognition during life, when the investigation of structure cannot be so complete as after death, and their subsequent treatment. We then perceive that the various changes of structure, which we cannot ourselves see, are accompanied by certain other changes, secondary diseases or functional disturbances, which are so many indications, or *symptoms*, of the deeper change. Thus, with pleurisy we have pain, cough, and shortness of breath; with carcinoma of the stomach, pain and vomiting. These indications may be subjective experiences on the part of the patient, like pain, tingling, inability to eat or move; or objective signs to the physician, such as swelling, hardness, elevation of temperature. And among these objective signs must be mentioned especially the indications known as *physical signs*, a term used mostly in reference to the examination of the chest and abdomen with the eye (inspection), the hand (palpation), and the ear (auscultation or percussion). Thus *symptoms* and *physical signs* are regarded as forming the sum total of all the results of a structural lesion which can be manifest to the physician and guide him in his opinion.

Now, first, the patient has certain experiences of disorder and discomfort, which are to him a very real illness or disease; secondly, the physician, by his special knowledge, can show the existence of other disturbances unperceived by the patient; and, thirdly, there are still facts which he may be unable to observe, and which are only revealed by the pathologist on post-mortem examination.

Are we to limit the term *diseases* to the last or to the second of this series, or may we allow it to be used also for the headaches, vomiting, dropsies, convulsions, etc., which are the most prominent part of the patient's suffering, but which are to the physician only the symptoms of something deeper, which he is constantly striving to identify? Provisionally, no doubt, we must allow the term a wide application. For, in the first place, there is the whole class of functional disorders, which have, as far as our present knowledge extends, no anatomical basis, and which must, nevertheless, be distinguished from one another and treated; and, secondly, there is the fact that lesions of the deeper organs may be very imperfectly revealed even by conditions which cause much suffering to the patient.

But though we may be sometimes compelled to think of dropsy, or headache, or neuralgia as a disease, we must remember that we should never rest content with that position until every effort has been made to find out what organ or structure may be at fault; and we should never permit ourselves to fall into the slovenly habit of ticketing all symptoms with the name of diseases, and treating them without any attempt to remove or modify the ultimate cause, when it may be with no great difficulty ascertained.

A thorough knowledge of every disease requires an acquaintance with several separate branches of study, which are as follows:—

*Ætiology*, the study of its causes in general. *Pathology*, the study of its causes within the body, and the processes resulting therefrom. *Morbid Anatomy*, the alterations in the structures caused by it. *Symptoms*, the indications—subjective on the part of the patient, and objective to the physician (usually called *physical signs*)—by which we arrive at a knowledge of what is wrong. *Diagnosis*, the method of distinguishing from one another the diseases that may have symptoms more or less nearly alike. *Prognosis*, the art of foretelling the course, duration, and termination of any given case. Finally, the *Prevention* of disease (prophylaxis), and the *Treatment* of disease when it occurs, both of which are the chief aims of the study of the science and art of medicine.

**Ætiology.**—The causes of disease are commonly divided into *predisposing* and *exciting*, but no broad line can be drawn between them. A predisposing cause may be in operation for a great length of time without the disease being produced, whereas the exciting cause is usually only of short duration; but conditions which act as predisposing causes at one time may act as exciting causes at another. *Ætiology* strictly covers the whole of the causation of disease, but it is perhaps more often applied to the remoter causes and to those conditions which are constantly associated with a disease, although we are unable to say how they influence its occurrence. The relations to disease of age, sex, climate, hygienic surroundings, food, and preceding illnesses are commonly considered under this heading. On the other hand, changes taking place in the body, immediately preceding or causing the disease, are considered rather as pathological processes than ætiological factors.

**Pathology** is sometimes used to signify the study of diseased structures, but it is better limited to the study of diseased processes—that is, practically, the influence which the causes of disease have upon the function and structures of the body; while **morbid anatomy** or **pathological anatomy** describes in detail the diseased structures themselves.

**Symptomatology** is the study of the symptoms of any disease, and associated with this and with its morbid anatomy we have to consider what have been called **complications** and **sequelæ**. Emphasis has with perfect right been recently laid on the study of the early symptoms of disease, so that treatment may be carried out in the earliest stage.

Complications are certain lesions or symptoms which are the result of the original disease, but only occur from time to time, and are not regarded as a necessary part of the disease; thus abscesses are a complication of enteric fever, hæmoptysis a common complication of pulmonary tuberculosis, parotitis a very rare complication of pneumonia. But the term is applied somewhat arbitrarily; for instance, in enteric fever rose spots and diarrhœa are not universally present, and yet they are regarded as part of the disease, and never as complications. We must regard in a different light the case where one disease occurs at the same time as another, but, as far as our own knowledge goes, is quite independent of it; the complication may seem to be purely accidental, yet the primary disease may have disposed the individual, in some way not hitherto ascertained, towards the acquirement of the second. Two common cases are (1) the complication of any slight or severe local disorder by an infectious disease, such as scarlet fever, caught by contact; (2) the termination of some chronic nervous disease, like hemiplegia or locomotor ataxy, by pneumonia or bronchitis.

A sequela is a symptom or lesion appearing or persisting after the original disease has subsided.

**Diagnosis** is the recognition of diseases by certain symptoms, physical signs or facts in the history of the cases which, taken together, indicate that the patient is suffering from a particular ailment. By *differential diagnosis* is meant a knowledge, in any given disease, of all the other diseases which most clearly resemble it and the points of difference upon which reliance may be placed to distinguish it.

If two complaints are likely to be confounded together, and one is much more serious than the other, a wrong decision that the less serious is present is dangerous for the patient, since his undiagnosed complaint may progress to a fatal point without proper treatment. On the other hand, a wrong decision that the more serious is present may cause the physician to alarm the patient unnecessarily and ultimately bring discredit upon himself. Especially is it desirable in the case of the commoner, temporary, and more curable complaints to bear in mind the possibility of their being simulated by rarer and more dangerous diseases. The confusion of angina pectoris with various forms of chronic indigestion and of appendicitis with acute indigestion are instances which too often occur.

Probability is an important element in diagnosis. In exceptional instances a disease may present a physical sign, symptom, or indication which is not caused by any other known condition. Such a sign or symptom is said to be *pathognomonic* of the disease in question. As a matter of fact, there are very few *pathognomonic symptoms.*

It will have been gathered from the remarks on diseases that by diagnosis we shall always aim at finding out the *primary* lesion; thus we must not be content with calling any pain rheumatism or neuralgia, but we must try to find out whether such pain is caused by pressure on a nerve, by inflammation of a nerve, or by degeneration of nerve tracts. But in many cases the patient suffers from numerous symptoms: pain, cough, sickness, dropsy, albuminuria, and others. As a rule, we should try to see how far all these conditions may be due to one single primary lesion, such as valvular disease of the heart or granular disease of the kidney; but we must not forget that frequently two or more independent lesions co-exist, and produce a complex arrangement of symptoms.

**Prognosis.**—Successful prognosis requires a thorough acquaintance with the natural history of every disease, with the extent to which the disease is influenced by age, sex, and other ætiological factors, and a careful judgment on the variations of the patient from day to day.

The questions that arise in prognosis are such as these: Will the patient recover? Will he recover completely or be left with any organ damaged? Will he ever have the disease again? If it is a fatal disease, how long will he live?

In the early days of a disease the question of recovery can only be answered by a consideration of the percentage mortality as shown by statistics. As the case proceeds the rapidity or severity of the symptoms, the conditions of the circulation, the ability to take food, and the integrity of the nervous system are the points which have most bearing on one's opinion. In practice, prognosis is often of the greatest importance for the physician's credit, and a hasty conclusion which turns out wrong is often remembered against him more than any want of success in treatment.

When in this volume it is stated that the prognosis of any disease is favourable, this means, not that it is never fatal, but that *most* cases recover; if any particular symptom or complication renders the prognosis *less* favourable, this means that the percentage mortality of cases with such complication is greater than it was before or without the complication.

**Prevention.**—The *prevention* or *prophylaxis* of disease may be looked at from two points of view: (1) that of the community, and in this connection it is known as the study of *hygiene* or *public health*, and involves all measures by which the community endeavours to ward off all external influences adverse to health; (2) that of the individual, by which the body itself may be prepared against the operation of the causes of disease, partly by judgment in matters of diet, exercise, clothing, etc., and partly by such special treatment in relation to particular diseases as is effected by vaccination and antityphoid inoculation. For efficient prevention in this sense it is essential to recognise the very beginnings of disease, so that remedies may be applied before the disease becomes established and irremediable structural alterations in organs have taken place. For this purpose the early symptoms exhibited by patients require special study. Prevention of this kind is of growing importance in medicine, and it will be considered in detail in the appropriate places.

**Treatment.**—In this we should aim first at the removal of the cause where this is possible; if not, we may succeed in neutralising its influence. One or other of these methods may suffice to cure all the symptoms and troubles of the patients; but in most cases we are also called upon to deal directly with the symptoms, using remedies that have no influence upon the underlying disease.

We must, when doing this, never forget that such symptoms hold a position secondary in importance to that of the disease which causes them. Lastly, we must in all cases counteract the tendency to death, which may, indeed, be the natural course of the disease, or may arise rather as an accident from some infrequent complication. As an example we may take phthisis, which is due primarily to the invasion of the lung by the tubercle bacillus. The removal of this, when once it has obtained a footing, cannot be directly effected. Its influence can be neutralised by the best hygienic surroundings, by fresh, bracing air, and by special climates which enable the body to resist the action of the bacillus. In the meanwhile there are numerous symptoms—cough, expectoration, pain, sweating, diarrhœa—which will diminish as the condition of the lung improves, and which can be also controlled by suitable medicines. In addition serious complications may arise, especially hæmoptysis, or spitting of blood, by which life is directly threatened, and such a death may be averted by proper therapeutical means. Pleuritic effusion is an instance in which we have cough, dyspnœa, pain, and distress due to the presence of liquid in the pleura; the removal of the liquid, either by tapping or by the use of drugs, is followed by relief of all the symptoms.

It is more than ever necessary to realise that the treatment of an illness does not consist simply in giving internally some form of medicine, but that the processes of the body can be influenced in many other ways. So much is this the case that it seems desirable that a short account should here be given of the therapeutical measures which are available, and which are, of course, described in full detail in works on therapeutics.

*Rest.*—Not much need be said under this head. In many diseases, *e.g.*, acute infections, rest is the most valuable method of general treatment we possess. The presence of pain is one of its most important indications, as was pointed out by Hilton in his book "Rest and Pain." The subject of rest will be constantly referred to throughout this book.

*Diet.*—Food consists of proteins, fats and carbohydrates, and the proportions of these substances may have to be altered in certain cases. Thus carbohydrates must be restricted or omitted from the diet in diabetes. Again, flesh foods contain purin substances which may be harmful in gout, and strict moderation must be exercised. Patients confined to bed require less food than persons taking active exercise; in febrile states more food will be needed than when the temperature is normal, because the energy output is greater; at the same time, it must be taken in an easily digestible form, and the latter applies to various gastro-intestinal diseases. The diet must be arranged so that it contains sufficient of the various *accessory food substances*, or *vitamins*, the presence of which is essential to life.

*Drugs.*—The British Pharmacopœia gives the official list of drugs which are required in treatment, but fresh drugs are constantly being introduced, and older drugs not now in the Pharmacopœia may still be of service. Besides the older methods of administration by the mouth and rectum, and through the skin by means of ointments and by medicated baths, drugs are now introduced by hypodermic, intramuscular, intravenous, and intraspinal injection, and also by *ionisation* or *cataphoresis*. In this method ionisable salts, of which one or both of the component elements are efficient drugs, are applied to the skin; and, by means of a galvanic current, one or other of the ions is driven into the tissues, and exerts the desired effect.

*Organo-therapy* or *Opotherapy.*—As special forms of drugs, which can be given internally or *per rectum*, or by subcutaneous injection, must be mentioned the extracts of various organs of animals (thyroid, suprarenal gland, pituitary body, insulin) which may be made to supply defects in the corresponding organs of the human subject.

*Antitoxic and Antibacterial Sera.*—These are of great value in some infectious

bacterial diseases, for instance, in diphtheria. They consist of the serum of an animal which has been rendered immune to the particular disease; and the serum contains anti-bodies which will neutralise the disease in the human patient (*see* p. 16). They are standardised by careful experiments upon animals. They may be injected subcutaneously, or into the muscles, or into the veins, or within the theca of the spinal column.

*Bacterial Vaccines.*—A vaccine is a solution containing several million dead bacteria of the same species as those of the disease requiring treatment, and preferably cultivated from some obtained from the patient to be treated. The injection of these bacteria increases the opsonic power of the patient's blood serum, and thus assists in the ultimate destruction of the bacteria causing the disease.

In the remaining therapeutical methods the forces of nature are applied in various special ways.

*Heat.*—Besides the local application of warmth for purposes of stimulation or counter-irritation, and of heat for its destructive effects (cautery), *radiant heat*, by means of incandescent electric lights, is employed for the local treatment of rheumatic joints and allied conditions.

*Diathermy* is a more recent method of applying heat locally. The heat is produced by continuously maintained high frequency currents, and in this form it penetrates more deeply into the part, *e.g.* a joint, than any heat, that can be borne, supplied by sources external to the limb.

*Light.*—The general advantages of a bright, sunny climate are well known, and even in less clear climates it is of use in some complaints to expose the patient to bright sunlight whenever the opportunity offers. The Finsen light treatment of lupus and rodent ulcers consists in directing an intense light upon the diseased part for specific periods of time. The light consists of violet and ultra-violet rays, and is produced by an arc-light from which the heat rays are cut off.

*Roentgen Rays or X-rays.*—The powerful effects of these rays are well known, both for good and evil. Constantly playing upon the unprotected skin, as in the case of X-ray operators, they have caused intense and incurable dermatitis, leading on to carcinoma. Death has also been caused by their destructive action on the blood-forming organs, producing *aplastic anæmia*. Used with proper precautions for limited periods, they modify the growth of cells in the body, and have been of value in the treatment of rodent ulcer, carcinoma, enlarged glands, leukæmic spleen, syringomyelia, ringworm, and other affections. In deep X-ray therapy the less penetrating rays are filtered off and the more penetrating rays alone used. Structures lying deep inside the body, *e.g.* the uterus affected with carcinoma, can be reached by the rays and, by varying the direction from which the rays come, in other words the portal of entry, the skin is saved.

*Radium.*—The rays emanating from this substance have also powerful effects for good and evil. Their effect somewhat resembles X-rays.

*Electricity.*—The chief uses of this force have been in the treatment of paralysis and other nervous diseases. Muscles which cannot be stimulated by the will can be made to contract by electric stimulation, so long as their nutrition is normal and they are not the subject of atrophy. This contraction, effected at stated intervals, maintains the circulation of blood and lymph in the muscles, and facilitates the return to health. Such contractions can be effected by the faradic or by interruption of the continuous current. Many painful neuralgic affections are benefited by a continuous current of electricity. Another application of electricity now often used is that of high frequency currents. These are currents of high potential, perhaps 10,000 volts, alternately positive and negative, and changing their sign about every millionth part of a second. They are consequently too rapid to stimulate sensory nerves or motor nerves or

muscles, which can only respond to stimuli of about $\frac{1}{1000}$ second duration; nevertheless they have certain effects upon the tissues, which are claimed to be increased cellular activity, changes in the vascular system, and inhibition, *i.e.* diminished susceptibility of the neuromuscular system to ordinary stimuli.

*Baths and Douches.*—By these means heat and cold and mechanical effects may be produced, and so the vasomotor system and the circulation may be affected both locally and generally. Medicated baths, alkaline baths, and sulphur baths are extremely useful in some circumstances. The hot air bath and vapour bath are valuable means of promoting diaphoresis or sweating.

*Massage.*—By this is meant the manipulation of the patient's limbs and body by an operator in such a way as to assist the circulation of blood and lymph in the part, and to supply the stimulus to metabolism which is furnished in the healthy person by active exercise. In the same way stiff joints may be improved and adhesions loosened. Locally it is employed after injuries to the limbs, and it is valuable when applied to the body as a whole in several nervous disorders, such as hysteria and neurasthenia.

The methods of manipulation include friction, pressure, and percussion of the limb, and passive movement of the joints. In *friction*, or *effleurage*, the part is stroked with the palm of the hand in an upward or centripetal direction, and the hands are used one after the other with regularity, and more or less quickly according to circumstances. *Pressure* includes *pétrissage*, by which a portion of muscle is picked up with the fingers and thumb of one or both hands; it is subjected to firm pressure and rolled between the fingers and the subjacent tissues. Other portions are similarly treated one after another, the operator working from periphery to centre. *Percussion* includes *tapôtement*, or percussion with the tips of the fingers, the palmar surfaces of the tips, the palm of the hand, the back of the half-closed hand, the ulnar or radial border of the hand (*hachure*), or the whole hand hollowed so as to enclose some air between it and the surface of the limb, and *flagellation*, or striking with a wet towel. *Kneading* and *vibration* are also employed.

The joints may be subjected to movements of circumduction, flexion and extension, and traction. Rubbing movements should be centripetal; they should be done with the dry hand, without the intervention of oil, ointments or liniments.

The duration of massage should be from five to fifteen minutes on any one occasion; but in recent cases the sittings may be three or four in a day. The effect of these manipulations is to promote the flow of lymph and blood in their respective vessels, and to stimulate the muscles of the skin and the skin reflexes.

*Swedish Movements, Nauheim Treatment, Regulated Exercise, Fraenkel's Movements.*—All these are active movements made by the patient himself under the direction of the physician or operator, with the object of providing a daily amount of exercise either locally or generally, in relation to the functions of a joint, or to the powers of a weak heart, or to the cure of obesity, or to the working of the digestive functions. Or, as in the case of Fraenkel's exercises, the object may be the re-establishment of the function of co-ordination in muscular movements.

## CLASSIFICATION

Some time ago diseases were divided into *general* and *local*, the latter being those in which particular organs or parts of the body were mainly or alone affected, such as the heart, the lungs, the brain, or the skin, and the former those in which the whole economy was disturbed, so that it could scarcely be said that one organ was more affected than another. An important feature of most of these *general* diseases was that they were accompanied by fever, that they were contagious, or transmissible from one person to another, and that one

attack protected the individual more or less completely against a subsequent attack.

Improved methods of research with the microscope led to the proof that not only did the *contagion*, or *virus*, or medium of transmission from man to man, consist of visible particles, but that these particles were themselves living organisms, different in different diseases, and capable of cultivation and reproduction both within and without the body. The study of these micro-organisms is the science of *bacteriology*.

The general diseases were thus seen to be both infectious and specific, and of these typhus, scarlet fever, measles, small-pox, and influenza are examples. But extended researches showed also that many diseases hitherto considered local were also due to specific and infective agents, for instance, pneumonia, tetanus, and diphtheria; and in their intimate pathology these disorders with their pronounced local manifestations must fall into line with the well-known fevers, such as typhus and small-pox. Classification then is rendered difficult by the fact that if the organs of the body are taken as a basis, there are *general* diseases which affect the whole body simultaneously and no one organ in particular; but when these are investigated and their bond of union is found to be the infective micro-organisms, then it is seen that a number of diseases affecting mainly one organ or part of the body (hence *local* disease) must be included with the infectious disorders; and as the tendency of all modern research is to discover micro-organisms as the cause of every inflammatory and degenerative lesion, and even of some new growths, the list of infectious diseases is constantly being increased by the addition to its general disorders of diseases hitherto considered local and non-infective.

Within the limits of one physiological system the same difficulty arises, and the attempt to separate the diseases of the brain from those of the spinal cord, or those of the stomach from those of the intestine, frequently fails, because the parts are simultaneously affected by some common cause. Thus locomotor ataxy and general paralysis of the insane are both results of syphilis; and several organisms will cause inflammatory lesions at the same time of the brain and spinal cord, of the cerebral and spinal meninges, or of the gastric and intestinal mucous membranes.

There is, however, an advantage in retaining many of the diseases formerly called local as long as possible in their old groupings, and it is this, that in their physical signs and symptoms they are necessarily comparable with the other local disorders of the same group, whether the infectious nature of these latter has been shown or not. Consequently the first section, on "Infectious Diseases," will be found to contain those which are obviously general diseases, with some only of those which have pronounced local characteristics. The succeeding sections will deal with the diseases of the various systems, nervous, respiratory, cardiac, alimentary, etc., amongst which it will be admitted that many have an origin in specific infection.

# INFECTIOUS DISEASES

## NATURE OF INFECTION

By infectious diseases are meant the diseases which depend upon the introduction into the body from without of a *virus;* and this infective agent, wherever it can be demonstrated as a visible thing, has proved to be a microscopic living being, or *micro-organism*, which can multiply within the body.

But even after years of research it has been found impossible to discover by the help of the highest powers of the microscope the organisms of some of these obscure diseases, such as measles and hydrophobia. The organisms are sometimes said to be ultra-microscopic, and the limit of visibility is apparently a diameter of 0·16$\mu$. But it is, of course, conceivable that still more powerful lenses may be produced, by which organisms may be found in these diseases; and it is further possible that, in addition to the chemical stains which are absolutely necessary for the demonstration of now familiar bacilli and cocci, others may be discovered which will reveal the organisms hitherto invisible. It was many years after the discovery of the bacilli of tubercle and influenza that the much larger spirochæte of syphilis was, by special staining methods, made obvious.

That in most of these diseases the size of the organism is the chief cause of our failure to see it is shown by the fact that in several of them the tissue fluids, or fluids prepared from the tissues, will pass through the finest known filters, as proved by the possibility of inoculating monkeys and other animals with the disease after the use of the filter. Such a virus or organism is provisionally spoken of as a filter-passer, and the following are regarded as belonging to this group, namely, the organisms of measles, hydrophobia, dengue, sand-fly fever, poliomyelitis, encephalitis lethargica, variola and vaccinia.

Of the micro-organisms, which have been demonstrated to be the causative agents of specific, definite, often febrile illnesses by the combined use of various stains and of very high powers of the microscope, the greater number belong to the class of *Schizomycetæ*, or *fission fungi*, and are called the *lower bacteria*. These are all minute cellular bodies, formed of nuclear material; and they possess the faculty of being stained, when dead and dried, by certain dyes, such as methylene blue, gentian violet, and fuchsin. They occur in various forms, such as minute, spherical or ovoid bodies, called *cocci* or *micrococci;* straight rod-like bodies, called *bacilli;* spiral or screw-like bodies, called *spirilla* or *spirochætæ*. Cocci may adhere together in long threads or chains (*streptococcus*), or in cubical groups (*sarcina*), or in irregular bunches (*staphylococcus*). Some bacteria possess *ciliæ* or *flagella*, by means of which they acquire the power of independent movement. The flagella are few or many, generally longer than the body of the cells and spirally twisted.

These micro-organisms are reproduced by *division* and by the *formation of spores* within the bacterial cell (*endogenous*). The first leads to the most rapid multiplication of the organisms, and is spoken of as a *vegetative stage*. The second, or sporulation, takes place under special circumstances in bacilli and some spirilla; growth and multiplication are relatively slow, and the process is regarded as a *resting stage*. Spores are more resistant to destructive agents like heat, drought, and disinfectants than the micro-organism itself.

Another group, the *higher bacteria*, belonging to the class of *Trichomycetæ*, are also sometimes the causes of infectious disease. These are of somewhat greater size, consist of filaments made up of simple cells, and have special organs of reproduction in the cells called *gonidia*. The recognised forms are *beggiatoa*, *thiothrix*, *leptothrix*, *cladothrix*, and *streptothrix*.

A little higher in the scale are *Moulds* or *Hyphomycetæ*, to which the organisms of ringworm and favus belong.

A few infectious diseases are dependent upon organisms which are admitted to belong to the animal kingdom in the class of *Protozoa*. They are generally less minute, have more variety of structure, and in some cases multiply by a definite sexual process. Examples are the *entamœba* of dysentery, the *hæmamœba* of malaria, the *trypanosoma* of sleeping sickness, and probably the organisms of relapsing fever, syphilis, yaws, and kala-azar.

In relation to disease another division has to be made. Some bacteria are proved to be actual causes of disease, and are called *pathogenic bacteria* or *parasites*. They thrive on the living animal and vegetable tissues. Others are not usually the causes of disease; they may be found in association with the parasites; but they flourish in dead and dying animal tissue and in vegetable and inorganic matter; they are called *saprophytes* or *saprophytic bacteria*. However, some saprophytes may become parasites and cause disease; and, conversely, most parasites can thrive on artificial media, and hence behave as saprophytes.

With the first discoveries of pathogenic micro-organisms it was naturally supposed that the *specific* cause of the disease had been found; but, in the sense that each particular organism is peculiar to one disease alone, this has not proved to be true. In some diseases only one pathogenic micro-organism is found, and this may be regarded as *specific*, *e.g.* in anthrax and syphilis. In other diseases, such as infective endocarditis, septicæmia and pneumonia, more than one pathogenic organism has in different cases been found.

The conditions which an organism must fulfil in order to be regarded as pathogenic were first laid down by Koch, and these were subsequently amplified by Kanthack. A pathogenic specific germ (*a*) must be a parasite or a facultative parasite; (*b*) it must be found invariably in the tissues of an animal dead from, or affected with, the disease in question; (*c*) it must never under any circumstances occur in other diseases, nor within the normal tissues; (*d*) the organism transmitted from the diseased or dead animal to an affected susceptible animal must reproduce the lesion, and in this second diseased animal the original organism must be found; (*e*) if the organism can be cultivated outside the animal body, then an artificial cultivation inoculated experimentally into a susceptible animal must again produce the disease, and this animal must again contain the organism in its tissues or blood; (*f*) these processes must occur in invariable succession under identical conditions; (*g*) the toxins and poisonous substances obtained from the artificial cultivations must agree chemically and physiologically with those obtained from the diseased animal.

The organisms of a limited number of diseases, including anthrax, diphtheria, and tetanus, fulfil all these conditions; those of glanders, tuberculosis, actinomycosis, gonorrhœa, and malignant œdema fulfil all but the last. Diseases in which there is more than one pathogenic organism have been already mentioned. On the other hand, though presumably existing, no specific organism has been identified in the following diseases: hydrophobia, dengue, mumps, the exanthems, and some others.

Many lesions complicating the above diseases are due to *secondary infection* by the organisms which stimulate the body to produce pus (pyogenic) (*streptococcus*, *staphylococcus*), pneumonia (*pneumococcus*), and others.

**Action of Virus in the Recipient.**—The virus, or the micro-organism, where such exists, enters the system by the respiratory tract (scarlet fever, smallpox), the throat (diphtheria), the alimentary canal (enteric fever, cholera),

the genital mucous membranes (gonorrhœa, syphilis), by the bites of insects (malaria, yellow fever, sleeping sickness), or by coarser lesions of the skin (syphilis, hydrophobia). An attack of the corresponding disease does not necessarily follow, for the individual may not be susceptible (*natural* or *acquired immunity*), or the organism may not have the necessary degree of virulence. Streptococci, pneumococci, and diphtheria bacilli may often be found in contact with the tissues of healthy persons.

If, however, the organism is virulent and the individual susceptible, the entry of the virus is followed by a period of *incubation*, during which no changes are manifest, and which varies generally from two or three to twenty-five days, being constant within limits for each particular disease. During the period of incubation the organisms are developing and multiplying, and elaborating the poisonous products to which for the most part the different symptoms and effects of an infectious disease are due. The possible products of bacterial action are many, for instance gases, fatty acids, bodies of the aromatic series, pigments, ferments, and ptomaines; but the most important of all in reference to disease are the toxalbumins, albumoses, or toxic proteins, which have been found in the fluid in which bacteria have been cultivated, and which have been shown to be the agents to which the symptoms can in many cases be attributed.

It is by the action of the bacteria and their toxins that the greater number of the pathological changes are produced which we know as the basis of disease in the body. These can be only briefly referred to here. They consist of acute local changes both at the seat of inoculation (or entrance of the virus) and elsewhere, whether inflammation, hæmorrhage, œdema or necrosis (vaccinia, syphilis, diphtheria, enteric fever); eruptions on the skin, or *exanthems* (ἐξ, out, and ἀνθέω, I blossom), which may be either acute or chronic (scarlatina, measles, syphilis); various chronic local lesions, with cell growth, such as the so-called infective granulomas (tubercle, syphilis, actinomycosis); more widely distributed lesions, such as the cloudy swellings of glandular cells, hæmorrhages in various parts of the body; and, lastly, changes in metabolism, which result in malnutrition, cachexia, and often in febrile reaction or *pyrexia*, of which an account will be given later (*see* p. 18).

The micro-organisms are sometimes confined to the seat of inoculation or invasion, while their poisons or toxins alone are diffused through the system (*toxæmia*); or the micro-organisms multiply in the blood vessels, and are carried by them to the organs and tissues (*septicæmia*). In the latter case they may become impacted in different parts of the circulatory system, and thus form fresh foci of disease. In general *pyæmia* these foci are seen in the lungs and in the organs of the body supplied by the systemic circulation, and infection has gained access to the liver by the portal vein in *portal pyæmia*.

During the progress of the illness the bacteria or their germs are given off from the patient in various ways, and may thus become a source of infection in other individuals.

The *duration* of a specific disease is often very strictly limited. Thus typhus, relapsing fever, scarlatina, measles, small-pox, and vaccinia have all a definite duration, which is rarely more than three weeks, and is adhered to with some constancy. In other acute disorders the duration is longer and more variable, but can be generally measured by weeks. In others, again, as syphilis, leprosy, and tubercle, the infection may be lifelong; but in the first of these there are limitations to the duration of the earlier lesions, which assimilate it closely to the typical specific fevers. The question what terminates the infection—that is, what kills the micro-organisms or renders their poisons innocuous—is not yet completely answered. Probable causes are: the influence of the febrile temperature (*see* Relapsing Fever); the destruction of the bacilli by leucocytes (*phagocytosis*); and, especially, the formation in the blood or tissues of substances (*anti-bodies*) prejudicial to the bacilli and their poisons.

**Transmission of Infectious Diseases.**—This is really a branch of Public Health, but a brief notice of it cannot be excluded from a work like this. The infectious diseases having been defined as those in which a virus, or micro-organism, is introduced into the body, it must be here stated that the virus is derived, first, from other human beings ill of the disease directly or indirectly, as in scarlatina, measles, and many others; or, secondly, from animals, as in rabies, anthrax, foot-and-mouth disease; or, thirdly, from the soil or other source independent, as far as is known, of the previous participation of other men or animals in the process, as in tetanus. When transmitted from one human being to another, it may be, apart from experimental inoculation, conveyed in solid tissues, in liquid secretions, normal and pathological, in expired air, in clothes, or other articles. In many cases, as in scarlet fever and diphtheria, the secretion from the respiratory mucous membrane, in the form of droplets of sputum, is the means by which the poison is conveyed; in others, as in cholera or enteric fever, the fæcal discharges; and in others, as in syphilis and glanders, the pus from sores. The exhalations from the breath and skin render the patient *contagious* in the proper sense of the term—that is that those who are near to the patient for a longer or shorter time run some risk of catching the disease; the fæcal evacuations commonly reproduce the disease by infecting the water or milk which others drink or, possibly, the air which others breathe; and, lastly, pus containing the virus must come into direct contact either with the mucous membrane or with an abrasion on the surface of the skin.

In an increasing number of diseases it is becoming evident that the virus or micro-organism is conveyed from the sick to the healthy by means of biting or sucking insects, which either take up the infecting agent in blood from the patient's skin and discharge the virus by puncture into the skin of a new host, or discharge on to the healthy skin fæces which subsequently get rubbed into punctures, or otherwise infect it. Thus malaria, yellow fever, and sleeping sickness are conveyed by mosquitoes or tsetse flies; typhus by lice; relapsing fever by lice; a similar disease in Africa by ticks; plague by fleas; while the common house fly may possibly have a share in the transmission of cholera, typhoid, infantile diarrhœa, ophthalmia, and some other diseases. In some cases the virus, or micro-organism, undergoes development in the body of the insect.

The period during which a patient suffering from an infectious disease can convey it to others is determined by the duration of the infection in him (*see* p. 11). It begins, no doubt, with the appearance of the earliest symptoms—that is, at the end of the period of incubation—and in acute diseases is generally limited to three, four, or five weeks. If contagion is conveyed by the scabs of pustules (small-pox, varicella), secretions from the throat (diphtheria, scarlet fever), or unhealthy stools (enteric, cholera), the duration will depend on the persistence of these conditions. Increasing importance attaches to the fact that the organisms may persist in the individual for months or years after convalescence is complete, and thus may be the cause of infection in others. This happens in typhoid fever, diphtheria, cholera, cerebro-spinal fever and poliomyelitis, and the persons conveying the infection are called *carriers*.

**Mixed Infections.**—Bacteriological study soon showed that the old doctrine that two infectious disorders could not attack the body at the same time was incorrect, and, on the other hand, that the occurrence of one infection often rendered the body even more susceptible to a second. Moreover, the virulence of many organisms is an extremely variable quantity, and is dependent in part upon the pre-existing operation of others. Some of the more familiar instances of mixed infections are the co-existence in the same person of scarlatina with diphtheria, of scarlet fever with whooping-cough, of scarlet fever with chicken-pox, of diphtheria with measles, of whooping-cough with broncho-pneumonia, of

tubercle with lobar pneumonia; but the most important and frequent, perhaps, is the secondary invasion of the body in a great number of infectious diseases by the pus-forming organisms *Staphylococcus pyogenes aureus* and *Streptococcus pyogenes*, leading to suppurative lesions, septicæmia, and pyæmia as complications or sequels of the original disease.

**Prevention of Infection.**—There are three ways by which the transmission of infectious diseases from one person to another, or others, may be prevented. One is by separating the sick from the healthy (*isolation*). Another is by destroying the virus in the sick person, or in whatever clothes, books, room or furniture he may contaminate, or in whatever excreta may pass from him (*disinfection*). If insects are a factor in the contagion, they should be exterminated if possible or, at least, prevented from contact with the sick. The third method is by so modifying the condition of the possible recipient that he becomes insusceptible to the influence of the virus, even if brought into contact with it (*production of immunity; immunisation*).

**Isolation.**—The patient should be placed in a separate room, if possible on a separate floor of the house, which may be screened off by a sheet wetted with a solution of carbolic acid (1 in 40). Thorough ventilation must be as far as possible maintained, as the dilution of the poison by a constant influx of fresh air is a most important part of the process. All unnecessary furniture, curtains and carpets, clothes, etc., to which contagion may adhere, should be removed from the room. The attendants should be, as far as possible, those who are protected by a previous illness; and it should be remembered that their clothes may convey the disease as they pass from the sick-room to other parts of the house, unless such over-clothing is changed before coming into contact with others. Only such books, papers, or toys should be allowed in the sick-room as may be afterwards burned; and food removed from the sick-room should not be eaten by other people.

Isolation from susceptible or unprotected persons should be maintained as long as the patient is believed to be infectious. The Medical Officers of Schools Association has adopted the following as the shortest times which should elapse between the appearance of the rash or other commencement and the return of the patient to his home or school: In rubella, seven days, provided there is no persistence of nasal or other symptoms; in measles, two weeks; in mumps, two weeks, including one clear week from the subsidence of all swelling; in diphtheria, four weeks, providing all discharges have ceased and no specific bacilli can be found in the nasal or pharyngeal mucus; in pertussis, six weeks, including two weeks free from spasmodic cough or whoop; in scarlatina, four weeks, provided convalescence is completed and there is no sore throat, discharge from ear or nose, suppurating gland, or eczematous patch. In small-pox and varicella, all scabs should have fallen off, and all sores should be healed.

**Disinfection.**—*Disinfection of the Excreta.*—In enteric fever the infective agent is contained in the urine and stools. These should be disinfected by thorough mixture with carbolic acid, ½ ounce of the crude acid to each pint of the evacuation, or by formalin 1 ounce to the pint, or by "chloride of lime" ½ ounce to the pint, and left to stand for two hours before being thrown away. It is better, if practicable, to burn the stools after mixture with sawdust and the addition of turpentine or naphtha, or to destroy them with strong mineral acid and bury them in the earth. The sputa should be also disinfected in enteric fever.

*Disinfection of the Clothing.*—Linen may be disinfected by prolonged soaking in 1 in 50 solution of lysol, and the linen should be boiled when it is being subsequently washed. Many local authorities remove clothing and bedding, etc., and return it after disinfection by steam; this is the only reliable method for bedding and clothes, etc. Woollen clothes must be exposed to a dry heat of 180° or 200°, and this is best done in special ovens constructed for the purpose, now in possession of the local sanitary authorities.

*Disinfection of the Patient.*—During the illness it is important to prevent discharges from the patient drying, and so being spread about as dust. All secretions, etc., are wiped away with moist swabs, which are put in some antiseptic solution. The mattress and pillow are protected by mackintosh or jaconette. After the patient has recovered, and before he mixes with his friends, he should have several warm baths and be rubbed with carbolic soap. In scarlet fever, the prolonged desquamation of the epidermis requires special treatment (*see* p. 32).

*Disinfection of the Room.*—After the patient has left the room in which he has been ill it requires to be thoroughly disinfected before it is occupied by others. This may be done with *formalin* or with *sulphurous acid gas.*

In using formalin, a special apparatus (the Alformant lamp or Lingner's glycoformal apparatus) is required; the room must be securely sealed and exposed to the vapour for at least four hours.

Sulphurous acid gas is obtained by burning sulphur. Three pounds of sulphur should be used for every 1,000 cubic feet of space in the room; it is placed in one or more earthenware vessels or pipkins, and each should rest on two or three bricks in a large pan of water. The chinks of the windows should be pasted up with slips of brown paper; the sulphur should be set alight, and the door should be closed and pasted up in the same way as the windows. After twenty-four hours the room may be entered, and the windows should be thrown wide open. Sulphur has the disadvantage of tarnishing metal work, and injuring pianos, sewing machines, etc., and these should be removed before the fumigation.

After gaseous disinfection the wall paper should be stripped off and burned, the floor and woodwork thoroughly scrubbed with carbolic soap, sanitas, formalin solution (2 per cent.), or izal (1 per cent.), and the ceiling whitewashed; or the floor, ceiling, walls, woodwork, and furniture may be thoroughly rubbed with bread, which must be afterwards burnt with all the fragments that drop about.

**Notification of Infectious Diseases.**—The diseases specified for notification in the Infectious Disease (Notification) Act, 1889, are small-pox, cholera, diphtheria, membranous croup, erysipelas, scarlatina or scarlet fever, typhus, typhoid, enteric, and relapsing, continued and puerperal fever. The diseases generally notifiable under regulations of the Local Government Board and Ministry of Health are plague, cerebro-spinal fever, acute poliomyelitis, acute encephalitis lethargica, acute polioencephalitis, tuberculosis, ophthalmia neonatorum, trench fever, dysentery, malaria, acute primary pneumonia and acute influenzal pneumonia. The following diseases are notifiable in particular districts either under (*a*) orders made by local authorities extending the Infectious Disease (Notification) Act, 1889, to the disease; (*b*) local Acts; or (*c*) special regulations made by the Minister: anthrax, glanders and hydrophobia, chicken-pox, whooping cough, "fever," summer diarrhœa of infants or gastro-epidemic enteritis, measles and German measles. Other diseases which have been made notifiable temporarily in particular districts include mumps, impetigo contagiosa and epidemic influenza.

**Immunity.**—Persons who are insusceptible to a particular disease are said to be immune, or to have immunity. Such immunity may be partial or complete, temporary or lifelong, innate or acquired. Of the conditions of *innate* or *natural immunity* little can be said. Some species of animals are immune towards the diseases from which other species suffer; among different races of men the nearest approach to an innate immunity is that of the negro towards yellow fever. In a given race, however, susceptibility varies very much. It is a fact observed every day that of a number of persons exposed to the contagion of a particular disease only a certain number will catch the illness; the rest will escape, even though they are not apparently protected by any of the methods to be mentioned below. Moreover, in those who are affected from the same exposure to contagion the disease may present very different degrees of severity. It is not

only matter of observation, but has been shown by experiment, that susceptibility to infectious diseases is increased by starvation, fatigue, cold, damp, unsuitable diet, and other conditions unfavourable to the general health; while a more local influence seems to be in operation when pneumonia or bronchitis is succeeded by a tuberculous invasion of the lungs. But the working of the law is not always clear; and it is quite certain that the fattest and most healthy-looking children of a family often suffer from, and succumb to, the most violent attacks of scarlatina, while others, apparently more delicate, may come off with a mild illness. A factor in the susceptibility to some diseases is the age of the patient, and this point will be referred to when these diseases are described.

*Hereditary disposition* may be alluded to here as the converse of innate immunity. Tuberculosis is looked upon as the best example of this occurrence. What is transmitted from parent to child is an undue susceptibility to infection. But there can be little doubt that in many cases the younger generation is directly or indirectly infected by the elder (*see* Tuberculosis).

*Acquired immunity* is that which is imparted in one or more ways to individuals previously susceptible. The most common cause of immunity towards an infectious disease is the fact that the individual has already had the disease. There are relatively few exceptions to the rule that scarlet fever, small-pox, chicken-pox, measles, and other such illnesses do not occur a second time in the same patient. This protection is probably closely related to the conditions which terminate a given infection in those who are suffering; the altered condition of the blood and tissues which destroys the micro-organisms persists afterwards for many years, or a lifetime, and antagonises the influence of any subsequent contagion of the same kind.

In contradistinction to this accidentally acquired immunity is *artificial immunity*, or the immunity intentionally or purposely acquired by the inoculation of the individual with some substance related to the virus or micro-organism which causes the disease. Artificial immunity may be *active* or *passive*.

In *active* or *direct immunity* the body cells or fluids are themselves stimulated by the inoculation to the production of substances (*antitoxins*) which will neutralise the toxins of the disease anticipated. The substance injected may be the micro-organisms in *living culture*, weakened in virulence or *attenuated*; or it may be the same micro-organisms in their full virulence, but in very small amount; or it may be the dead organisms; or it may be the bacterial products or toxins of the disease without the organisms.

The first of these methods has been employed in the cholera of chickens, in anthrax of sheep, and in the human subject in hydrophobia. The micro-organism or virus may be attenuated by growing in a current of oxygen or of air, by passing through the tissues of an animal, by growing at abnormal temperatures, and by growing in the presence of weak antiseptics. The method employed by Pasteur to prevent hydrophobia is described in the chapter on that disease.

The use of *vaccination* to protect against small-pox (*see* p. 42) may be regarded as another example, since *vaccinia* (or *cow-pox*) is almost certainly small-pox attenuated by transmission through the cow. Bacterial vaccines are successfully used to protect from enteric fever, cholera, and plague. In animals immunity has been obtained by feeding them with dead cultures of bacteria, or with their toxins.

In *passive* or *indirect immunity* the neutralising, and therefore protecting, substance is not provided by the body cells or fluids, but is supplied from without. A susceptible animal (*e.g.* horse) is first rendered immune by repeated injections of the virus of a disease, and the blood serum of this animal is then injected subcutaneously into the individual (man) it is desired to protect. If the animal has been rendered immune by injections of toxin, the resulting serum is *antitoxic*; if by the injection of bacteria, it is *antibacterial* or *antimicrobic*. In either case the effect of the injection has been to produce *anti-substances* in the horse's

serum which operate in the blood of the animal (man) to be protected; and the materials injected for the purpose of modifying the serum are therefore called *antigens*.

*Antitoxic sera* have been employed as cures; that is, the serum of immunised animals has been injected in order to neutralise the toxins of organisms already in the body, and causing symptoms (*serum therapeutics*), as, for instance, in diphtheria, tetanus, pneumonia, and septicæmia.

An important factor in protection from bacterial invasion is the process known as *phagocytosis*, or the destruction of the bacteria by the leucocytes and other cells of the body. The chief *phagocytes* are the large mononuclear and polymorphonuclear leucocytes, endothelial cells, and some tissue cells; they are attracted to the bacilli, and this attraction is called *chemiotaxis*.

Polymorphonuclear leucocytes and eosinophils are classed as *microphages*; hyaline leucocytes, endothelial cells, connective tissue cells, and other large cells are classed as *macrophages*. The former are more powerful in dealing with the bacteria of acute disease, the latter with those of chronic infections.

*Anti-bodies*.—It has been shown that the entrance into the body of toxins, whether contained in the bacteria (*endotoxins*) or produced from bacteria (*exotoxins*), will cause the formation of *antitoxins*. This is only one instance of a large group of similar occurrences, for the injection not only of bacteria and toxins, but of cells, blood corpuscles, ferments, and other bodies, will cause the formation of *anti-bodies* or *anti-substances*—that is, of substances which act adversely to, and destroy, the bacteria, cells, and other substances which have been injected. The anti-bodies which result from infection are formed in the spleen, lymph glands, and bone marrow, by leucocytes or by endothelial cells, or by both.

*Agglutinins* form another group of anti-substances the development of which is stimulated by bacterial infection, or even by injection of the red corpuscles of another animal (*hæmagglutinins*). If in certain diseases (enteric fever, Mediterranean fever, dysentery, cholera) the blood serum of a patient or convalescent is mixed with cultures of the organism of the same disease, in a short time the bacilli are seen under the microscope to lose all active movements, and to become densely aggregated together (*agglutination*, or *clumping*). The same effect may be obvious to the naked eye if the serum and a culture fluid be mixed in a test-tube, when after a time precipitation of the bacilli takes place, leaving the upper part of the fluid clear (*sedimentation*). These facts form the basis of the diagnostic method known as *Widal's test* (*see* Enteric Fever).

The action of agglutinins is not absolutely specific. Thus the typhoid agglutinin will clump not only the *Bacillus typhosus*, but also the paratyphoid bacilli and the *Bacillus coli*. The results vary with the time employed and with the extent of dilution of the serum.

Agglutinins may also be obtained artificially, for the blood of an animal inoculated with sublethal doses of a given bacillus will acquire *agglutinative* properties towards the bacillus which has been injected (Bordet-Durham reaction).

*Precipitins* are similar substances, developed in the serum of animals which have been inoculated with bacterial culture fluids, albumose, milk, etc. The serum containing them precipitates the corresponding culture fluid, or a solution of the corresponding organic substance which has been used for inoculation.

The actions of *anti-bodies* in regard to bacteria, blood corpuscles, leucocytes, kidney cells, and other animal cells are known as *bacteriolysis*, *hæmolysis*, or *cytolysis*. They are for the most part specific, as in the former instance; that is, if certain bacteria are inoculated into an animal the serum subsequently has a destructive effect on the same kind of bacteria only; if rabbit's blood corpuscles are injected into a guinea-pig, the guinea-pig's serum will afterwards dissolve (or lake) rabbit's blood outside the body; if the liver cells are inoculated, the serum will dissolve liver cells, and so on. These different forms of cytolysis and hæmolysis are dependent not only upon the anti-body produced in the

process (also known as *immune body*), but they also require the assistance of an enzyme which exists normally in the serum, and has been called the *complement*. Its presence is shown by the fact that the blood-dissolving power of a hæmolytic guinea-pig's serum may be neutralised by a temperature of 55° to 60° C., which destroys the complement; but it can be restored by the addition of serum from a healthy guinea-pig. The complement is probably a product of the leucocyte, and is identical with the *cytase* of Metchnikoff and the *alexine* of Buchner.

The facts connected with hæmolysis and the action of the complement are utilised in some important diagnostic methods (*see* Syphilis).

Another element in the protective power of the blood is the existence of *opsonins*, bodies which act on the bacteria so that they are more readily digested by the leucocytes (*opsono*, I cater for, or prepare food). The opsonic power of the serum is measured by the number of bacilli which the phagocytic leucocytes can take up (Wright and Douglas).

**Anaphylaxis.**—In close relation to immunity and the operation of toxins are the phenomena known under the name of *anaphylaxis*, or *supersensitiveness*. This was discovered by Richet in 1902. He had injected a dog with a small dose of a poisonous protein from a sea-anemone, with the production of evanescent symptoms. Some weeks later he injected a second dose, expecting to find that some degree of immunity had been established, but instead there was a violent intoxication, and the dog succumbed. The name "anaphylaxis" means the opposite of immunity or "phylaxis." The explanation is as follows: Any foreign protein (antigen) injected into an animal causes the production of anti-bodies, which have the nature of *precipitins* (*see* p. 16). While these remain in the blood they produce immunity by precipitating further doses of the antigen. But after a time they largely disappear from the blood and enter the tissues. At this stage there will not be enough anti-body in the blood to precipitate the antigen, if injected, and so the latter will invade the tissues; but here the meeting of the two produces a violent reaction, viz. anaphylactic shock (**1**). One of the most striking results in the guinea-pig is contraction of plain muscle, which shows itself chiefly in a violent asthmatic attack. The injection of many poisonous substances into animals produces symptoms somewhat resembling anaphylactic shock, and it has been suggested that the latter is really due to a poison, "anaphylatoxin," produced when the second dose of antigen is given; but a careful comparison of the two sets of symptoms shows that there is a difference. In medicine the term "anaphylaxis" has been used loosely to describe the sudden onset of symptoms, *i.e.* dyspnœa, œdema, fall of blood pressure, collapse, vomiting, or other gastro-intestinal symptoms, convulsions, and the features of the allergic state (*see* p. 224), which are often preceded by certain changes in the blood, called the "hæmoclasic crisis" by French authors (**3**) (*see* p. 226). But there is usually no evidence that the body has been previously sensitised to foreign substances, as in anaphylaxis, and it is much more correct to regard the symptoms of the allergic state as due to the direct action of some poison, especially as the injection of poisons into animals, *e.g.* histamine, has been found to cause such symptoms.

When an immunised serum is used for the treatment of diphtheria or other disease (*see* p. 15) the patient sometimes (in about 7 per cent. of the cases) suffers from toxic symptoms, which develop in from seven to twelve days, and are due to the serum injected, and not to the anti-bodies in it. They consist of urticarial swelling at the site of injection, with swelling of the associated lymphatic glands; spread of the eruption—urticarial, circinate, morbilliform or scarlatiniform—to the rest of the body; glandular swelling in other parts; œdema of the face, body or glottis; pains in the joints; slight pyrexia; and occasionally albuminuria, diarrhœa or bronchitis. Leucopenia of the polymorphonuclear cells also occurs following a leucocytosis in the stage of incubation. This is known as

" serum disease." The symptoms pass off in a day or two. It follows, from what has already been said, that it is doubtful whether serum disease is really " anaphylactic " in origin.

These, or similar symptoms, are still more likely to occur after a second injection and are then certainly anaphylactic. The anaphylaxis requires from six to twelve days for its development; hence a second injection before the period of six days usually produces no result. If the interval between the two injections is from twelve days to six or eight weeks (sometimes six months), two types of reaction may occur: (1) the *immediate reaction*, in which symptoms occur *shortly after* the second injection or within twenty-four hours, and pass off quickly, the general symptoms in twenty-four hours, the local in two or three days. These symptoms are œdema at the site of injection, pyrexia, swelling of glands, urticaria, œdema of the face and leucopenia. Occasionally serious symptoms (so-called *anaphylactic shock*) may occur within half an hour of the injection. The patient complains of difficulty in breathing, the chest becomes expanded and rigid, the face becomes congested, and the mucous membranes are cyanosed. Relief comes in from fifteen to thirty minutes. Patients have died in the attack. (2) The *accelerated* reaction, which comes on four to eight days after the second injection. It is called *accelerated* because it comes on sooner than the symptoms which follow a *single* injection. The symptoms are similar to those which occur after a single large dose (serum disease), but are generally more acute, passing off in twelve to eighteen hours.

The danger of the occurrence of anaphylactic shock should not prevent the administration of serum if it is indicated on therapeutic grounds. It is impossible to tell if a patient has become sensitised by a previous dose of serum; but if it is thought that such may be the case, he must be *desensitised* if the second injection is made. The best plan is to inject $\frac{1}{2}$ or 1 c.c. of the serum five or six hours before the chief injection is to be made. If greater rapidity is desirable, such a preliminary injection may be followed in five or ten minutes by a rather larger one, and so on at similar intervals with increasing doses until the whole has been given. The treatment of anaphylactic shock consists in intravenous or intramuscular injections of pituitary extract, atropine or adrenin, while pure oxygen is also administered, if necessary, by a nasal catheter. Chloral hydrate may also be given.

## PYREXIA

The terms *fever* and *pyrexia* are not always used in the same sense, pyrexia being sometimes limited to the mere fact that the body temperature is elevated, while by fever is understood the rise of temperature together with all the other bodily disturbances which usually accompany it.

**Range of Temperature.**—In health, with the subject at rest in bed, the *mouth* temperature varies from about 97° F. in the early morning up to 98·4° F. in the middle of the afternoon. The *rectal* temperature varies between 97° and 99·2° F. under the same conditions. Muscular exercise may raise the rectal temperature by 2° F., while the mouth temperature remains the same or actually becomes lower. This is because the sweating and increased respiration tend to cool the mouth. The rectal temperature, on the other hand, approximates more closely to the internal temperature of the body. The temperature of the urine as discharged from the meatus is always slightly above the rectal temperature. The temperature of the axilla is about the same as that of the mouth, being somewhere intermediate between the skin and body temperature. In disease the temperature ranges from 93·3° (35·5° C.), or even lower, to 110° or 111° (43·8° C.). Temperatures of 116° and 122° have been recorded, but considerable doubt attaches to their genuineness.

Many terms have been used to denote the different degrees of temperature

above or below normal; Wunderlich gives eleven in all, but the following are all that are practically wanted :—

| | |
|---|---|
| Collapse temperatures . . . | 92·3° (33·5° C.), or lower, to 96° (35·5° C.). |
| Subnormal ,, . . . | 96° to 97·5° (36·4° C.). |
| Normal ,, . . . | 97·5° to 99° (37·2° C.). |
| Slight or moderate pyrexia . . | from 99° to 101° (38·3° C.) in the morning, or 102·5° (39° C.) in the evening. |
| Severe pyrexia . . . . | from 101° to 103° (39·4° C.) in the morning, or 105° (40 5° C.) in the evening. |
| Hyperpyrexia. . . . . | above 105°. |

These terms must be used with discretion, because the temperature varies according to where it is taken and according to the time of day, so that what might be called a subnormal temperature in the afternoon would be quite normal in the early hours of the morning.

**Registration of Temperature.**—The temperature of the body is taken for ordinary clinical purposes by means of the clinical mercurial thermometer, which registers the temperature after it is removed from the body by a portion of the mercurial column being prevented from returning into the bulb of the instrument. The bulb of the instrument may be placed in the axilla, the groin, the mouth, or the rectum.

In the two former situations it is necessary to see that there is complete contact of the skin with the bulb, and it must remain there sufficiently long for the surface of the skin to attain the temperature of the body generally; from one to three or five minutes suffice, according to the sensitiveness of the thermometer. In the mouth the bulb should be placed under the tongue, and the stem must be grasped by the lips. When the rectal temperature is taken, the bulb is introduced for 1½ inches. The result can be depended on, but it is obviously a method that is not always convenient.

In consequence of daily variations, both in health and disease, it is desirable to record the temperatures at least twice a day; the best times would be 5 or 6 A.M. and 5 or 6 P.M., so that the lowest and highest temperatures should be observed. Social arrangements do not usually allow of this in slight cases of illness, so that 10 A.M. and 6 to 9 P.M. are more often the times selected; but it must be remembered that at 10 A.M. the temperature is already rising, and that after 7 P.M. the maximum is generally passed. In severe illnesses, like typhoid fever, pneumonia, etc., the temperature should be taken at least every four hours, so that the daily variations may be more closely watched; and it should in all cases be recorded on a *chart*, with a dot for each observation and lines drawn from dot to dot. It is important to state on the chart whether the temperature has been taken in the mouth, axilla or rectum.

## CONDITIONS ASSOCIATED WITH PYREXIA

Pyrexia, fever, or febrile reaction is accompanied by many other disturbances besides elevation of temperature; indeed, every function of the body is more or less disturbed whenever the temperature is raised for more than a very short time. That this is in part at least a direct result of the high temperature can be shown by experiment; but in nearly all cases of its production by disease it must be recognised that toxins are circulating in the body, and that they are probably the cause of numerous conditions formerly attributed to the high temperature alone.

*Skin.*—It is hot to the touch, sometimes intensely so, and generally dry, but it may be moist, usually when the temperature is falling. In some diseases profuse sweats may occur, which sometimes perceptibly, sometimes scarcely at all, reduce the temperature. Such perspirations may cause an eruption of sudamina or miliaria. The colour of the skin over the body is generally

normal, unless there are eruptions, such as miliaria, or the specific rashes of scarlatina, measles, typhus, and others. But the face is often flushed, especially at the commencement of a fever; often the cheeks and lips are flushed, and the face is elsewhere pale; later on, with a failing circulation, the face becomes deeply congested or livid, and the extremities show the same change.

The petechiæ and subcutaneous hæmorrhages which occur in the most malignant forms of infectious disorder (small-pox, typhus, measles, scarlatina) result, no doubt, from the action of virulent toxins upon the capillary walls.

*Alimentary System.*—The tongue becomes furred; generally at first the fur is white, and the tongue is still moist; then the tongue becomes dry: the fur peels from the edges or tip, and shows the bright red tongue beneath. Later on the tongue becomes very dry, stiff, hard, dirty brown in colour, fissured on the surface, and caked with dried remains of saliva, buccal secretions, food, and sometimes blood mixed with epithelium, fungoid growths and bacteria which are allowed to accumulate in the passive state of the organs of mastication. In this stage also the gums are covered with a similar collection, which is called *sordes*. Loss of appetite, or anorexia, is one of the first signs of fever; sometimes sickness is present, and in all cases digestion is feeble. The bowels are usually constipated. The spleen is often slightly, in certain diseases very much, enlarged and tender.

*Circulation.*—The heart's action is quickened, at first excited, then feebler. The pulse ranges from eighty to 120 or more. It is at first full, bounding and firm; it soon becomes softer and dicrotic. In later stages, as the heart becomes more feeble, it is quick, very small, very compressible, running or flickering. With progressive weakening of the heart's action the first sound becomes faint or inaudible, and the impulse may be detected outside the nipple, showing that the heart is becoming dilated.

*Respiration.*—This is quickened in proportion to the pulse and the rise of temperature; it may rise to thirty or forty in the minute. When the illness has lasted some time, the bases of the lungs become congested (hypostatic congestion), and the respiratory movements of the upper part of the chest in front are exaggerated.

*Kidneys.*—In consequence of the loss of aqueous vapour through the skin and lungs the urine in fever is scanty; and, as a direct result of this, it is high-coloured and deposits a brick-red sediment of urates on cooling. In severe febrile illnesses there may be a small quantity of albumin.

*Metabolism.*—Pyrexia involves increased heat production, and so increased metabolism in the body. This shows itself in the fact that the respiratory exchange (intake of oxygen and output of $CO_2$) is increased. There may also be increased wastage of the body tissues, with increased nitrogen excretion in the urine. This may be partly due to the poisonous action of toxins on the body cells; but the nitrogen loss can be largely prevented by administering sufficient amounts of non-nitrogenous foods, especially carbohydrates. The body can then utilise these and so spare its own proteins. A high body temperature artificially produced in a normal subject does not cause a breakdown of body proteins if there are sufficient non-nitrogenous foodstuffs available (4).

*Nervous System.*—Headache is common at the commencement of pyrexia; there is also a heavy feeling, dulness, or disinclination to think or make any mental effort; after a time the patient not only does not wish to, but cannot, exert the intellect; he becomes drowsy, and when he drops off to sleep, begins to talk. Later on he is delirious without really sleeping, and the delirium may be muttering, and is occasionally maniacal, the patient getting out of bed, struggling with his nurses or attendants, or jumping out of the window. In the last stages there is profound unconsciousness or coma. In the earlier stage of coma the patient frequently picks with his fingers at the bedclothes, or catches at imaginary objects in the air in front of him. The disturbance of the

muscular system shows itself in general bodily weakness, tremor of tongue or limbs when they are moved, and twitching of the muscles (*subsultus tendinum*), while the relaxation of the sphincter ani allows the uncontrolled passage (incontinence) of fæces, and the diminished visceral sensations lead to retention of urine and dangerous distension of the bladder.

**Daily Variations.**—The temperature in fever shows daily fluctuations, which are, as a rule, similar to those observed in health; that is to say, the temperature is lower in the morning and higher in the evening; the lowest point is commonly reached about midnight or 2 A.M., and the highest from 4 to 6 P.M. Occasionally the reverse obtains; the temperature is highest in the morning, lowest in the evening—*typus inversus*. The pulse and respiration rise and fall with the temperature, and the general discomfort of the patient varies in the same manner.

**Varieties of Pyrexia.**—The pyrexia which accompanies or constitutes an illness may be one of three kinds: it may be *continuous*, *remittent*, or *intermittent* (Fig. 1). A continuous fever is one in which the temperature is constantly above the normal, and the differences between the morning and evening temperatures never exceed the variation in health—that is, one and a half degrees. A remittent

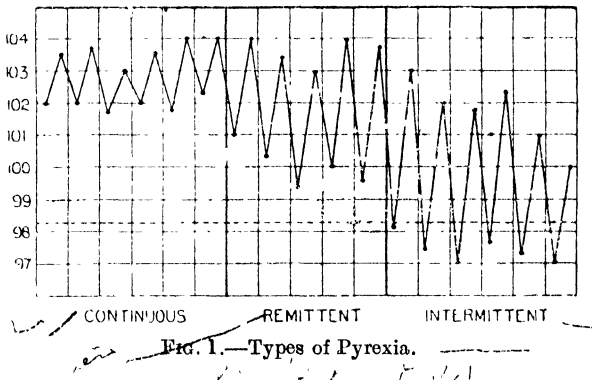



Fig. 1.—Types of Pyrexia.

fever is also one in which the temperature is constantly above the normal, but the difference exceeds one and a half degrees. Thus in the morning it may fall two or three degrees, but never reaches the normal. An intermittent pyrexia is one in which the morning temperature falls to the normal or below it, while the evening rise is two, three or more degrees above it. This last form is sometimes called *hectic fever*.

In some cases of illness there may be at one time a continuous pyrexia, at another a remittent, and at a third time an intermittent pyrexia, as, for instance, in enteric fever and in phthisis.

**Course of Pyrexia.**—In many cases of fever a very definite course may be recognised, the beginning and the end being marked by certain characteristic phenomena.

The fever begins with a sensation of cold—it may be a mere chilly feeling, so that the patient seeks the fire, or he shudders or shivers, and finally he may have a definite *rigor*. This is an attack of shivering, in which the patient trembles all over, his teeth chatter, he feels intensely cold, his face is pinched, and the nose, ears and finger tips are livid (*see also* Clinical Examination of the Nervous System; Motor Symptoms). But though the surface is cold, the internal parts are hot, and the thermometer will show that the temperature is constantly rising from the first. The rigor may last from a few minutes to half an hour, an hour, or more. In young children rigors do not generally occur, but their place is sometimes taken by a convulsion.

The second stage of the fever is the *fastigium*, in which the skin is hot and the various phenomena already recorded are present.

In some fevers the temperature may rise to its maximum or *acme* just before the onset of the third stage.

The third stage is that of *defervescence* or decline, which occurs either by *crisis* or *lysis*. In *crisis* there is a rapid fall of temperature to the normal within twelve to thirty-six hours; it is sometimes accompanied by profuse sweating, sometimes by diarrhœa (critical sweat or diarrhœa). In *lysis* the temperature falls more slowly, taking three or four days to reach the normal.

For some days after a pyrexia the temperature may be unusually low (subnormal), *e.g.* 97° or 96° in the morning, and from this time the period of *convalescence* commences.

Rigor, fastigium, and crisis occur typically in some forms of malaria, and may then all be completed within a period of six to twelve hours.

**Death in Fevers.**—This results, first, either in a few days, from the virulence of the toxins acting upon the nervous system and involving all the functions of the body, or from their continued action over a longer period; secondly, from hyperpyrexia in a small number of instances only, though it is not uncommon for the temperature to rise very high when the patient is at the point of death from other causes; thirdly, from cardiac failure; fourthly, from pulmonary complications.

Certain anatomical changes, probably chiefly due to bacterial infection, are common to nearly all deaths in high fever. The red corpuscles are diminished, and leucocytosis is common. Small petechiæ or hæmorrhages are found under the serous membranes of the pleura and pericardium. The solid organs—liver, spleen, and kidneys—are large and soft, and the kidneys and liver show, under the microscope, cloudy swelling with some granular change or fatty degeneration in their secreting cells. In hyperpyrexia, the cells of the central nervous system stain diffusely, and Nissl's granules are absent. The lipoids in the cortex of the suprarenals are discharged.

The muscles may be soft and friable, or may show the degeneration which was first described by Zenker, and is now regarded as a coagulative necrosis. In this condition the muscular fibres are converted into a homogeneous, colourless, waxy-looking material, forming cylinders, which break up into fragments, and finally crumble into a granular detritus; it is sometimes accompanied by hæmorrhage. This change is most common in the abductors of the thighs, in the recti abdominis, in the diaphragm, and in the muscles of the tongue.

**The Cause of Pyrexia.**—The maintenance of the temperature of the body at its normal standard is dependent upon two factors, *Heat production* or *Thermogenesis* and *Heat loss* or *Thermolysis*. In fever there is increased heat production. During a malarial rigor the heat production has been found to increase by 200 per cent., while the heat loss remains the same. During this time, about an hour perhaps, the body temperature is rising. When the high temperature is established the heat production falls to about 80 per cent. above normal, while the heat loss increases to cause a balance. In other fevers, unaccompanied by rigor, during the rise of temperature, both the heat production and heat loss rise step by step, the former being, of course, greater than the latter. When a high temperature is established, the heat production depends on the temperature and is about the same as all fevers, increasing by 7·2 per cent. for each degree F. Curiously enough, the amount of the rise is about the same as that of chemical reactions *in vitro* (5). The regulation of temperature is a function of the central nervous system, although it is probable that a single special "heat-regulating centre" does not exist. Heat production takes place in the organs of the body, particularly the skeletal muscles, and in certain cases this over-production of heat may be due to stimulation of efferent nerves to these structures from the central nervous system, either from irritation of the latter or from reflex irritation from

the periphery. Heat loss occurs to the extent of 87 per cent. from the skin by radiation, convection, conduction, and evaporation, to the extent of about 10 per cent. from the lungs, and to the extent of about 2 per cent. from the urine and fæces. The nerve apparatus concerned is chiefly the vasomotor system, by which the circulation through the skin is affected. If the skin becomes hot, the vessels dilate, so that the warm blood comes to the surface of the body. Sweating enormously increases the heat loss owing to the evaporation that occurs. With high fever respiration is quickened, and the loss of heat from the pulmonary surface is proportionately increased.

Febrile disorders have been grouped as follows: (1) diseases due to general infection, or *specific fevers*: (2) fevers occasioned by local inflammation, formerly called *symptomatic fevers*: and (3) fevers which arise from local disease of, or are associated with even functional failure of, the nervous system: the last have been spoken of as *neurotic fevers*.

To take the third group first: it has been shown by experiment and by cases of disease that lesions involving the cortex cerebri, or the nucleus caudatus, or the fibres proceeding thence to the muscles, may be followed by pyrexia of a marked kind. It is seen, for instance, occasionally in cases of cerebral hæmorrhage, of cerebral tumour, and of meningitis. This is less often a continuous pyrexia than an occasional rise of temperature. The high temperatures caused by peripheral irritation, such as the passage of gall-stones, may be attributed to stimulation through afferent nerves of the same centres, and reflex action there operating. In cases of disease and injury of the spinal cord, patients may develop an excessively high or excessively low temperature. This is because the body is cut off from the regulating centres in the brain and resembles to some extent a cold-blooded animal. If, owing to the presence of hot-water bottles and blankets, the temperature rises, metabolism increases, and the higher the temperature the greater the metabolism; a vicious circle is established, because there is no reflex increase in the heat loss. Similarly if the body gets too cold the metabolism diminishes, and the body becomes progressively colder for the same reason. It is very important in such cases to regulate carefully the temperature of the surroundings.

There are other febrile disturbances for which no local cause can be found, nor any association with the groups which follow. These are commonly regarded as functional, hysterical, or *neurotic*, and may possibly be due to alterations in the centres in the brain. Besides being very variable in duration, they are often unaccompanied by the disturbances characteristic of fever, such as anorexia, furred tongue, and loss of flesh.

The *specific fevers* are, as already stated, dependent upon the entrance into the body of bacteria, or micro-organisms, which multiply in the blood or tissues; and the pyrexia may be regarded as due to the operation of bacterial toxins on the brain. Some other poisons, vegetable and animal, also determine changes in the temperature of the body.

The pyrexia associated with local inflammation (*symptomatic fever*) is no doubt due to the absorption of the toxins of the micro-organisms (streptococci, staphylococci) concerned.

**Prolonged Pyrexia.**—While we recognise that the duration of a pyrexia is determined mostly by the infection with which it is associated, and may therefore be from a few hours to several months, it may be useful here to mention the diseases which are most commonly found to be the causes of a pyrexia prolonged for several weeks or months. They are the following: typhoid and paratyphoid fevers, Mediterranean fever, malarial fevers, tuberculosis, septicæmia, bacilluria, malignant endocarditis, pernicious anæmia, leukæmia. Among less common conditions are syphilis, cirrhosis of the liver, Hodgkin's disease, and malignant growths.

**Subnormal Temperatures.**—The subject of abnormally low tempera-

tures cannot properly be separated from the consideration of pyrexia, and the following list of the causes of subnormal temperatures, given by Janssen, may be found useful: (1) Direct withdrawal of heat from the body, as in cases of exposure of unconscious or drunken persons in very cold atmospheres, or of immersion in very cold water. (2) Loss of great quantities of fluid from the body, as in severe diarrhœa, cholera, enteritis, or profuse hæmorrhage. (3) Conditions of cachexia and inanition, such as carcinoma of the various parts of the alimentary canal, severe diabetes, pernicious anæmia, convalescence from febrile affections, and many chronic mental diseases. (4) Grave circulatory disturbances, as cardiac failure and stenosis of the respiratory passages. (5) Various diseases of the central nervous system, the onset of cerebral hæmorrhage and embolism, some cases of cerebral tumour, and general paralysis of the insane. (6) Irritation of sensory nerves, as in intestinal strangulation, perforations of the intestine, and surgical operations. (7) Extensive skin affections, such as universal eczema, and large burns. (8) After fevers, when the temperature may long remain subnormal, or in the course of pyæmia. (9) Poisoning by phosphorus, atropine, morphine, carbolic acid, and alcohol; uræmia and diabetic coma.

**General Treatment of Diseases attended with Pyrexia.**—The treatment of each particular case will depend more or less upon the cause; but the general principles of treatment are as follows: The patient should be at rest in bed, in a thoroughly ventilated apartment, from which all mirrors, striking pictures, or other objects likely to excite him in the event of his becoming delirious have been removed. He should be watched day and night, preferably by trained nurses, and should be kept scrupulously clean. He should also be kept cool, the amount of clothes being lessened if the fever is very high. A distinct lowering of temperature may be sometimes effected in this way, a point to be remembered all the more as the tendency of the patient's friends is to heap clothes upon him to prevent his "catching cold." The extremities, however, must be carefully watched, and specially covered or warmed if necessary.

At the present day there is not the same tendency as previously to restrict the amount of food allowed the patient. Pyrexia is associated with increased metabolism, so that 2,000 calories or more may be allowed an adult if the pyrexia is long continued. It is essential that the diet should be easily digested, and it should be to the liking of the patient. Milk (2 or 3 pints in the day) will form an important element in the diet, and lactose or extract of malt may be added, so as to increase the calorie value. In cases where milk disagrees, or is felt to load the stomach, or is rejected, it may be mixed with half its bulk, or an equal quantity, of barley water or soda water; or it may be peptonised or predigested by warming for a little time with liquor pancreaticus. Arrowroot, cornflour, blanc-mange, custards, milk puddings, bread and butter, may be allowed, and fish and minced chicken if the patient can tolerate them. It must be remembered that beef tea, mutton broth, chicken broth and veal broth, although they may act excellently in stimulating the appetite, have practically no value as foods.

The application of an ice bag to the head is often useful, especially when headache is a prominent symptom.

Frequent attention to the mouth is necessary, since the accumulation therein of secretions and sordes is likely to be followed by parotitis and other complications. The mouth should be washed out after each feeding, or more frequently, according to the amount of the secretions, with an antiseptic solution, such as a solution of thymol in water or 2 per cent. boric acid.

When the intensity of the toxæmia reaches a certain point, and the patient's organic sensations are dulled, the bladder should be carefully watched, to provide against retention of urine. Fulness and dulness on percussion above the pubes are the signs of a full bladder, and the mere statement of the attendants that the patient is passing his urine must not be accepted as a proof that no urine is being

retained. The frequent involuntary discharge of small quantities of urine is, indeed, the result of the bladder being already distended; and even the discharge of larger quantities at the request of the nurse may still leave a pint or more in the bladder, which will be revealed on manual examination. The catheter must then be used twice daily as long as the disability persists.

In some cases of pyrexia the temperature has been directly dealt with by methods known as *antipyretic*. It must be distinctly understood that such treatment will not lessen the duration of the illness; that in many illnesses the temperature will of itself fall in a few hours, that is, in the early morning; that in temperate climates there is very rarely (except in rheumatic hyperpyrexia) any danger that it will rise to a height which can be directly fatal; and that any considerable lowering of the temperature by artificial means falsifies to a certain extent the information which the temperature gives to the physician as to the course of the disease. But most important of all, pyrexia is to be looked upon as a defensive mechanism on the part of the body against the infecting micro-organisms, and hence it should not be interfered with without careful consideration. Such treatment, however, often increases the comfort of the patient during the time that each successive dose or application is in operation, and possibly, in some diseases, diminishes the risk of damage to the viscera.

Antipyretic methods may be divided into three groups:

**Milder Refrigerants.**—These are the ordinary saline remedies—citrate of potassium, acetate of ammonium, dilute acids—which were formerly given in every fever, but have very little influence.

**Stronger Antipyretic Drugs.**—These drugs are very little employed at the present time for their purely antipyretic action. If one of them is given in a single dose to a patient with a temperature of 103° or 104°, the temperature falls within two or three hours to normal or even lower, but it rises again in six or seven hours to a height not much different, if at all, from what it would have reached had no antipyretic been given. The following have been most often employed: quinine sulphate, 20 to 30 grains; salicin, 30 grains; salicylic acid, 20 grains; antipyrin (phenazone), 15 grains; antifebrin (acetanilide), 2 to 5 grains; phenacetin, 5 to 10 grains. The last three are the most certain in their antipyretic action, but in doses beyond the limit stated these drugs are apt, especially the last two, to cause alarming cyanosis and collapse.

**External Application of Cold.**—This may be done in several ways: the cold bath; the wet pack; sponging; ice applications; Leiter's coils. Though more troublesome than the administration of drugs, its use can be better controlled, and there is less risk of harm to the patient.

*Cold Bath.*—This has been largely used in the treatment of enteric fever. The temperature is taken every three hours, and whenever it is found at any of these periodical observations to be 102° F. or higher, the patient is placed in a bath of a temperature of 70° F., in which he remains for ten or fifteen minutes, according to its effect upon him. He is then removed, lightly dried, and replaced in bed. The temperature will then be generally found to have fallen to 99°, 98°, or even lower. The system is open to modifications: the observations may be made less frequently, the bath may be only used when the body heat is 102·5°, or 103°, or 103·5°, and the temperature of the bath may be as low as 60° or as high as 80° or 90°. Sometimes the patient is put in the bath at a temperature of 90°, and ice is then introduced to bring down the heat to 75° or 70°. It is obvious that the greater the number of baths, and the lower their temperature, the greater will be their effect upon the mean body heat. Continuous immersion has been also successfully employed.

*Wet Pack.*—A sheet is wrung out of ice-cold water and wrapped round the patient for ten or fifteen minutes, the application being made under the same conditions of bodily temperature as are directed for the bath.

*Sponging.*—The body is uncovered and sponged over with cold or ice-cold

water for from seven to ten or fifteen minutes. This method is not generally so effective as the two former; the temperature commonly falls from one and a half to two degrees.

*Ice-bags.*—These may be placed on the chest or abdomen for varying periods, or hung within a cradle placed over the patient.

**Stimulants.**—In all febrile illnesses a stage may be reached when the heart's action and the nervous system are so profoundly affected that some kind of artificial stimulation is required; but although the signs indicative of such a stage are, as a rule, severe in proportion to the elevation of the temperature, they are due not so much to the fever itself as to the toxins which circulate in the blood, and are themselves the cause of the pyrexia.

The results of such cardiac failure and nervous prostration are quick and feeble pulse, inaudible first sound of the heart, irregular action of the heart, cyanosis, congestion and œdema of the bases of the lungs, dry tremulous tongue, muscular tremor, sleeplessness, and delirium.

The simplest method of stimulation, and one long in vogue, is the administration of alcohol in the form of brandy or whisky; the quantity may be from 2 to 6 or 8 ounces in the twenty-four hours. But the larger amounts must not be continued for many days; and especially in prolonged illnesses, like typhoid fever, the effects of this drug must be carefully watched, since an excessive amount will keep up a quick pulse and a drowsy muttering delirium, deceptively like the very condition for which it was originally given.

If the delirium assumes a maniacal form, it is very likely to be aggravated by alcohol.

In more recent times there has been an increasing tendency to prefer other modes of stimulation, which may often be used with success. Such are the following: the subcutaneous injection of liquor strychninæ in doses of 2, 3 or 5 minims twice a day, or more frequently as occasion requires; the internal administration of tincture of digitalis in doses of 15 or 20 minims three times a day; the intramuscular injection of the double salt of caffein and sodium salicylate or of caffein and sodium benzoate in the dose of 5 grains for an adult; the intramuscular injection of camphor in 10 or 15 or 20 per cent. solution in olive oil (or oil of sesame), the dose of camphor for an adult being from 3 to 5 grains; the intramuscular injection of adrenalin.

In any case the result of each dose will be the guide to the time at which the next should be given.

## HEAT STROKE

*(Sunstroke, Heat Apoplexy, Thermic Fever)*

During spells of unusually hot weather in temperate climates individuals, especially those whose health is already impaired, are liable to certain symptoms usually known as heat exhaustion. The patient suffers from faintness, vertigo, nausea or vomiting, and may occasionally lose consciousness. The pulse is rapid and thready, and the breathing shallow. The temperature of the body is subnormal or only very slightly raised. As a rule, if the patient is placed in a cool place and stimulated by dashing cold water on the face and neck, the symptoms pass off. Occasionally in the aged or debilitated death may occur from cardiac failure.

In tropical countries, however, the effects of heat may manifest themselves in symptoms of much greater severity, usually known as heat stroke. Exposure to the sun's rays will produce heat stroke, especially if the head or the back of the neck are exposed; but heat stroke may develop independently of direct exposure to the sun. Any individual, especially if already suffering from some other disease, who is subjected to a high air temperature, may develop heat stroke. The disease is rare until the external temperature rises to about 110° F., and is more frequent when the humidity of the air is high.

The term *sunstroke* is often used to express an effect that exposure to the sun's actinic rays is supposed to produce apart from a rise of body temperature. However, the evidence is against this supposition.

**Ætiology.**—Undoubtedly the most important predisposing cause of heat stroke is a disturbance of the normal physiological activities by malaria, enteritis, or other disease. Patients already in hospital for some such condition frequently develop heat stroke if the weather is hot. Next in importance among predisposing causes is alcoholism, especially if spirits are taken during the heat of the day. Constipation also is a very frequent antecedent of heat stroke. Heavy clothing or clothes that are tightly fitting, over-exertion and fatigue are also important predisposing factors.

**Anatomical Changes.**—Very little is noted in the cases of rapid death from heat exhaustion. In the hyperpyrexial variety the blood remains fluid; the left ventricle is contracted from coagulation of its myosin, and the right ventricle is dilated; the lungs are intensely engorged, with hæmorrhage under the pleura, the cerebral meninges are congested, and there are hæmorrhages in the white matter of the brain. The nerve cells show coagulative necrosis with disappearance of the Nissl bodies, and swollen chromatolytic nuclei.

**Pathology.**—A high external temperature acting on an individual whose normal physiological functions are deranged by disease causes a temporary breakdown of the mechanism for heat regulation, and in consequence the body temperature tends to rise. As this occurs metabolism is increased, and a vicious circle is established until the temperature becomes incompatible with life. It has been shown experimentally that at a temperature of 108° F. the neuroglobulin of the nervous system quickly becomes coagulated.

It is, however, thought by some authorities that high atmospheric temperature alone is not sufficient to cause heat stroke. They regard the temperature as probably due to a specific infection by some unknown organism, which demands for its development a high atmospheric temperature. No such organism has been isolated, and the theory is based chiefly on arguments from the epidemiology of the disease.

**Symptoms.**—The onset is usually sudden. The patient feels restless, giddy, and dyspnœic, and may suffer from nausea or vomiting. There is often frequency of micturition, and sweating is diminished or ceases altogether. At this stage the rectal temperature is rising. Very soon after these premonitory symptoms the patient becomes unconscious, with laboured, stertorous breathing and rapid pulse. The skin is absolutely dry and feels hot to the touch. The face becomes congested in appearance, or there may be marked cyanosis. Violent convulsions occur frequently with incontinence of urine and fæces. The rectal temperature at this stage may range from 108° to 112° F. The patient, if left untreated, dies in coma with gradual weakening of the pulse and respiratory failure. On the other hand, if the temperature is reduced by the methods described below, consciousness is regained within an hour or less, and, though the temperature may remain somewhat raised for a few days, convalescence is gradually established. Persistent headaches, increased susceptibility to high atmospheric temperature, and sometimes insanity, are not infrequent sequelæ to a severe attack of heat stroke.

**Prognosis.**—This depends almost entirely on the rapidity with which treatment is started and on the facilities available for really efficient treatment. In comatose patients with temperatures over 110° F. the prognosis is always grave. Even if the temperature is reduced the patient, especially if debilitated, may die of collapse.

**Treatment.**—With the more severe forms of heat stroke treatment must be both immediate and vigorous. The patient is stripped naked and placed on a waterproof sheet, preferably on a wire mattress, and is rubbed over with large blocks of ice or sprayed with ice-cold water. The rectal temperature must be

taken at intervals of a few minutes to control the effects of treatment. If violent convulsions are occurring, they should be stopped by inhalation of chloroform, as they tend to raise the patient's temperature still higher. If congestion is at all marked, it is best to venesect early and withdraw 15 to 20 ounces of blood. In view of the fact that it is seldom possible to exclude presence of cerebral malaria, it is well as a routine measure to give quinine bihydrochloride 15 grains intravenously. If the blood appears very viscid, this may be given diluted in a pint of normal saline. If, in spite of these measures, the rectal temperature does not fall, an enema of a pint of iced water may be given. This, however, has the disadvantage of preventing rectal temperatures being reliable for some considerable time. When the rectal temperature has fallen to 102° F. ice should be withdrawn and the patient placed in a dry bed and carefully watched. If the temperature again shows a tendency to rise, treatment with ice must be used to control it, or in cases of collapse with low temperature stimulants such as strychnine may be given and hot-water bottles applied to the skin. If there is any evidence of malarial infection, quinine treatment should be continued, and as soon as convalescence is sufficiently established the patient should be removed to a cool climate.

## SCARLET FEVER

(*Scarlatina*)

Scarlet fever is a contagious disease, characterised by fever, sore throat, a bright red eruption on the skin, and a tendency to certain complications, of which the most important is acute inflammation of the kidneys.

**Ætiology.**—Scarlatina is usually derived from a preceding case of the disease, either by direct contact, by close proximity, or by means of clothing, books, papers, and other articles to which the virus clings with great tenacity. It has been conveyed by such means over miles of country, or has lain dormant for weeks or months, and then, meeting with a suitable nidus, has again developed the disease in its complete form. The infection is also conveyed by milk used as food, and though the milk has sometimes been infected by contact with scarlatina in the person of the milkman or his associates, it is very probable that it may be infected from a diseased condition of the teats or udder of the cow supplying it. It is generally believed now that the secretions from the throat and nose are the chief sources of infection, and that the desquamating skin which has long been considered responsible is much less often concerned.

Sex, occupation, and social position have no influence in its production, but the vast majority of those attacked by scarlatina are young children; it is comparatively infrequent in adults, and very young infants are less susceptible than older children. Still old people occasionally have it; and protection is not always perfect, so that some people have a second attack. Patients with open wounds and women in the puerperal state appear to be particularly susceptible to scarlatina, probably because the wound or the uterus provides an easy entrance for the virus. The disease in the surgical cases is often mild, and the rash is partial and of short duration, so that the connection with scarlatina was for a long time misunderstood; even now it is doubtful whether some of the cases may not be due to a streptococcal infection apart from true scarlet fever.

**Morbid Anatomy.**—The organs after death from scarlatina present little that is peculiar. In malignant cases there are the changes (*see* p. 22) common to the pyrexial and septic disorders: undue fluidity of the blood, soft liver and spleen, petechial spots on the serous membranes, and hypostatic congestion or œdema of the bases of the lungs. The tonsils present the conditions of ulceration or suppuration that have been observed during life.

The virus of scarlet fever is probably a specific hæmolytic streptococcus. The

blue masses called *Döhle's bodies*, or *inclusion bodies*, seen in the polymorphonuclear leucocytes, are found in unusual numbers.

Many of the complications involving the throat, ears, and other parts are secondary affections caused by pyogenic organisms.

**Symptoms and Course.**—The period of *incubation* is two, three, or four days, rarely as much as six days. Generally the invasion is sudden: the patient has a rigor, or vomits, and complains of frontal headache, with languor, pains in the back and limbs, and loss of appetite. The temperature rises to 103° or 104°, the pulse becomes very rapid, and the respiration is quickened. Very soon there is some complaint of sore throat, and swallowing is painful.

On the second day—that is, generally between twelve and thirty-six hours from the first symptom—the *rash* appears. It is first seen on the upper part of the chest, in front and on the sides of the neck, but soon spreads to the abdomen and back, and then to the upper and lower limbs. It consists of minute red spots, bright in the centre, fading towards the edge, and set closely together, so that the paler edges almost coalesce. Sometimes the coalescence is complete, so that the skin has a uniform bright red colour; sometimes the eruption is more discrete, and areas of pale skin are visible between the spots. The face, forehead, and cheeks are, as a rule, deeply and uniformly flushed, without showing the puncti-

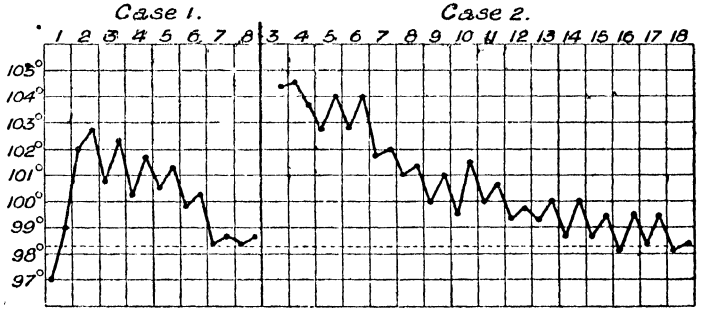



FIG. 2.—Temperature in Scarlatina.

form arrangement of the rash which is seen elsewhere; but the skin round the mouth remains pale, forming a sharp contrast to the cheeks (circum-oral pallor). With an abundant rash the skin becomes slightly swollen. The eruption presents many varieties as to depth of colour and distribution. It may be only pale pink, or it is deep, livid purple; and in some severe cases papules may be raised above the surface, and may even vesicate or form minute points of pus; and occasionally petechiæ occur. In its distribution the rash may be very limited, occurring only on the chest, or in patches on the thighs, elbows, or ankles, and this occurs frequently in second attacks, and in the mild cases sometimes seen in patients with open wounds, to which reference has just been made. The rash reaches its height on the third or fourth day, and begins to fade on the fourth, fifth, or sixth day; altogether it may last from five to ten days. After the subsidence of the rash *desquamation* takes place—that is, the superficial layers of the cutis are shed. This occurs in the form of white, branny flakes on the sides of the neck, preceded (as pointed out by Caiger) by an appearance of pin-point depressions, due to the rupture of the epidermis at the top of each papule. This may be as early as the sixth or seventh day, while the eruption is still visible on the legs; but the amount of epithelium that is shed and the size of the particles are very variable—sometimes there is nothing more than a little roughness about the tips of the fingers or toes, or in the folds of the palms of the hands; while in other cases the epidermis peels off in large flakes, and in rare instances complete glove-like moulds of the

hands and fingers are thrown off. Desquamation commonly takes from four to six weeks, but in these special cases a much longer time is required.

In the throat it is seen that the uvula, soft palate, and fauces are deep red, and often slightly œdematous; the tonsils are reddened, swollen, projecting towards the middle line, and presenting a number of yellow points, from the follicles being distended or covered irregularly with ashy or yellowish secretion. In later stages they may suppurate, or sloughs may form in them. The nasal mucous membrane also inflames, and secretes a quantity of mucus; and the submaxillary and cervical glands become enlarged and tender. The tongue is at first thickly covered with white fur, but in a few days this clears off from the tip to base, leaving a bright red, raw surface, on which the fungiform papillæ are unusually prominent, so as to give the appearance known as "strawberry tongue."

The temperature is frequently high, reaching 104° or 105° on the first day and remaining at this level for some days. Even a temperature of 106° may be reached. With this the skin is pungently hot and generally dry, but profuse sweating may occur. The pulse rises to 120, 140, or even 160. In severe cases the mental faculties are dulled; delirium is frequent, especially towards night; and drowsiness and coma supervene. The disease may reach its height about the fourth, fifth, or sixth day, and then, with the fading of the rash, the temperature begins to fall, generally subsiding rather gradually, but sometimes more suddenly, till the normal is reached, and convalescence is gradually established. In fatal cases death may occur about the fifth day or later from exhaustion, or from typhoid conditions, with low delirium, semi-coma, and dry brown tongue; or it may occur later still as a result of complications.

**Complications and Sequelæ.**—These are numerous and important.[1] Not only the tonsils, but also the soft palate and the uvula, may slough. More frequently the *glands* under the jaw and in the neck are much swollen, and the subcutaneous tissue about them is infiltrated, becoming brawny and indurated. The skin then assumes a dusky red colour, and sloughing takes place beneath it, separating it from the subjacent tissues over a large area. Such cases are often fatal. During convalescence also *adenitis*, both *simple* (*4·78*) and *suppurative* (*1·23*), is likely to occur. Extension of the inflammation from the throat up the Eustachian tube may cause *otitis* (*8·1*), or inflammation of the ear, resulting in abscess of the tympanum, rupture of the membrana tympani and otorrhœa. This occurs most commonly in the first five years of life. In the course of the scarlatina this may seem of little importance; but it lays the foundation for serious or fatal results months and even years afterwards, among which may be enumerated suppuration of the mastoid cells, meningitis, abscess of the brain, thrombosis of the lateral sinus or jugular vein, with pyæmia as a result, hæmorrhage from the lateral sinus, and facial paralysis. Deafness on the affected side may of course happen, and a double otitis in a young child may be the cause of permanent deaf-mutism. Other local lesions may occur as sequelæ, for instance, sloughing of the cornea, abscesses in the subcutaneous tissues, or cancrum oris.

The kidney is often involved in connection with scarlatina, and nephritis and albuminuria may occur in the following three ways: *Albuminuria* is often present in the stage of rash and fever, small in quantity, temporary in duration, and probably arising in the same way as it does in severe cases of other infections (*6·97*). Secondly, *nephritis* (*2·38*) may be first recognised as a sequela from two to three or four weeks after the beginning of the illness, when the patient is quite convalescent or suffering only from desquamation. It then begins with a chill and rise of temperature and the passage of turbid brown or blood-coloured

[1] The bracketed figures in italics represent the percentages obtained from 21,006 cases in the Metropolitan Asylums Board's hospitals, 1914.

albuminous urine, the whole subsiding again without the occurrence of dropsy. In other cases the first thing noticed is some swelling of the feet and face, and then the urine is found to be scanty, high-coloured, and albuminous, with blood pigment and granular, hyaline and epithelial casts. Recovery from slight cases is common; but the dropsy may become general, and death may result after six, twelve, or eighteen months, with the severe secondary complications which will be described elsewhere (*see* Nephritis).

*Broncho-pneumonia, bronchitis, pericarditis,* and *endocarditis* occur from 0·1 to 0·4 per cent. in each of the cases, and *pneumonia, laryngitis, meningitis, pyæmia, chorea, jaundice,* and *cervical cellulitis* still less often. Pneumonia and broncho-pneumonia are responsible for one-eighth of the deaths. Broncho-pneumonia may be caused by inhalation of septic materials from the throat. *Pleurisy (0·05)* may happen as a sequela; and if effusion take place, it often becomes purulent quite early. As in other severe fevers, dilatation of the *heart* sometimes occurs, and is recognised by displacement of the impulse. An acute general *arthritis,* which is indistinguishable from rheumatic fever, often follows upon scarlet fever so closely that the joints may be swollen when the rash is still present. Although generally known as *scarlatinal rheumatism,* it is possibly a synovitis due to the direct action of the septic organisms of the primary disease. It may affect many joints, but is not, as a rule, severe; and it may be accompanied by endocardial murmur, and be followed by permanent valvular disease. It is often valuable as clinching the diagnosis in a doubtful case of scarlatina. Exceptionally the joints suppurate. About one-fourth of the published cases of *purpura fulminans,* itself a rare condition, have occurred as a sequela of scarlet fever. Some relations of scarlatina to *diphtheria* are mentioned under the latter (*see* Diphtheria).

**Varieties.**—Besides the ordinary forms of scarlatina of moderate severity, which end in recovery, one recognises cases that are called *scarlatina maligna.* This form—also called, but not very logically, the *toxic* form—mostly includes cases that are fatal within five or six days from the intensity of the disease without complication other than sore throat. Sometimes the patient is struck down with convulsions and collapse, and dies in twelve or twenty-four hours before the rash has had time to develop. In other cases there are severe rigor and vomiting, early intense or livid rash, high fever and delirium; and the patients die in two or three days.

Cases with severe throat symptoms have been called *scarlatina anginosa.* To this nearly corresponds the *scarlatina ulcerosa* or *septic form* of Caiger, in which the faucial ulcers form a septic focus, from which the system may be poisoned.

The term *latent scarlatina* includes cases in which the rash and sore throat have been so slight as to escape detection, and the illness has only been discovered by the occurrence of desquamation or anasarca.

**Diagnosis.**—Scarlatina is recognised, especially when the disease is known to be prevalent at the time, by the occurrence of feverishness with sore throat, followed in a day by the characteristic punctate erythematous rash. The other characteristic features that may help in the diagnosis are—the sudden onset often with vomiting, circumoral pallor contrasting with the red cheeks, the state of the tongue, at first furred with a red edge, later patchy, and finally "strawberry," pinhole-and-flake desquamation (*see also* p. 36).

In the early stage, when the presence of sore throat is the most prominent feature, scarlet fever is to be distinguished from diphtheria, tonsillitis, the anginous form of influenza, and secondary syphilis. When the rash has developed scarlet fever must be distinguished from measles, rubella (*see* p. 36), syphilitic roseola, typhus, and the "septic" rashes which may occur in puerperal fever and other septic conditions. Even quite insignificant septic foci, such as impetigo, may give rise to a scarlatiniform rash. Various drugs may also give rise to a

rash resembling scarlet fever, and there are the rashes associated with intestinal disturbances from food or following the injection of horse serum.

Such cases do not generally have the sore throat or characteristic tongue, nor even the typical course; but the skin may peel afterwards.

The roseola of small-pox often has a distribution which is distinctive—namely, about the axilla, groins and thighs; and it is more purple in colour.

In the first four days the polymorphonuclear leucocytes almost invariably contain the so-called *inclusion bodies* of Döhle; they are not peculiar to this disease, but if they are absent at this time, scarlatina is unlikely.

The **Prognosis** must be in all cases very uncertain. Even in the mildest cases, renal complications may be serious or fatal. The mortality, however, is variable, some epidemics being exceedingly mild, while in others the mortality may be 30 or 40 per cent. In the Metropolitan Asylums Board's hospitals in 1914 the mortality was 1·4 per cent., and in 1915 2·03 per cent. In individual cases the prognosis may have to be determined by the condition of the patient from day to day; complications increase the danger. Very severe angina and an intense or livid rash coming out late are unfavourable, and cases with sloughing of the cervical glands are generally fatal. Dr. W. Hunter states that the initial angina, the adenitis, and the complications in the throat, nose, and ears are more severe in those previously suffering from bad teeth, inflamed gums, or pyorrhœa alveolaris (oral sepsis), than in others whose mouths are perfectly healthy. Scarlet fever attacking women recently confined shows a large percentage of deaths.

**Preventive Measures.**—The period of quarantine is eight days. Isolation must be carried out at the earliest possible moment (*see* p. 13).

The frequency with which convalescent patients returning from fever hospitals to their own homes have conveyed the disease to their brothers or sisters, not till then infected, has given rise to the name *return cases* for the new sufferers. It is allowed now that contagion is far more often transmitted by secretions from the throat, nose, or ear, than by the desquamating skin, and as much attention should be therefore directed to the former as to the latter. Hence, while no patient should be allowed to mix with the unprotected till at least four weeks have elapsed, this period must be prolonged if there is still any sore throat or any discharge from the nose or ears.

The final disinfection consists in the patient taking a warm bath and having his hair well washed. If in hospital, he should be kept separate from other acute cases for some time before going home.

Susceptibility of individuals to scarlet fever has recently been determined by means of the "Dick" test. 0·1 to 0·2 c.c. of a dilute solution of the toxic filtrate of the specific hæmolytic streptococcus is injected intradermally, the heated toxin being used as a control. A local area of redness beginning to appear in four to six hours, and reaching a maximum in twenty-four hours, indicates a positive reaction. It is rather early yet to say whether larger injections of the toxin will render susceptible individuals immune, like the Schick test in diphtheria.

**Treatment.**—A specific treatment of scarlet fever has only been possible in recent years. Antitoxic sera prepared from streptococci cultivated from scarlatina cases have been used rather frequently abroad, and are said to modify the course of the disease favourably. They are still on their trial.

Apart from this, the treatment must be carried out in the same way as that of other fevers: a well-ventilated room, the recumbent position in bed, light diet, and careful nursing. Isolation is essential in the interests of others. The body may be usefully sponged with tepid water daily; simple salines may be given internally, and the sore throat may be relieved by sucking lumps of ice. If the tonsils are much swollen, and much covered with secretion, the latter should be removed by pledgets of moist lint, and disinfectant or astringent

solutions applied by the same means. Formalin (1 in 200), liquor ferri perchloridi (℥ss to aq. ℥j), dilute hydrochloric acid (℥ss to ℥j), eusol, or izal may be thus used; and similar remedies in appropriate strength may be used to syringe the nose when that is involved. If a condition of oral sepsis is present, it should be treated with antiseptics from the first. For pain or swelling in the neck and about the angles of the jaws hot fomentations or boric lint wrung out of hot water should be used. Complications will require special treatment. Abscesses should be opened early. In otitis, relief of pain may be obtained by hot fomentations and the gentle introduction of warm water into the meatus. If suppuration of the middle ear is recognised, the membrana tympani may require puncture; and the meatus may be gently syringed with warm water, or diluted Condy's fluid, or solution of boric acid (1 in 20). For synovitis salicylate of sodium should be given in 10 or 15-grain doses, and chloroform or belladonna liniment may be used locally. The treatment of scarlatinal nephritis will be considered hereafter (*see* Nephritis). In the severe typhoid forms, with quick, feeble pulse, stimulants, such as brandy and ammonia, must be given; and in these toxic cases Gordon recommends a polyvalent antistreptococcic serum in doses of at least 50 c.c. Where there is very high fever with much delirium and restlessness, relief is often obtained by cold affusion to the head or body.

The occurrence of nephritis is possibly favoured by exposure to cold; this should, therefore, be especially guarded against as long as desquamation is going on. A free action of the bowels should also be maintained by occasional saline laxatives. Quinine and other tonics may be necessary where strength is recovered slowly.

## MEASLES

(*Morbilli*)

Measles is a contagious febrile disease, characterised by an eruption of pink or red spots and catarrh of the respiratory mucous membranes.

**Ætiology.**—In civilised communities its spread is determined by circumstances very similar to those influencing scarlatina. It occurs in epidemics, which attack the young rather than the old, chiefly because nearly all the older members of the community have had the disease when young, and are thereby protected from a second infection; but very young infants appear to be less susceptible, and to have the disease when caught in a milder form than children somewhat older. In large towns it is almost continuously present, spreading from point to point in the form of limited outbreaks, which subside, and are succeeded by others in different places; but where introduced among populations that have never been visited by the disease, or have been entirely free from it for years, it attacks the majority of the people, young and old alike, in one great and often destructive epidemic. This was the case in the Faroe Islands in 1846.

Infection is due to an unknown virus which is probably present in the secretions from the nose and throat. Experimentally the disease has been transmitted by inoculation from this source. Infection is thus spread by contact with the patient, or with the air infected by him; the virus also adheres to clothes, toys and other articles, though with much less tenacity than does the contagion of scarlatina. It is especially contagious in the catarrhal stage before the rash and during the continuance of the rash.

**Morbid Anatomy.**—This must depend much on the complication causing death, since uncomplicated measles is so rarely fatal. Redness of the mucous membrane of the larynx and trachea is observed; the spleen is moderately swollen. The lesions of bronchitis and pneumonia present no special features. There may be, however, fluid in the pleural cavity, and petechiæ under the pleural membrane; the bronchial glands are often enlarged and softened. Congestion of the mucous membrane of the ileum and colon is also sometimes found.

**Symptoms and Course.**—The period of *incubation* varies in measles as in

other contagious diseases, but has been most often found to be ten or eleven days. The disease commences with pyrexia and catarrhal symptoms: the temperature rises perhaps to 102°; the child loses appetite, is drowsy and unwell; there may be at first vomiting or chills, or, in children, convulsions. With this the conjunctivæ become suffused; the eyes water; there is a mucous discharge from the nose, and cough as a result of bronchial secretion. The catarrhal symptoms continue, but the temperature frequently falls after the first day, and continues at a lower level for another day or two, when it again rises. Occasionally in this early period there is a so-called prodromal rash, which may be like that of scarlatina, or urticarial, or like the true measles rash. It lasts only twenty-four or thirty-six hours.

The characteristic eruption appears most commonly on the fourth day, but it may be as early as the third. It is first seen on the face, at the roots of the hair, on the forehead, temple, or behind the ears, and it subsequently spreads to the neck, trunk, and limbs. It consists of pink spots, round, oval, or irregularly shaped, slightly raised above the surface, running together into irregular groups, which may have a somewhat crescentic shape, and leave some intervening area of skin unaffected. In colour, it is generally darker red, or more purple than that of scarlatina; but a distinction may be difficult, especially if the spots are uniformly distributed and do not coalesce. Occasionally a few petechiæ occur in the darkest part of the eruption, and in other cases a few vesicles may form in the centres of some papules. It comes out most fully on the face, giving it a blotchy, swollen appearance, and though less abundant on the extremities, it may form continuous patches of infiltration on the back and arms. It takes from one to three days to reach its height, and then rapidly declines, mostly beginning to fade first where it first appeared. It commonly leaves some mottling of brown or yellowish-brown colour, which lasts for some days; while petechiæ leave still more pronounced stains. It is also succeeded by slight desquamation in minute branny scales, but never in the large flakes seen in scarlatina.

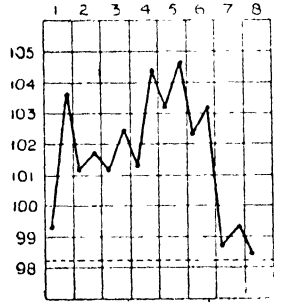



FIG. 3.—Temperature in Measles. (*After Strümpell.*)

If the temperature has fallen in the prodromal stage, it rises again, with the appearance of the rash, to 102°, 103°, or even higher, and reaching a maximum in two, three, or four days, falls generally rather suddenly as the rash begins to fade, and may reach the normal in about thirty-six hours. The catarrh continues throughout the eruptive stage; it may extend into the frontal sinuses and cause headache. There is more or less general bronchitis, indicated by cough, expectoration of mucus, and diffused rhonchi; and the larynx may be implicated, as shown by hoarseness, croupy cough, and in occasional cases by stridulous breathing. Even before the appearance of the rash the palate often shows abnormal redness, diffused or in patches, and almost invariably the lesions known as *Filatow's* or *Koplik's spots*. These are small, raised, white or opal dots, the size of a small pin's head, generally on a reddened base. They are seen best on the buccal mucous membrane opposite the premolar teeth of the lower jaw, and to a less extent opposite the other teeth. These appear two or three days before the eruption. The tongue is usually furred, and the fungiform papillæ are prominent. After the subsidence of the fever the return to normal appetite and sleep is generally rapid.

**Complications and Sequelæ.**[1]—The most important complications are

[1] The bracketed figures in *italics* are percentages from 747 cases in the Metropolitan Asylums Board's hospitals in 1914.

those connected with the respiratory organs, and it is to them that the majority of the deaths in connection with measles are due. Inflammation of the lungs is of frequent occurrence, and has generally been attributed to an extension of the bronchitis, which is common in all cases of the disease. But the pneumonia may be lobular or lobar in its distribution, and it may present in one case the features of a *broncho-pneumonia* (*16·19*), and in another case those of a croupous or pneumococcal pneumonia. The *laryngitis* (*2·66*) may be so severe as to threaten asphyxia, and may be accompanied with the formation of membrane; in some such cases it is a true diphtheria; in others it is caused by pyogenic organisms. Other complications are conjunctivitis (*1·33*), keratitis (*·26*) or iritis, stomatitis (*1·74*) and parotitis, inflammation of the Eustachian tube, *otitis* (*8·3*), diarrhœa or dysentery from enteritis of the small or large intestine respectively, albuminuria (*3·34*), and intestinal hæmorrhage. Gangrene of the mouth, called *cancrum oris*, or *noma*, occurs occasionally (*·93*), and gangrene of the vulva, also called noma, more rarely. Amongst sequelæ may be mentioned especially chronic inflammatory conditions of parts affected during the illness, such as chronic catarrh of the respiratory organs, chronic ophthalmia, enlarged tonsils or enlarged glands, otitis with discharge and its result (*see* p. 30), tuberculosis, endocarditis, and general malnutrition.

**Varieties.**—Measles without eruption and measles without catarrh have both been described; but it is doubtful if the former occurs, and some cases of the latter may really have been instances of rubella or German measles (*see* p. 36). In either case, the disease is mild. Of the severer or malignant forms *hæmorrhagic* measles is an example, in which bleeding takes place from mucous membranes, and the eruption becomes *purpuric*. Other severe forms are merely characterised by intense fever, dark or livid rash, often imperfectly developed, rapid and feeble pulse, prostration, delirium, dry brown tongue, and a generally typhoid condition.

**Diagnosis.**—A confusion with *scarlatina* and *rubella* is most likely to occur (*see* p. 36). In *typhus* the rash is not so papular, the face is but little affected, the spleen is swollen, and there is no nasal or conjunctival catarrh. The early stage of a *small-pox* eruption is sometimes simulated by that of measles; the absence of catarrh and the history of headache, back pain and sickness, are in favour of variola. Roseolous eruptions, apart from specific fevers, may resemble measles, but will be distinguished by the absence of the characteristic fever and catarrh. Koplik's spots are of value in diagnosis, as they rarely occur in other eruptive diseases.

**Prognosis.**—This is usually favourable. For the most part the mortality is from 1 to 2 per cent., though occasionally epidemics of much greater severity have occurred; and the prevalence of pulmonary and laryngeal complications increases considerably the percentage of deaths. Apart from these, the malignant cases are recognised by intense fever, dark or livid eruptions, and early collapse or prostration.

**Treatment.**—The treatment of measles is not essentially different from that of scarlatina. The child must be placed in a suitable room—warm, well ventilated, and free from draughts, and so arranged as to prevent infection of children hitherto free. Confinement to bed is scarcely necessary till the eruption appears. The diet, as in other febrile affections, should be light and easily digested. The catarrh which is present from the first should be treated by expectorants, such as squills, or ipecacuanha wine, or small doses of compound tincture of camphor. Antiseptic treatment of the fauces and nose may serve to prevent complications, both pulmonary and aural; thus glycerin of boric acid should be applied to the fauces three or four times daily; and the nose may be sprayed with resorcin in 10 per cent. solution, or syringed with dilute solution of potassium permanganate. If fever runs high, it may be reduced by the application of tepid water, either by sponging or by immersion in a bath at 95° or 90°,

which may be lowered still more by pouring in cold water. The temperature seldom remains at a high level, such as 105°, for many hours, but it may be desirable to spare the patient this prejudicial condition as much as possible; and even at a temperature of 102° or 103° a good deal of comfort and even sleep may be obtained by sponging with cold water (*see* p. 25). Stimulants are only required in the very severe forms. The child may generally be allowed to get up two or three days after the subsidence of the fever, but should be confined to the room for another week or ten days. During convalescence the general health should be attended to, and iron or cod-liver oil may be given, with, perhaps, change of air to the seaside or other bracing locality. Pneumonia or diarrhœa may be treated in the usual way, and discharge from the ears should be met by frequent washing with antiseptic lotions—*e.g.* potassium permanganate or boric acid.

## RUBELLA

(*Roseola, Rötheln, German Measles*)

This is an exanthem, resembling in many points both measles and scarlatina, but undoubtedly distinct from both. No specific micro-organism has yet been discovered in connection with it.

**Ætiology.**—It is not so infectious as measles, being spread in much the same way, especially by close association with some one suffering from the disease. Restricted outbreaks are a common result, older children and young adults being readily infected.

**Symptoms.**—The period of *incubation* is often sixteen or seventeen days, and may be a few less or more. A prodromal stage is either entirely absent, or at most lasts half a day before the appearance of the eruption; and this stage may be represented by a slight catarrh of the mucous membranes of the air passages or of the conjunctiva. But in some cases the *eruption* is the first indication of anything wrong with the patient. It consists of a number of pink spots, round or oval, very slightly raised above the surface, uniformly scattered, and generally discrete, though sometimes very closely set. The spots vary in size; when small and closely set there may be much resemblance to a scarlatinal rash; when larger there is more likeness to measles, but they are not commonly confluent, and do not take any crescentic form. Slight itching of the skin may be experienced. The eruption occupies the face, trunk, arms, and legs, appearing mostly on the face first and rapidly occurring on the other parts; it is generally of shorter duration than measles, often lasting only two days, sometimes three or four. As in measles, it may leave a little discoloration of the skin for some days afterwards; desquamation is commonly absent, and it is never in large flakes, as in scarlatina. The palate and fauces usually show some injection or spots and streaks of redness, and the tonsils may be a little swollen. The conjunctivæ are reddened, and coughing and sneezing are generally present to a slight extent. The *lymphatic glands* at the back of the neck in the occipital mastoid and posterior cervical regions are frequently swollen and tender, and sometimes those in other parts of the body. The swelling may persist two or three weeks, but suppuration has never been observed. Fever is, in the majority of cases, entirely absent; if it occurs, the temperature is only 1·5° or 2° above the normal, and it lasts one, two, or at most three days, showing the greatest variability in different cases, but often falling to normal before the eruption is completely developed. Many patients do not feel ill at all, and retain their appetite throughout. Any further complications than those indicated already are quite uncommon, and the prognosis is exceedingly favourable.

**Treatment.**—This must be conducted on the lines laid down for measles.

**Differential Diagnosis of Scarlatina, Measles and Rubella.**—*Initial*

*stage.*—In scarlatina the onset is sudden, and the first stage lasts twenty-four hours. There are nausea and vomiting, but not much catarrh. In measles there are definite catarrh and conjunctival suffusion for over three days before the rash. In rubella there is slight catarrh, which may last only a few hours.

*Adenitis.*—In scarlatina the glands below the angles of the jaw are definitely affected and are sometimes palpable in other regions. In measles there may be slight swelling below the angles of the jaw; very occasionally the posterior glands are affected, as in rubella. In rubella there is characteristic enlargement of the glands at the back of the neck.

*Nature of Rash.*—In scarlatina there are minute papules and a general erythema, often brick-red in colour. It may be patchy on the extremities. There may be some apparent swelling of the skin. In measles the spots are large and red and may form definite patches with curved outlines. In rubella the spots are smaller, rounder and pinker than in measles, and more discrete. Coalescence into patches may sometimes occur.

*Distribution of Rash.*—In scarlatina the palms, soles, and face are reddened but not affected by the rash. There is marked circum-oral pallor. The scalp also escapes. In measles and rubella all parts may be affected, and the circum-oral region is very constantly invaded.

*Fever.*—In scarlatina the pyrexia is marked and the pulse rapid; sore throat is prominent, and there is intense injection of the palate, and, except in the septic form, there is usually no bronchial catarrh. Defervescence is rather gradual. In measles the pyrexia is definite, and there is much constitutional disturbance, without a sore throat. Koplik spots are present in over 90 per cent. of cases on the buccal mucous membrane. Bronchial catarrh is a usual feature, often leading on to broncho-pneumonia. In rubella the pyrexia is slight and sometimes absent, and the patient feels well. Sore throat, if present, is very slight, and there is usually no bronchial catarrh.

*Tongue.*—In scarlatina the tongue passes quickly through three stages:—furred with red edges, patchy, strawberry. In measles it may be well coated, and is occasionally patchy, but not typically strawberry. In rubella there may be slight furring.

*Skin after Rash fades.*—In scarlatina the skin looks rather opaque, like parchment, and more or less sallow from pigment. Desquamation is of the pinhole-and-flake type; the pinholes are usually best seen at the outset on the neck. Desquamation is often very copious. In measles there is dirty brown mottling. Desquamation is fine, branny and not very copious. In rubella there may be faint yellowish-brown staining for a few days, but this is unusual. The desquamation is as a rule almost inappreciable.

## SMALL-POX

### (*Variola*)

Small-pox is a specific contagious disease, with a characteristic pustular eruption, due to a virus which is probably ultra-microscopic.

**Ætiology.**—This disease arises solely by contagion, chiefly, no doubt, by inhalation of the atmosphere surrounding infected persons. When cases are aggregated in hospital infection by aerial convection is believed to occur for at least a quarter of a mile. It is also conveyed by clothes, bedding, and other things, which have been in contact with patients; and it can be inoculated by means of the contents of the pustules. But patients are infectious before the eruption, and the virus is given off even from the bodies of those who have died. The susceptibility to the disease is common to all ages and both sexes; even the fœtus *in utero* may catch it from the mother; but the susceptibility then and in the first year of life is stated to be less than afterwards. Negroes are said

to be more liable than white people. The disease commonly occurs only once in the same individual; but second and third attacks occasionally occur, and the second attack may even be more severe than the first, though it is generally milder.

The liability to the disease, and consequently the number and severity of its epidemics, have been reduced considerably since the introduction of vaccination at the end of the eighteenth century. For instance, in this country there were annually several thousand deaths from small-pox in the middle of the last century (**2**). In 1871, owing to infection introduced with refugees from Europe at the time of the Franco-Prussian War, the number of deaths amounted to 23,000; but since that time there has been a steady diminution, and since 1905 the number of deaths has always been under thirty. This favourable result cannot be due chiefly to vaccination, because the proportion of infants vaccinated to the number of births has actually decreased from 65 to 43 per cent. since the beginning of this century. It is due rather to increased facilities of diagnosis since the disease became notifiable, and also probably to the mild form of the disease in England at present.

Epidemics of small-pox vary greatly in severity. The milder forms, sometimes called "alastrim," occur in Africa and America, the severer forms in Asia and Europe. There can be no doubt that the mild and severe forms are really one and the same disease, because, although it usually remains true to type, instances have occurred where the mild type has developed into the severe type.

The danger of small-pox is always with us, and when it is remembered that there are about a million cases of small-pox in the world, and that with our wide commercial relations the introduction of the disease into this country is more than a possibility, it would be reasonable to take all the preventive measures possible, viz. general vaccination (*see*, however, p. 43) and early diagnosis, notification, and isolation of infected individuals and contacts, and re-vaccination of the latter.

**Morbid Anatomy.**—In hæmorrhagic cases blood may be found effused into the solid viscera. Examination of the pustules shows that the process begins with hyperæmia of the papillary layer of the cutis; then the superficial layer of the cuticle is raised from the deeper layers to form a vesicle. The umbilication is sometimes determined by a hair, or the duct of a sweat gland preventing distension at this spot, or merely by cells of the rete Malpighi stretched into a fibre; bands and fibres formed in the same way constitute the septa dividing the vesicle into *loculi*. The pustule becomes hemispherical, in the later stages of suppuration, by the central band or *retinaculum* giving way. Whether the resulting scars are superficial or deep depends upon the extent to which the suppurative process involves the papillary layer of the skin. Micrococci due to secondary infections have been found in the pus and in various organs. The liver cells show fatty degeneration early in the disease.

**Symptoms and Course.**—The period of *incubation* may vary from five to twenty days, but is usually about twelve days. The disease mostly begins suddenly with a distinct rigor or chills, with severe backache, severe headache and vomiting. The temperature rises rapidly to 102°, 103°, or 104°, and the next day it may be still higher. The patient is mostly very ill, is unable to continue his work and probably takes to his bed. Anorexia, thirst, furred tongue, vomiting, and constipation are also present. On the third day the typical eruption appears; but in the initial stage in a certain number of cases rashes occur, with which it is important for diagnostic purposes to be acquainted.

**Early Eruptions.**—These are (1) *erythematous* and (2) *hæmorrhagic*.

(1) The erythematous rash tends to appear before the hæmorrhagic. In mild or modified small-pox it takes the form of irregularly shaped patches, which tend to be evanescent and migratory, and are not appreciably raised above the surface. It is often supposed to resemble measles, but rarely spreads any higher on to the

face than the level of the lower jaw. In severe small-pox the erythema forms large areas, or plaques, of a brilliant colour, with well-defined margins on the external surfaces of the limbs and on the trunk. More rarely the whole body surface is covered with a continuous sheet of erythema, but this is not punctate like scarlet fever.

(2) The hæmorrhagic rashes indicate a more severe degree of infection. They have a special affinity for flexures and may begin as an erythema. One of the most characteristic is the "triangular rash," which occupies the lower half of the abdomen, from the umbilicus downwards, covers the groins, and extends on to the thighs. It has the form of an inverted isosceles triangle, with the base about the level of the umbilicus. It also frequently appears in the axillæ, and on the adjacent parts of the arms and trunk, and extends thence along the flanks to the lower patches. It consists of small hæmorrhagic spots, or petechiæ, which on fading leave brown or yellowish-brown stains for a time. These initial rashes commonly appear on the second day, and last for about two days, co-existing with the early stage of the papular eruption, but disappearing before its full development.

Another form of hæmorrhagic rash is the *purpura variolosa*, which constitutes a very severe variety of the disease. On the second day, or even within twenty-

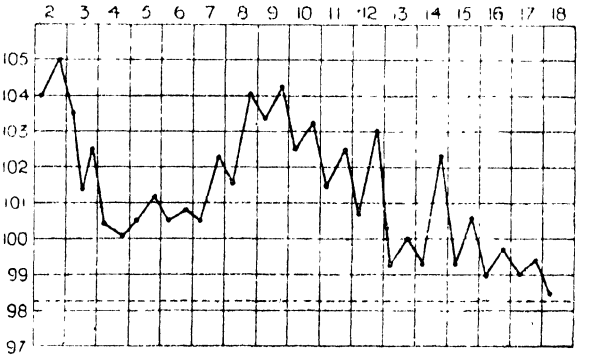



FIG. 4.—Temperature in Small-pox. (*After Strümpell.*)

four hours of the first symptom, a scarlatiniform rash appears, quickly followed by subcutaneous hæmorrhage, partly petechial, partly in larger patches. The face is red and puffy, the eyes suffused; there may be vomiting of bilious matters or of blood, with the passage of bloody stools, and the urine contains albumin or blood. The mind is generally clear till near the end; exceptionally delirium or coma is observed. The cases are nearly always fatal, often within three days of the commencement, and even earlier.

**Specific or Focal Eruption.**—This commonly begins on the third day of the illness by the formation of red small papules on the face, forehead, and scalp, and the same appear subsequently on the chest, back, arms, and hands, finally on the lower part of the body, the legs and feet. These papules soon become prominent; they are firm, and give to the finger the impression of extending deeply, a condition sometimes described as "shotty." Towards the end of the second day of their appearance a small vesicle appears in the centre, which is at first clear and transparent. As it gets larger, during the next two days, a very characteristic change takes place: the centre becomes depressed, and the circumferential part forms a prominent ring round it. This process is known as the *umbilication* of the vesicle. If the vesicle be punctured, only a small quantity of the contained serum will escape, the retention of the remainder being due to septa which divide the vesicle into separate cavities or *loculi*. Almost coincidently with

the umbilication of the vesicle, the contents become more opaque; and finally, about the sixth day (eighth of the disease), they are completely purulent. During this change in the vesicle the surrounding skin becomes pink, forming an inflammatory halo around it, and if there are many pustules—for instance, on the face—this leads to a great deal of swelling, which is often so considerable as to render recognition of the features quite impossible. The scalp becomes tense and tender, and the fingers also are often much swollen from the same cause. The stage of suppuration lasts two or three days, and then the pustules gradually dry up, beginning at the centre, and ultimately forming a brown or blackish-brown scab, which adheres for several days. Sometimes the desiccation is preceded by the escape of some pus from the vesicle, and with the drying up the swelling of the face and other parts subsides. Finally, the scab falls off, leaving a dark red spot, which is at first slightly raised above the general surface, but in the course of some weeks forms a depressed white scar. The pustules form most abundantly on the face, and on the backs of the hands, and are less numerous on the trunk and covered parts of the limbs. Parts that have been the seat of initial erythematous or petechial rashes are by many said to be less liable to the specific eruption. All the stages of the eruption occur first on the face, and follow, a day or so later, on the trunk and extremities. On the other hand, parts which have been irritated, as, *e.g.*, by the application of plasters or blisters, are liable to an abundant formation of pustules. The pustules are not confined to the skin, but occur on the mucous membranes also; they are especially well seen in the mouth, on the hard and soft palate, but present different appearances from those on the skin on account of the constant moisture to which they are subject. They scarcely develop into well-formed pustules, but are only grey or pearly elevations, which are liable to become abraded and form superficial erosions or ulcers. The tongue is generally coated and more or less covered with pustules; rarely its substance is inflamed. Ulceration of the larynx or even perichondritis may occur; and the process may extend to the nasal mucous membrane, so that the breathing is obstructed by the swelling and the formation of scabs.

**General Condition.**—In mild cases the *primary fever* subsides with the appearance of the specific eruption, and the temperature may become quite normal, so that patients who have remained at home during the first three days will go to their doctor or the hospital with an abundant crop of papules all over the body, but feeling comparatively well, free not only of the fever, but of the headache, lumbar pains, vomiting, and general malaise.

But when the pocks become purulent there is a fresh accession of fever, a *secondary* or *septic fever*, which may be ushered in by chills, or a rigor, and which lasts from three to six or eight days. The temperature rises to 103° or 104°, is mostly remittent in its course, and is accompanied with sleeplessness, headache, and delirium, and a pulse of 100 to 120. All this again subsides as the scabs dry and the swelling of the skin decreases. With the fall of the scabs some patients lose their hair, and even their nails.

**Varieties.**—Many subdivisions of small-pox have been made. The following are the varieties usually recognised at the present time: (*a*) ordinary or discrete small-pox, to which the previous description mainly applies; (*b*) a mild form of discrete small-pox characteristic of certain epidemics; (*c*) confluent small-pox; (*d*) malignant small-pox, the purpura variolosa before mentioned; (*e*) hæmorrhagic small-pox (*variola hæmorrhagica pustulosa*); (*f*) modified small-pox; and (*g*) inoculated small-pox.

(*b*) The following are the characteristics of the very mild form of the disease prevalent in the Midlands during the last three years: In 2,000 cases in Derbyshire there were no deaths. The prodromal symptoms were very mild and sometimes absent. Their onset was insidious, with headache, dizziness, and aching of the limbs. The pyrexia varied from 100° F. to 103° F. There were no prodromal rashes or hæmorrhages, and there was no prostration. The focal eruption did not

appear constantly at the end of the third day, but sometimes immediately after the primary toxæmia, and in a third of the cases it was delayed so as to leave an interval of from one to four days after the end of the primary toxæmia. The pocks were smaller, more superficially situated, and their evolution was more rapid than in virulent small-pox. They failed to pustulate completely, but dried up into amber-like beads. The vesicles were unilocular. There was usually no secondary fever.

(*c*) *Confluent small-pox* is a form in which the eruption is very abundant, and the general illness is correspondingly severe. The initial fever is high, and the temperature does not fall to normal with the appearance of the rash, as it does in mild cases. The rash appears early, even by the second day, and is very abundant, so that on the face, which is most affected, the pustules are closely set, the skin is enormously swollen, and in the stage of suppuration several pustules coalesce and form irregular and more or less extensive purulent blebs. The implication of the mucous membranes of the nose, throat and larynx is much more constant and severe. The secondary fever is also high, and is accompanied by prostration, rapid pulse, and delirium or coma. Complications are more frequent and serious, and the mortality is great, death taking place from exhaustion, or hyperpyrexia, or pyæmia.

(*d*) *Malignant small-pox*, or purpura variolosa, in which hæmorrhages appear in the skin within the first forty-eight hours, has been mentioned (*see* p. 39); in another form (*e*) the hæmorrhagic tendency shows itself later. The specific eruption appears, and then hæmorrhage takes place into the papules, or later still into the pustules, or into the skin between the pustules. The petechiæ often occur first on the lower extremities. The mucous membranes are also affected with hæmorrhages, or membranous patches, and bleeding occurs from the nose, lungs, rectum, kidneys, or uterus. The cases are mostly fatal.

(*f*) *Modified small-pox*, often called *varioloid*, occurs in those who have been vaccinated but in whom the protection is incomplete, either from deficient vaccination originally, or from the decline of its influence with lapse of time. No broad line of distinction can be drawn between this and the mild form of discrete variola already described. Sometimes the initial fever is severe, with a high temperature, but it is generally of short duration; the secondary fever is very slight, and the eruption is only imperfectly developed. Recovery is the rule.

(*g*) When small-pox is *inoculated* a pimple rises on the second day at the seat of the operation; this develops into a vesicle or pustule. The patient then has rigors, swelling in the axillary glands, and some fever; and about the eleventh day the usual eruption of small-pox appears, and passes through its stages. The attack is generally mild, but the disease is contagious and occasionally fatal.

The **Complications and Sequelæ** are chiefly as follows: abscesses and erysipelas, conjunctivitis, and sometimes destruction of the eye from suppurative keratitis; chronic otitis and caries of the bones of the ear; in the respiratory system, bronchitis, broncho-pneumonia, and pleurisy, and the changes in the larynx above described. On the side of the nervous system the following may occur: hemiplegia, probably from arterial thrombosis or encephalitis, myelitis in different forms, occasionally disseminated sclerosis, and multiple neuritis.

**Diagnosis.**—This is best considered according to the stage of the disease: (1) In the pre-eruptive stage, the occurrence of shivering, with severe pain in the head and back and vomiting, characteristic of small-pox, might also suggest influenza, acute rheumatism, lumbago, or other acute diseases. (2) Early eruptive stage: The petechial eruptions on the lower parts of the abdomen and in the groins are very characteristic, but when the hæmorrhages are more generalised (*purpura variolosa*), purpura, septicæmia and other causes of petechiæ, *e.g.* scarlet fever and food-poisoning must be differentiated. The erythematous eruptions may closely simulate scarlatina or measles. The scarlatiniform eruption of variola is not punctate and is unaccompanied by inflammation of the throat.

The morbilliform eruption is not raised like that of measles and is rare on the face. Syphilis also sometimes presents an eruption like it. (3) The specific eruption in the papular stage, appearing first on the face and often grouped in twos and threes, may resemble papular erythema, *e.g.* a papular measles rash. (4) In the vesicular stage, small-pox must be differentiated from chicken-pox (considered later on, p. 45) and pemphigus. (5) In the pustular stage, it may resemble pustular eczema. (6) In the crusting stage it may resemble chronic eczema or exfoliative dermatitis. (7) Finally, it is necessary to consider the possibility of an individual being recently scarred with small-pox, and this may give the clue to the nature of an obscure acute affection in other members of the family. Careful account must also be taken of the recent history of the patient.

The **Prognosis** depends on vaccination. Nearly all persons attacked in the first few years after vaccination recover. In later years any protective action may be difficult to trace. The typical hæmorrhagic form is always fatal. About one-third of ordinary confluent cases die, and 5 to 10 per cent. of discrete cases. The profuseness of the suppuration is the surest measure of the fatality. The existence of very mild epidemics must be remembered.

**Treatment.**—The general lines of treatment are the same as for the infectious diseases already described. The patient must be isolated for the sake of others; he must be in bed in a well-ventilated room, and should have abundance of milk and easily digestible food, and cooling drinks to quench thirst.

When small-pox is fatal death occurs most commonly in the stage of secondary fever from suppuration of the pocks. The method introduced by Dreyer of Cairo in 1910 is the best treatment for severe small-pox. The skin of the whole body is painted over with a 3 or 5 per cent. permanganate solution once daily. A 1·5 per cent. solution may be used if the skin is sensitive. In confluent cases it is best to place the patient in a permanganate bath at body temperature for a quarter of an hour twice daily. This treatment, if begun early, lessens the suppurative process, prevents the formation of bedsores and general sepsis, increases the comfort of the patient, and lessens the risk of infection of the attendants. Opium may be given to procure sleep. In the severer cases stimulants may have to be given freely.

If an unvaccinated person has incurred the risk of small-pox he should be vaccinated at once, as it is certain that the disease may be favourably modified by this procedure.

## VACCINATION

**Prevention of Small-pox.**—*Inoculation and Vaccination.*—The observation that small-pox, when conveyed by inoculation of the contents of the vesicle under the skin, produced a milder attack than that commonly conveyed by contagion, led to the use of inoculation as a means of protecting the individual from the more dangerous forms of the disease. Lady Mary Wortley Montagu introduced the custom into England in the early part of the eighteenth century, and her example was widely followed. But a serious disadvantage attached to this proceeding: the small-pox induced by inoculation, though mild, was contagious, and the spread of the disease was thus decidedly favoured. Inoculation consequently fell into disrepute, and finally yielded to vaccination—*i.e.* the inoculation of *cow-pox*, or *vaccinia*—first practised by Jenner in 1796. He was led to make the experiment from the facts, long observed in dairy farms, that cows were liable to a pustular disease of the udders and teats, which was often accidentally communicated to men and women milking them, and that these persons were subsequently insusceptible to small-pox, either by contagion or by the inoculation then in vogue. Conversely it was observed that those who had had small-pox did not catch the disease from the cows. Jenner inoculated patients with cow-pox, which produced its characteristic effects, and he subsequently found that certain of these patients were insusceptible to a small-pox

virus, which set up typical variola in other unvaccinated persons. The power of vaccination to protect from small-pox has been abundantly proved since then by facts which I need not here repeat.

The cow-pox may be conveyed from man to man by means of the lymph contained in the vesicles a great many times without any very obvious diminution of its power to reproduce the disease and to protect from variola. This arm-to-arm vaccination was in common use in the early days, and occasionally caused syphilis and other diseases to be transmitted, so that it has now been superseded by the use of lymph obtained direct from the calf previously inoculated with the virus. The introduction of this lymph was much facilitated by Copeman's demonstration that by thoroughly incorporating six parts by weight of a 50 per cent. solution in water of chemically pure glycerin with one part of the calf lymph or vesicle pulp, and afterwards storing the mixture for some weeks prior to use in sealed capillary tubes protected from light, any streptococci or staphylococci existing in the lymph, and even tubercle bacilli if introduced, are completely destroyed. The use of *glycerinated calf lymph* was recognised in the Vaccination Act of 1898.

**The Method of Vaccination.**—The part selected for the operation is generally the outer side of the left arm, near the insertion of the deltoid muscle. The skin is first thoroughly washed and rendered aseptic, and put on the stretch by the use of the left hand. The vaccine may be introduced by puncture or by scratching. If by the former, the sterilised lancet charged with lymph from the capillary tube is inserted obliquely at three, four, or five places from $\frac{1}{8}$ to $\frac{1}{2}$ inch apart, so as to ensure the introduction of the lymph, if possible, without drawing blood. If by the latter, the skin is scratched in two or three directions at the selected spots by a sterilised lancet or needle, any blood is wiped away, and the lymph is rubbed in, or a drop of lymph is first placed on each spot, and the skin scratched through it. After the lymph has dried the spots may be painted over with flexible collodion, but later on when a vesicle occurs a pad of boric lint should be applied.

The English law requires that all children should be vaccinated before the age of six months, unless the parent provides a medical certificate that the child is unfit to undergo the operation or makes a statutory declaration of his conscientious belief that the proceeding will prejudice the child's health. It should be pointed out that there are at present two views as to how vaccination is to be used to prevent small-pox. One view insists on the importance of infant vaccination, with periodic re-vaccination as far as possible, and that individuals who have been in contact with a small-pox case shall also be vaccinated. The other looks on infant vaccination as unimportant, so long as vaccination of contacts is carried out. The former view is more widely held, and is the safer one to adopt.

**Vaccination in Man.**—When lymph from a vesicle of cow-pox is inoculated under the human skin, nothing occurs till the end of the second or the third day, when a papule appears at the seat of inoculation. This increases in size, and on the fourth or fifth day a vesicle forms, which enlarges and forms a circular bleb, flat or slightly depressed in the centre, and pale grey in colour. On the eighth or ninth day the contents begin to be purulent, and a pink zone of inflammation forms around it. The vesicle becomes more opaque; the redness increases in extent, and is accompanied by induration. The neighbouring lymphatic glands become swollen and tender, and a slight degree of fever and malaise is present at this time. About the tenth or eleventh day the pustule begins to dry, and a brown scab forms during the next few days. The surrounding inflammation subsides, and about the end of the third week the crust falls off, leaving a depressed, pitted, and permanent scar.

Vaccination is, in the vast majority of cases, a perfectly harmless procedure, but occasional accidents are observed. Erysipelas may attack the wound of vaccination, as it may any other wound by accidental infection; and gangrene

has very rarely occurred. There can be also no doubt now that syphilis may be transmitted by human vaccine lymph, even though it is to the naked eye perfectly clear and free from blood; but this is, of course, obviated by the use of calf lymph. If human lymph is used, it should be known that the child supplying it is free from any suspicion of disease, and comes of healthy parents. On the other hand, the operation should not be undertaken at all on children who are badly nourished or suffering from eczematous or other eruptions, except in times of epidemics, when the risk from small-pox may overshadow every consideration of possible aggravation of ill-health already existing.

According to the special report of the Ministry of Health, out of 3,266,897 primary vaccinations in the period 1911–19, there were sixty-eight certificates of death concerned with vaccinations. Not all these deaths were due to vaccination; in some cases vaccination was carried out shortly before death from other diseases, *e.g.* measles, broncho-pneumonia.

Cases of *aberrant vaccinia* are sometimes observed. Milkmen may become inoculated on the hands while milking, and in a case seen by the writer inoculation on the cornea led to blindness in one eye. Secondary vaccinia on the lips, tongue, tonsil, eyelids, etc., have been reported in individual cases, following ordinary vaccination on the arms.

**Revaccination.**—The extent to which the first vaccination is efficient is generally estimated from the number and depth of the scars, and amongst fatal cases in epidemic times an inverse proportion has been shown between the number of the scars and the percentage of fatal cases, the mortality being least in those with four or more scars, greater in those with only one scar, and most of all in those stated to have been vaccinated, but without any visible scar at all. But in any case the protective influence of vaccination has only a limited duration, probably from twelve to fifteen years. It thus becomes desirable that every one should be again vaccinated in childhood or early puberty, and subsequently at any age, if small-pox should become epidemic. According to the extent to which the influence has faded, revaccination will have different results. It may fail entirely, or only produce a little local irritation; or it may produce a typical vesicle.

## CHICKEN-POX

(*Varicella*)

Chicken-pox is a specific infectious disease characterised by an eruption of vesicles. The nature of the virus is unknown. It commonly occurs in children, but may attack both infants and adults. Contagion is conveyed by the air or by clothes, and possibly by the pus from vesicles or scabs, as it has been successfully inoculated. An attack confers immunity, as a second attack in the same person is quite uncommon. Although it has often been confounded with small-pox, it is certainly a different disease: vaccination does not protect from varicella, nor varicella from small-pox. The association of varicella and herpes zoster has often been observed, but the relation between them is as yet obscure. The period of *incubation* is about a fortnight, but may be as short as ten or as long as nineteen days. The eruption consists at first of pink spots or papules, on which, in twelve or twenty-four hours, vesicles form. These are generally tense, hemispherical, and from $\frac{1}{8}$ to $\frac{1}{4}$ inch in diameter. At first the fluid is clear and colourless, but it soon becomes opalescent or milky, and then the vesicle shrivels, and a yellow or brown scab forms, which adheres for a few days, and then separates, leaving a pink stain. The perfectly formed vesicle is surrounded by an inflammatory zone, which subsides as the vesicle dries. They are more superficial and less loculated than in small-pox. Some of the pocks, but never a large number, result in depressed cicatrices.

The period of invasion is represented by febrile reaction, which is generally

very slight; a diffuse or patchy erythema may come out before the proper rash, which, however, shows itself within twenty-four hours. It appears earliest, and is seen most commonly on the chest, but the face, trunk, and limbs are also affected. The spots are not very numerous, but fresh ones form for two or three days after the first appearance, and altogether they number, as a rule, from fifty to 200. A few vesicles form on the mucous membrane of the mouth, palate, and lips. Whatever fever preceded the vesicles continues for a few hours, or for two or three days; it is generally not above 102°, but may reach 104°. The lymphatic glands of the neck may be enlarged. Death very rarely occurs, but convalescence is not always rapid.

In *varicella gangrenosa* some of the vesicles increase in size, become purulent, form reddish-brown or black scabs under which the skin sloughs, and ultimately leave circular ulcers with clean-cut edges. The child becomes very ill, and death may take place. In *V. bullosa* large bullæ are found in addition to the usual vesicles.

Cases have been recorded in which *hæmorrhage* has occurred into and around the pustules, as well as at other points in the skin; while there have been bleeding from the nose, mouth, vagina, and rectum, and petechiæ under the serous membranes. Most of these cases have ended fatally.

**Treatment of Varicella.**—Children should be isolated, but confinement to bed is not often necessary. Light diet and attention to the bowels are often all that are required.

**Differential Diagnosis of Small-pox and Chicken-pox.**—Varicella is sometimes difficult to distinguish from modified small-pox, and in epidemics of the latter it has been found desirable to make varicella "notifiable" (*see* p. 14), so that no case of small-pox may escape the attention of the sanitary authorities. Since the differential diagnosis of these two conditions is one of the most important issues in the diagnosis of fevers, it is treated at some length. *See also* Diagnosis of Small-pox (p. 41). In the first place the patient must be examined in a thoroughly good light, and as much of the skin as possible should be seen at one time by making the patient take off his clothes and use a blanket as a covering.

According to Wanklyn (**2**) the four most important groups of clinical phenomena are as follows:

(1) Prostration. This is usually a reliable sign of small-pox when considered in proportion to the density of the rash. A copious rash preceded by complete absence of prostration is strongly against small-pox.

(2) Distribution of the rash is the most important of all. Killick Millard (**2**) writes as follows: "(1) Ignore the head and neck. (2) Divide the rest of the body into (*a*) trunk and (*b*) limbs. Then, if the lesions on a careful count be more numerous on the limbs, the case is almost certainly one of small-pox; whereas if they be more numerous on the trunk it is almost certainly chicken-pox. It is true that there are a few cases in which the lesions may be almost equally divided, and in order that any error may be on the safe side—that is, in the direction of suspecting small-pox even though the case be one of chicken-pox, rather than *vice versâ*, it is well to weight the test by counting the buttocks and shoulders as being part of the limbs; though in reality I believe that these regions should be counted with the trunk if the test is to give the greatest proportion of correct results."

(3) Localisation or depth in the skin. In varicella the papules are superficial, in small-pox deep. It is specially useful to regard those lesions which occur on the tenderer portions of the skin, such as over the axillæ, chest, or flanks; relative depth in the skin is more readily appreciated in such situations, and by rolling a loose fold between the finger and thumb.

(4) Maturation. This means that the rash of small-pox tends to progress at a definite rate. In *varicella* vesiculation of papules occurs within a few hours, and is complete within twenty-four or at most forty-eight hours, the contents being

turbid and not often really purulent. The pocks dry up in two or three days from the onset of the rash. In *small-pox* vesiculation of the papules is usually noticed on careful examination after forty-eight hours. It takes another four days before it is complete. Nearly all the pocks pustulate in severe small-pox, but the change is not complete until the sixth day of the rash. In modified small-pox fewer pocks become pustular. The whole rash may abort in the vesicular or semi-pustular stage. If a rash is papular and remains so for three or four days it cannot be small-pox. A hæmorrhagic case is often diagnosed by the supervention of the papular rash; that is only to say again that the rash of small-pox develops and matures.

Other features which have been used in the past to differentiate small-pox from varicella are generally considered now to be of very little value for this purpose. They are the "shottiness," "umbilication," "multiloculation" of the pocks in small-pox, their presence on the palms, soles or palate, their uniformity of development in a particular area, and in varicella the appearance of the pocks in crops in a particular area.

## MUMPS

*(Specific Parotitis)*

Mumps is a specific contagious disease, of which the essential lesion is an inflammation of the parotid gland.

It occurs mostly in children and young adults; young infants, as well as elderly people, are more rarely affected. Males are more susceptible than females. No micro-organism has been identified; but it appears that the virus is filterable, like that of poliomyelitis; and serum from the diseased parotid, injected into monkey's parotids, has produced symptoms comparable with those of mumps.

**Symptoms.**—The period of *incubation* varies from fourteen to twenty-six days, and is more often nineteen, twenty, or twenty-one days. The commencement may be shown by slight malaise for a day or two, but the first symptom is often a feeling of pain and stiffness in the jaw and cheek of one side. Swelling then takes place just beneath the lobule of the ear, so that this is pushed out, and the depression between the jaw and the mastoid process is filled up. The swelling then spreads lower, beneath the ramus of the jaw, and may involve the sublingual and submaxillary glands. After a day or two the glands of the other side become involved, and thus there is a collar of swelling round the whole jaw from side to side. The swelling is pale, shiny, doughy in consistence, and tender when touched; but suppuration rarely takes place. Internally the tonsils and fauces are somewhat swollen. As a result the teeth can be separated with great difficulty, and not for more than half an inch or so; and mastication and deglutition are very painful, the pain on movement of the jaw being darting, and lasting for some time. The secretion of saliva may be normal, or increased, or diminished. There is a moderate degree of fever, the temperature rising often to 102°. There is a relative and absolute increase of the lymphocytes in the blood, and this condition lasts for fourteen days. The swelling lasts from seven to ten days, and slowly subsides; and the patient is usually quite well within three weeks. Occasionally the skin over the gland desquamates. Cases of mumps have been described, in which the submaxillary glands have been alone affected.

**Complications.**—As a result of mumps it happens occasionally that *orchitis*, or inflammation of the testicles, occurs just as the parotitis is subsiding, *i.e.* about the seventh or eighth day; but it may be earlier or later than this, and may, indeed, precede the parotitis. It occurs in from 20 to 30 per cent. of male cases, and is more common in adults than boys. The process begins in the epididymis, the testicle swells, and there may be effusion into the tunica vaginalis and œdema of the scrotum; it is accompanied by pain and tenderness, a rise in temperature, which may reach 104°, and in rare cases by acute delirium. The

inflammation subsides in a few days, but it may be followed by a permanent atrophy. More rarely there is double orchitis. In females the mammæ may inflame (*mastitis*) or the external genitals swell, and rarely the ovaries are tender. Mastitis has also been seen in boys. *Pancreatitis* has also been recorded, occurring generally at the end of the first week, and lasting from two to seven days; but it has been known to precede the parotitis. It is shown by pain in the epigastrium and left hypochondrium, tenderness and swelling in the same region, pyrexia, nausea, vomiting, and occasionally by diarrhœa or the passage of fat in the stools. Transient diabetes has also been described. Meningitis, optic neuritis, otitis, peripheral neuritis, bulbar paralysis, endocarditis and nephritis are rare sequelæ.

The **Anatomical Change** in mumps is an inflammatory infiltration, serous and cellular of the interalveolar fibrous tissue of the salivary glands.

The **Diagnosis** presents no difficulties, and the **Prognosis** is favourable.

**Treatment.**—The patient should remain in one room; and confinement to bed is believed to lessen the liability to complications, especially in males, in relation to orchitis. Food must be given in a liquid form. Locally fomentations and opium or belladonna applications generally give relief; and an antiseptic mouth-wash, containing boric acid, should be used frequently. Internally small doses of potassium citrate or ammonium acetate may be grateful to the patient.

## WHOOPING-COUGH

(*Pertussis*)

Whooping-cough is a disease characterised by a peculiar convulsive cough, followed by a long-drawn inspiration through the nearly closed glottis, by which a crowing noise, or "whoop," is produced.

It is contagious, generally requiring rather intimate contact, but sometimes apparently conveyed by clothing, and sometimes certainly by the sputum. Children are very susceptible, and most people have the disease in early life, while it quite rarely attacks adults. A second attack in the same patient is even more rare than in the case of the exanthems. It is most common between the ages of one and eight years, and girls are more liable to it than boys. It occurs in epidemics, but there is not much evidence that such epidemics are determined by climate or weather. It has often been observed that an epidemic of whooping-cough has immediately followed an epidemic of measles.

**Pathology.**—In 1906 Bordet and Gengou described a bacillus about the size of the *Bacillus influenzæ*, but longer and plumper, and somewhat resembling it in cultural characters. They found it best in the early stages of the disease, in the viscid mucus expectorated apparently from the smaller tubes, and there in almost pure culture; but it is often associated with other influenza-like bacilli. It is now called *B. pertussis*. The blood serum of convalescent cases agglutinates this bacillus, and gives the deviation of complement reaction with it. Vaccines prepared from it have appeared to influence favourably the course of the disease (Freeman). While the cough must be due to the secretion set up by the organisms, the whoop is not so readily explained. It is generally thought to be caused by a spasmodic closure of the glottis, but a passive approximation of the cords, or a failure to open freely when the sudden inspiration takes place, would probably account for it.

The **Morbid Anatomy** of pertussis is that of its complications, chiefly broncho-pneumonia. The laryngeal and tracheal mucous membranes are injected, and the bronchial glands are swollen.

**Symptoms.**—The period of *incubation* is about ten days. The limits are four to fourteen days. The first stage is one of bronchial catarrh, which is not always distinguishable from an ordinary catarrh. There are cough, expectora-

tion in children old enough, a few rhonchi in the chest, and slight pyrexia; but sometimes with the cough there is an unusual repetition of the expiratory effort, which may lead to suspicion. This preliminary bronchitis lasts from seven to ten days, and then there is a more or less rapid transition into the whooping stage. First, perhaps, a long-drawn inspiration follows the cough, and then an unmistakable "whoop." But the cough itself is as characteristic as the whoop. The child may be playing with its toys, apparently well, when it suddenly stops, seems distressed for a moment, and then perhaps runs to its mother or nurse. A short cough occurs; this is quickly followed by another and another without any intervening inspiration, each successive cough getting less loud and more stifled until they have mounted up to fifteen or twenty expulsive efforts in the course of seven or ten seconds. Then follows a long-drawn inspiration with loud laryngeal sound, the "whoop"; another burst of short coughs succeeds, with another "whoop"; and this sequence may occur once or twice more, with less violence and less noise, until finally a little tough mucus is expectorated, or vomiting takes place. During the coughing efforts the face becomes congested or cyanosed, the features swollen, the eyes starting from the head, the tongue hanging from the mouth, blood-stained saliva is coughed in all directions, and little relief takes place even from the inspiration, until the final expectoration of mucus or the cessation of the paroxysm. During this time the child is quite given up to the absolutely uncontrollable reflex process; a child in bed, when it feels the attack coming on, will seize the porringer and place it under its mouth, and in another few seconds it will be entirely at the mercy of the cough, and regardless of what is going on around. As a result of the obstruction to respiration during the coughing efforts, hæmorrhages frequently take place, bleeding from the nose, mouth, or gums, subconjunctival ecchymosis, petechiæ under the skin, and in very rare cases cerebral hæmorrhage. After a time the face often acquires a puffy and bloated appearance from the frequent obstructions to the return of blood to the chest. Sometimes a small ulcer forms on the frænum linguæ, from the pressure of the lower incisor teeth during the cough. The attacks often appear to be spontaneous, but they constantly occur if the child cries or gets in a passion, or even if the child is disturbed, as when the nurse begins taking off the clothes for an examination of the chest. The attacks occur both day and night, and it has generally been noted that they are more frequent in the night hours, between 6 P.M. and 6 A.M., than during the other twelve hours of the day. Observations in the whooping-cough ward of the Evelina Hospital for sick children did not confirm this. The number of paroxysms, which may, as above shown, include three or four actual "whoops," ranges from one to sixty in the twenty-four hours, but it is very rare to have more than forty attacks, and many cases never reach thirty in the twenty-four hours. In the intervals the child may be perfectly well, and is free from fever, unless there is some complication; the appetite also may be good, and the child soon replaces what he loses by vomiting, which does not, as a rule, occur in more than a small proportion of the paroxysms. This second stage of whooping-cough lasts a variable time, often from three to six weeks, but sometimes up to three months or more. The attacks gradually get less frequent, until they cease altogether, or as they diminish they may be accompanied by attacks of simple cough, not followed by a whoop, and this may last a few weeks longer. Death rarely occurs directly from the paroxysms; it may occasionally do so from prolonged closure of the glottis, or from cerebral hæmorrhage.

Other **Complications and Sequelæ,** however, occur which make whooping-cough a serious and even dangerous complaint. Amongst the former may be classed *bronchitis*, which may continue throughout, and *broncho-pneumonia* (*11·47* per cent. of 889 cases at the M.A.B. Hospitals in 1914), which is revealed by high fever of remittent type, by crepitant râles or patches of dulness and

tubular breathing, and by continued dyspnœa in the intervals between the cough. Often, but not always, the whoop is absent during broncho-pneumonia, as it is if any other febrile complication ensues. *Otitis* (*6·52*) is less frequent than in scarlatina and measles. General convulsions (*2·69*) sometimes occur, either as a direct result of the paroxysm or less commonly as the indication of cerebral hæmorrhage or thrombosis, or, it may be, of the onset of pneumonia. As sequelæ continued bronchitis, emphysema, and bronchiectasis with clubbing of the fingers occasionally occur. There does not seem to be any relation between whooping cough and tuberculosis.

**Diagnosis.**—This mainly depends upon the whoop, on the convulsive character of the cough, and on the regularity of the course from the catarrhal to the convulsive stage. Enlarged bronchial glands may cause a cough something like that of pertussis, but there will be no history of infection and no whoops; while other symptoms of independent lung diseases may be present. In whooping-cough the leucocytes are increased to 15,000 or 30,000 per cubic millimetre; and a differential count gives 60 per cent. of lymphocytes to 40 per cent. of polymorphonuclears, with a few eosinophils. These changes occur quite early, and are of use in diagnosis (H. T. Ashby). A certain diagnosis can be made from a culture of the *B. pertussis*. It has been obtained from 75 per cent. of cases in the catarrhal stage, from 54 per cent. in the paroxysmal stage, and from only 9 or 10 per cent. after five weeks.

The **Prognosis** is to be made from the severity of the complications.

**Treatment.**—The child should be kept in a warm but well-ventilated room, but confinement to bed is not necessary in an uncomplicated case. A variety of drugs has been used to check the paroxysms of pertussis; the length of the illness may be diminished and the severity reduced by their means. Belladonna is much used in the form of tincture, of which 2 or 3 minims may be given to a child two years old three times a day, and larger doses to older children. The dose may be cautiously increased up to 10 to 15 minims in a child of five or six. Dilute hydrocyanic acid (1 to 2 minims), chloral (2 to 5 grains), potassium bromide (2 to 5 grains), hydrobromic acid (3 to 10 minims), antipyrin (2 to 5 grains), and bromoform (2 to 5 drops mixed with almond oil and mucilage of tragacanth or acacia), have been given. Recently benzyl benzoate has been tried with great success. It is a powerful antispasmodic. A 20 per cent. alcoholic solution is used, and the dose is 5 to 40 minims in water three or four times a day, depending on the age and severity of the attack. Benzaldehyde may be added in amounts varying from 1 to 5 per cent., and this increases the effect. Where the cough results from a tickling in the throat, relief may be obtained by painting the lingual tonsil with tincture of iodine. Various antiseptic vapours have been used to impregnate the room which the patient occupies, such as carbolic acid or creosote.

The complications must be treated, both as regards drugs and general management, in the same way as they would be apart from pertussis. (*See* Bronchitis and Broncho-pneumonia.)

## GLANDULAR FEVER

This complaint, described by Pfeiffer, Park West, Dawson Williams, and others, consists of an inflammatory swelling of the deep cervical and other lymphatic glands associated with fever. It may occur in epidemics, is no doubt infectious, and affects chiefly children under fourteen years of age. It has an incubation period of from five to seven days. The patient is taken suddenly with stiffness in the neck, difficulty of swallowing, and febrile reaction, with anorexia and perhaps vomiting. The fauces are often reddened, but otherwise little affected. On the second or third day of pain the cervical glands, and generally those under the sternomastoid muscle and along its anterior border, are

found to be enlarged and tender. In another day or two those of the other side are swollen, and the posterior cervical, axillary, and inguinal glands may be also involved. There is generally abdominal pain and tenderness; and the liver, spleen, and mesenteric glands may be enlarged. The glands begin to get smaller after from two to five days, and do not suppurate. The temperature may reach 104° on the third day, and it will continue high as long as the glands remain enlarged. Constipation is often troublesome. The disease subsides usually without complications; but hæmorrhagic nephritis occurs in 6 per cent. of cases, and convalescence may be retarded by anæmia. The bacteriology of the disease is at present negative; but a leucocytosis of 18,000 or more, with an increase of the lymphocytes, is a characteristic change, though it is often transitory.

**Diagnosis.**—The glands enlarge rapidly and there is no anæmia. In spite of the high temperature the constitutional disturbance is usually slight. This latter feature helps to distinguish the disease from acute leukæmia. Cases are often diagnosed as tonsillitis, mumps, acute tuberculous adenitis, or acute Hodgkin's disease (**12**).

The **treatment** consists in rest, a simple diet, the relief of the constipation by small doses of mercury or salines, and the use of preparations of iron during convalescence.

## SEPTICÆMIA

Septicæmia may be defined as a condition in which micro-organisms reach the circulating blood and are carried by it throughout the body. Very often bacterial cultivations can be made from a specimen of blood withdrawn aseptically from a vein (*see* p. 87); but this depends on the number of bacteria circulating in the blood, and it is unsafe to conclude that there is no septicæmia because the culture is negative. At the same time the term should not be used for the occasional detachment from some focus, *e.g.* an abscess round the root of a tooth, of a few streptococci which may pass into the blood stream and probably get destroyed. Many specific infections produce a septicæmia quite early in the course of the disease, and it is owing to this that the infection is spread into the various parts of the body characteristic of the disease. The specific bacteria may, for instance, be cultivated from the blood in the various enteric infections, in cerebro-spinal fever, pneumococcal pneumonia, etc. In such cases the condition is spoken of as a typhoid, meningococcal, or pneumococcal septicæmia. When the term *septicæmia* is used by itself, it usually suggests the invasion of the blood stream by streptococci, staphylococci, or other pyogenic bacteria which come from some primary focus, such as a septic wound, a septic uterus or an appendix abscess.

The **Symptoms** are very similar to those of pyæmia. A rise of temperature is one of the earliest signs, and this may be accompanied by a rigor. With this the tongue is furred, and there are anorexia, perhaps vomiting, prostration, weakness, and finally all the conditions of the typhoid state, such as delirium, stupor, dry brown tongue, sallow complexion, and tremor of the limbs. Sometimes there are loose motions, and sometimes patches of erythema on the skin, or petechiæ, and there may be nephritis. The duration of the symptoms is variable, and may be from two to three days to eight or ten in fatal cases. Milder cases may recover after a much longer period. After death the conditions are found which have been described already (*see* p. 22).

**Treatment.**—If possible, a serum prepared from a variety of the organism corresponding to that infecting the patient should be employed, otherwise a polyvalent antistreptococcus serum may be tried. Vaccines may also be employed. Blood transfusion from a donor who has been immunised against the particular micro-organism has also been tried with success. Surgical measures must be carried out as required.

## SAPRÆMIA

In this condition the general symptoms are identical with those of septicæmia. It is a toxæmia due to the circulation in the blood of poisonous substances (toxins or ptomaines), which result from the action of bacteria upon necrosed tissues, and sapræmia differs from septicæmia in the fact that the organisms themselves are confined to the local lesion and do not penetrate into the blood. Efficient local treatment by removal of the source of the toxins is promptly followed by improvement, for no more toxins are poured into the blood, and those already there are eliminated in the urine or otherwise. Sapræmia is one of the complications of uterine sepsis after childbirth.

## PYÆMIA

The formation of abscesses in various parts or organs which distinguishes pyæmia may be the consequence of an open wound, accidental or operative, or of a collection of pus, in any part of the body. Pyæmia was in former times the scourge of the surgical wards of a hospital, until the almost universal use of the antiseptic methods of treatment introduced by Lord Lister. It may, however, arise from lesions which come frequently under the notice of the physician, such as ulcerations of the mucous surfaces, and which are not amenable to antiseptic treatment.

The name *pyæmia* (pus in the blood) arose from the idea that pus was actually transferred in the blood stream from the original lesion to the seat of the secondary abscesses. Abscesses, which are the distinguishing features of this disease, are really formed as the result of infarction or embolism (*see* Embolism) of minute vessels, with portions of thrombus or *débris* carrying the infective organisms, so that not only obstruction of the vessels, but also inflammation and suppuration, occur. A pyæmia may or may not be accompanied by a general blood infection (septicæmia). The position of the primary infective focus determines to some extent in what organs the abscesses will be situated, owing to the direction of the blood stream, as follows: (1) Where the lesion is situated in the peripheral systemic circulation, *e.g.* in acute osteomyelitis, abscesses or septic infarcts occur primarily in the lung. However, certain minute emboli will succeed in traversing the pulmonary circulation, so that further abscesses may be found in the heart muscle and in other organs. (2) Where the primary focus is in the lung, *e.g.* in chronic bronchiectasis, or in the heart, *e.g.* in infective endocarditis, abscesses or infarcts will occur primarily in the brain and other organs of the systemic circulation. (3) In *portal pyæmia* the primary lesion is some form of ulceration of the parts which drain their blood into the portal vein, *e.g.* an appendix abscess; and secondary abscesses form in the liver, with or without a suppurative pylephlebitis (*see* Pylephlebitis).

**Ætiology.**—Apart from accidental and operative wounds, the lesions which lay the body open to pyæmic infection are carbuncles, boils, ulceration of the intestine, ulceration of the vermiform appendix (appendicitis), fistula, gonorrhœa, septic thrombosis of the prostatic veins, otitis media, and post-partum exposure of the uterine surface. Intemperance, such general conditions as Bright's disease, and acute fevers, have been believed to dispose to the occurrence of pyæmia. It is not always possible to find the primary focus.

**Symptoms.**—The disease often begins suddenly with a prolonged rigor, followed by profuse sweating and collapse, the temperature rises, and fever continues to be interrupted by fresh rigors daily, or two or three in the day, but often without any regularity. There are anorexia, thirst, and dry tongue, anxiety, prostration, rapid breathing, and loss of flesh. The face is usually sallow, or even distinctly jaundiced, and the urine may contain some bile pigment. Sickness is not infrequent, and diarrhœa may be present. Leucocytosis is marked. The rigors may cease after five or six days, but fever of an inter-

mittent or remittent type continues; occasionally there are transient erythematous patches in various parts of the body. As already stated, the local lesions vary, and the symptoms differ accordingly. When the lungs, as is common, are the seat of secondary abscesses, the respirations are rapid, with supplementary breathing in front, deficient entry of air at the bases, and perhaps sharp crackling râles, or there are dulness, tubular breathing, and other signs of pulmonary consolidation or pleural fluid. Pericarditis or peritonitis will be shown by their characteristic symptoms. The duration of these cases is often quite short—from six to ten days; a typhoid condition ensues, with prostration, stupor, delirium, dry brown tongue, quick feeble pulse, and death.

In the *chronic* cases, where the viscera are spared, and the abscesses form in the joints, the latter become swollen, tender, and hot; tender points appear on the surface of the limbs or body, and beneath them abscesses rapidly form, with thin, unhealthy pus and imperfectly developed limiting walls. Fresh abscesses occur from time to time for several weeks or months, and the patient may ultimately recover, sometimes with ankylosis of joints, or death may take place from persistent toxæmia. The symptoms in other cases may be modified by the special localisation of the secondary lesions. In pyæmia secondary to otitis the lungs are implicated, or there may be pleurisy or empyema with the substance of the lung nearly free. If meningitis occurs, the cerebral symptoms will largely mask the others.

**Diagnosis.**—The occurrence of rigors and profuse sweatings in the course of the treatment of a wound, followed by collapse and a typhoid condition, while the wound takes on an unhealthy appearance, is characteristic of septicæmia or pyæmia. Where these symptoms occur without any external wound the same diagnosis may be obvious, and search will have to be made for the primary lesion, which may prove to be otitis with discharge from the ear, disease of the nose, intestinal ulceration, or abdominal suppuration. In the last two cases the lesions are probably confined to the portal circulation (*see* Suppurative Pylephlebitis). Sometimes the rigors take place with such regularity as to resemble *malaria;* resistance to quinine, a causative lesion, and the absence of malarial parasites from the blood would be in favour of pyæmia. *Malignant endocarditis* is generally distinguished by the presence of a cardiac murmur, but endocarditis of the pulmonary valves may be caused by pyæmia secondary to a suppurative lesion.

In the late stages pyæmia may closely resemble *enteric fever*, especially if there is no discoverable lesion to suggest the former. Rigors, however, are uncommon in enteric fever. Lastly, joint pains, like those of *rheumatism*, within a few weeks of confinement or miscarriage, should always excite a suspicion of pyæmia. In this disease the inflammation persists in each joint as it is involved, whereas in rheumatism the pains often shift from joint to joint, and may return again in those first affected.

**Treatment.**—This is almost hopeless in the visceral forms, but less unpromising in those with synovitis and cutaneous abscesses. The injection of a polyvalent antistreptococcic serum has been of great use in many cases, and should certainly be tried. Failing this, or at the same time, quinine (5 grains) or sodium sulphocarbolate (10 grains) may be given every four hours; and in any case nourishment and stimulants must be supplied freely. If the primary lesion can be reached, it should be dealt with surgically, so as to get free drainage and asepsis; and secondary abscesses should be opened where accessible.

## ERYSIPELAS

(*St. Anthony's Fire, The Rose*)

Erysipelas is a specific contagious disease, characterised by a peculiar form of inflammation of the skin, and due to the invasion of the *Streptococcus pyogenes*.

**Ætiology.**—The most common determining cause of erysipelas is the presence of a wound, whether accidental or the result of operation; and infection, no doubt, takes place through this breach of surface, and spreads to the surrounding skin. Even though it sometimes arises apparently without any wound, it will in such cases generally be found that there is a slight scratch or an abraded pimple, or other very slight lesion of the skin.

It affects infants and people over forty years of age more frequently than others; men and women are about equally prone to it. Some conditions of the individual increase the liability: chronic disease of the liver and kidneys, chronic alcoholism, and malnutrition from insufficient food. Cold and damp weather, overcrowding, bad ventilation, dirt, and bad food and water may act in the same way. There may be also an individual tendency, for it often recurs in the same person; at any rate, the immunity conferred by it seems to be short-lived.

**Pathology.**—Microscopic examination of the skin of the affected part shows that the cutis and subcutaneous tissues are swollen, œdematous, and filled with large granular leucocytes, which in the upper layers of the cutis closely surround, as well as fill, the lymphatic vessels. The disease is spread through the superficial lymph spaces. It is often checked or stopped at lines where the skin is closely adherent to subjacent parts, as, for instance, along Poupart's ligament and the crest of the ilium.

Streptococci are found in the lymphatic vessels and lymph spaces at the advancing margin of the disease, as well as in the deeper layers of the skin of the central parts; and rabbits and human beings have been successfully inoculated from cultures.

**Symptoms.**—Apart from injury and operation, erysipelas most commonly attacks the face, and the present description applies especially to that region. The *incubation* of the disease is probably only a few days—from three to six, or in some instances much longer. The *invasion* is generally by a chill or rigors and such malaise as commonly accompanies the onset of the specific fevers—headache, anorexia, furred tongue, and general pains. Within a few hours a red, tender spot shows itself on some part of the face: the side of the nose, the inner canthus of the eye, or the external ear. It may be determined by a lesion of the skin if this exists, and not infrequently it begins at the point of junction of the skin with the mucous membrane of one of the orifices—the nose, mouth, or external ear. The spot enlarges, and the skin becomes bright red, swollen, and very tender, and pits slightly on pressure. The inflammation may confine itself to one side of the face, but more often affects both, and may extend to the scalp. It spreads with varying rapidity, the advancing edge is sharply defined, thick, and raised above the surface, and small tongue-like projections can be felt under the skin in front, which is not yet reddened. The whole face may be thus covered in three, four, or five days. At the height of the disease the face presents a remarkable appearance: the features are enormously swollen, of bright or dusky red colour; the eyelids are distended so as to look like bladders; generally some muco-pus is oozing from between them; the ears are thickened and much enlarged, and the patient is absolutely unrecognisable; the scalp is also swollen and puffy. Often blebs form upon the cheeks or eyelids, which contain yellow sero-purulent or purulent fluid, and these may burst and leave yellow scabs, which further disfigure the patient. The lymphatic glands in the neighbourhood are enlarged and tender, and may be thus affected even before the beginning of obvious inflammation of the skin.

The disease is generally accompanied by high fever. The temperature generally rises early to 102° or 103°, and reaches a maximum of 104° or 105° on the third or fourth day. About the sixth day it tends to fall rather suddenly, but may remain high if the cutaneous inflammation persists, or may rise again with any fresh outbreak of the local disorder. Indeed, it is closely dependent upon the

inflammation of the skin; in some cases, perhaps more often when the erysipelas is not extensive, the temperature may not rise above 102°. The pulse is quick and full, numbering 100 to 120, or more. The tongue is covered with a thick white fur. The urine is scanty, and in many cases contains some albumin, which may be present for some days. The inflammatory condition invades also the mucous membrane; the palate, fauces, tonsils, and occasionally the laryngeal mucous membrane, may be reddened and swollen, and cause difficulties in respiration and deglutition. The blood shows a condition of leucocytosis. Delirium is common in severe cases, and is generally of a low, muttering kind; and coma may follow. While the inflammation is still advancing on one side, it may begin to subside at the points first affected. This receding edge is then less well defined, graduating both in colour and elevation into healthy skin, as contrasted with the advancing margin. The swelling, tenderness, and pitting on pressure subside in turn over the whole of the affected area; the colour fades somewhat, but mostly changes to a brown tint; and large, thick flakes of dead epidermis now begin to desquamate. This process may take some days. After erysipelas of the scalp the hair often falls out at the same time as the skin is shed, or somewhat later.

Death takes place from exhaustion, with delirium and coma, especially in older patients, habitual drinkers, and those with chronic visceral disease. It may also occur from complications.

**Complications and Sequelæ.**—Abscesses may form under the skin, or the tense skin may slough, and induration, or rarely suppuration, of the lymphatic glands may ensue. The laryngeal œdema may cause asphyxia; pneumonia and pleurisy are occasional complications, and peritonitis and endocarditis have been recorded. Pyæmia and meningitis have been frequently mentioned in connection with erysipelas, but both are rare as direct results of the erysipelas itself. The former may arise from the wound which preceded the specific inflammation; the latter may occur if the original lesion has been a fracture of the skull, or if infection spreads inwards from the orbit. In erysipelas of the scalp, the delirium may be violent or maniacal, and accompanied by delusions, but of itself this is not sufficient to justify a diagnosis of meningitis. Mental disturbance may also be a sequela.

**Diagnosis.**—Facial erysipelas may be confounded with erythema, acute eczema, herpes zoster, alveolar abscess, and even mumps. Erythema occurs in red patches, generally two or more in number, much less raised, and without pronounced fever. The vesications of an acute eczema and of herpes are distinctive, and herpes is unilateral and confined to one of the areas of distribution of the fifth nerve. Characteristic features of erysipelas are the spreading of the redness so that the maximum is always remote from the point of origin, the implication of the ear in the inflammation, and the great tenderness of skin in the advancing zone.

**Prognosis.**—Though in most cases favourable, it is dangerous in proportion to the extent of surface involved; and it is often fatal in old patients, and in the subjects of chronic visceral disease, alcoholism, or malnutrition.

**Treatment.**—Since the infection spreads through the superficial lymph spaces of the skin, it may be arrested by contracting the spaces. This is carried out by painting non-flexible collodion in a ring outside the spreading edge. The application must be repeated when the collodion flakes off. Good results have sometimes followed the use of an *antistreptococcus serum*, injected subcutaneously in doses of 15 or 20 c.c. once or twice daily. This serum is obtained from the horse after the animal has been immunised by repeated inoculation with streptococcus (*see* Diphtheria Antitoxin, p. 70). Tepid or cold sponging may be resorted to where the fever is unusually prolonged or high. The diet should be light, easily digestible and nutritious, as in other febrile cases, and in most cases alcohol in the form of port wine or brandy may be given.

## RHEUMATIC FEVER

(*Acute and Subacute Rheumatism*)

Rheumatic fever is a febrile disease in which there is acute inflammation either of the joints, or of the heart and its membranes, or of both together.[1] The rheumatic affections, which include acute and subacute rheumatism and chorea, constitute a most serious menace to children, owing to the injury done to the heart. About half the deaths in the L.C.C. Schools for the physically defective are due to this cause, and many of those who are spared for the time being become no better than chronic invalids when they grow up.

**Ætiology.**—There can be little doubt that rheumatism is due to infection by a micro-organism; and that which has been described as a diplococcus by Poynton and Paine, and as a streptococcus occurring in pairs or short chains by Beaton and Ainley Walker, is accepted by many as the cause. It has been isolated in fatal cases from the blood, cardiac valves, pericardium, and tonsils; it can be cultivated on artificial media; and lesions resembling those of rheumatism have been produced in animals by inoculation of cultures. On the other hand, many competent observers have failed to isolate the organism.

The disease occurs in both sexes, and at nearly every age; but it is very rare after fifty years of age, and in infants. The tendency to its occurrence is by many thought to be hereditary. In a few cases the disease has been found contagious. Cold and damp have generally been regarded as exciting causes, but analysis of large numbers of cases and their relations to temperature and seasons show that the disease is frequent in proportion to high temperature, hours of sunshine, and to a certain extent to humidity and east winds, but that it is actually less in times of much rainfall. In London it is more frequent in spring and autumn. It is rare in the tropics. Chorea is one of the manifestations of the rheumatic virus; and it is also associated with scarlatina.

It is probable that the rheumatic virus gains its entrance to the body through the nose or throat in the majority of cases, the tonsils constituting the commonest primary focus of the disease.

**Morbid Anatomy.**—The joints have been found in fatal cases to contain a turbid synovia, with shreds of fibrin. Leucocytes are present, but the fluid is never purulent. The synovial membrane itself is vascular, and covered with a layer of lymph. Probably the synovial changes are even slighter than this when such rapid subsidence takes place as is often witnessed. In the tendon sheaths have been found opaque serum and greenish-yellow lymph. In cases dying with thoracic complications are seen the characteristic lesions described under Myocarditis, Endocarditis, Pericarditis, and Pleurisy. Where hyperpyrexia is the cause of death, there are not necessarily any lesions other than such slight changes as may be recognised in the joints. The lungs are mostly congested, and so are other organs. Meningitis was found in two out of twenty-four cases of hyperpyrexia noted in a report on the subject by the Clinical Society. In some cases of rheumatic fever with purpura, the intestine has presented an extremely congested and ecchymosed mucous membrane.

**Symptoms.**—In some cases rheumatic fever follows immediately upon an attack of tonsillitis. In others there is often a previous history of one or more attacks; and the tonsils are seen to be enlarged, or to contain deep crypts, or to exude pus on pressure, or adenoids may be present. The first symptoms of the rheumatism may be those of a multiple arthritis or synovitis, lesions which are

[1] The terms *rheumatism* and *rheumatic* have been both popularly and in medical works used with little discrimination for many disorders in which *joints* or *pains* or *cold* are concerned, the last perhaps more correctly, for the name is derived from ῥέω, I flow, and is associated with the word catarrh, καταρρέω. But it is very desirable that the terms should be restricted to the disease now being described, and to conditions presumably allied to it.

obvious because painful and accompanied by pyrexia; in other cases the first lesion is inflammation of the cardiac valves, muscle, or covering membrane, which may be entirely latent and insidious (subacute rheumatism). The first condition is commoner in adults, while the second forms a large proportion of the cases in children. But the carditis occurs as a complication in the first group, and joint affections may occur at some or other stage in the second.

The *joints* may be affected quite suddenly. The knee is often first attacked, and then the ankle, in other cases the wrist or the shoulder. Whichever is first attacked, the disease may soon spread to other joints of the body, so that the shoulder, elbow, wrist, and phalangeal joints, the hip, knee, ankle, and phalangeal joints of the toes, may all be inflamed at the same time or successively. Occasionally the sterno-clavicular joint and even the vertebral and costo-vertebral joints are undoubtedly affected. But the extent of the disease is very variable. In one only two or three joints may be inflamed, in another a great number; and an important feature of rheumatic fever is the way in which some inflamed joints will quickly recover while others become involved; and these last will get well while fresh joints suffer, or those first affected become again inflamed.

A joint attacked by rheumatic fever is swollen, red, hot, tender to touch, and painful. The swelling is most manifest in the knee where effusion can easily be recognised, in the ankle, in the wrist, and in the joints of the fingers. The colour is mostly a bright pink, and not the dark red of gout and some erythemas; it rarely covers the whole swelling, and may be in patches. The tenderness is sometimes extreme, so that a slight shock on the bed, and any clumsy handling of the joint, will cause intense pain. It may persist after spontaneous pain has subsided. In the shoulder, hip, and elbow joints, pain and tenderness are the chief evidences of rheumatism, as slight swelling is not easily recognised, and redness is generally absent.

In the vast majority of cases the synovitis clears up without leaving any sign of previous inflammation; but occasionally, especially if the same joint is repeatedly attacked, permanent alterations may be seen, with wasting of the muscles. These may be associated with rheumatic nodules, and resemble very closely acute rheumatoid arthritis. It has been stated that the synchondroses are also sometimes involved in rheumatic fever. Undoubtedly the sheaths of the tendons about certain joints are often inflamed, especially those about the wrists and ankles; and some of the redness that extends on to the dorsum of the foot and hand may be due to their inflammation.

With this multiple arthritis there is always associated some *pyrexia*. It is variable both in intensity and duration. It does not commonly rise above 103° F., oscillates with some irregularity, and mostly subsides with the inflammation of the joints. It may last nine or ten days if salicylates are not given, but it often ends sooner; and it recurs with any recurrence of the arthritis. It is also influenced by the cardiac lesions, especially pericarditis, or by pleurisy; and it sometimes rises to a great height, becoming thus a dangerous complication (*hyperpyrexia*). Profuse *sweating* is a characteristic of rheumatism. The sweat has a peculiar sour smell, but the reaction is not always strongly acid, and may be even neutral. The sweat may often be seen in clear vesicles at the orifices of the sweat pores, called *sudamina;* when the vesicles contain a point of pus, and are surrounded by a pink areola, they are known as *miliaria*. The fever is not generally accompanied by much cerebral disturbance, and delirium is not a marked feature in uncomplicated rheumatic fever. The *tongue* is usually large, broad, flabby, and covered with a thick, white, creamy fur. The appetite is bad, and the bowels are constipated. The *urine* is scanty, high-coloured, and acid; it contains only occasionally a trace of albumin.

In a large proportion—between a third and a half—of the cases of rheumatic fever beginning with arthritis, the *heart* is afterwards found to be affected by

one or more of the following lesions: endocarditis, pericarditis and myocarditis. *Endocarditis* commences almost invariably in the valves, and in the valves of the left side. Its occurrence is sometimes marked by some increase of fever, or by quickened action of the heart or acute heart block; but in most cases it is revealed only by auscultation, when a soft bruit may be heard muffling or replacing the first sound of the heart, either at the apex or at the base in the aortic area. It is, however, probable that this murmur when occurring at the apex is really due to endocarditis, and not to myocarditis weakening the muscle, and allowing regurgitation through the mitral valve. The murmur in any case may disappear in the course of the illness, or may persist into convalescence. Other physical signs are described under Acute Endocarditis (p. 329). The patient is often, but not always, anæmic. Exceptionally pronounced heart failure shows itself within a few months of the rheumatic attack. *Pericarditis* may accompany endocarditis, but is very much less frequent; its onset is more often attended with subjective symptoms, such as præcordial pain or distress, local tenderness, rapid action of the heart, and occasionally considerable elevation of the temperature. Friction sound is usually the first physical sign, and increase of præcordial dulness from effusion soon follows: the dulness may extend upwards to the first intercostal space, an inch or more beyond the left nipple, and an inch and a half to the right of the middle line; but the effusion is rarely sufficient to prevent persistence of the rub as long as the inflammation continues. *Myocarditis* is probably always present when the heart is affected in rheumatism. It is probably responsible for the shortness of breath and exhaustion after exercise which may be the earliest symptoms of the disease. It may be the cause of various cardiac irregularities and occasionally of acute dilatation of the heart.

*Pleurisy* is often seen when the heart is severely affected, and especially in association with pericarditis. A rub is heard, and there may be pain. When situated between the pericardial and visceral pleura, pleuro-pericardial friction may be present (*see* p. 195). There is not usually sufficient exudation to require tapping; but occasionally it may be extensive and give rise to physical signs at the left base, resembling the pressure of a pericardial effusion on the left lung—impairment of note, deficient vesicular murmur, or weak high-pitched bronchial breathing. *Acute pulmonary œdema* and *broncho-pneumonia* occur in children occasionally (Poynton). Laryngitis, venous thrombosis, nephritis and meningitis are rare complications.

Besides the sudamina and miliaria already mentioned, urticaria and different forms of erythema may occur, especially *E. multiforme*. *E. nodosum* is also associated with rheumatism. It may be of streptococcal origin (*see* p. 969). Occasionally one sees a purpuric eruption complicating rheumatism (*peliosis rheumatica*). This appears mostly on the feet, ankles, and lower parts of the leg, as a more or less continuous bright red eruption, made up of numerous small red petechiæ, which do not disappear under pressure. They commonly last only a few days, and give place to brown or yellow staining as they subside. Sometimes the purpuric spots are much larger and more generally scattered.

A very dangerous but happily rare complication is *hyperpyrexia*. In a certain proportion of cases, some warning is given: the joint pains subside rather suddenly, and the sweating ceases; the patient becomes restless, and after a few hours talkative or even delirious. The temperature, formerly perhaps under 100°, is now found to have risen to 105° or 106°, and unless measures be quickly taken to reduce it, it rapidly reaches 107°, 108°, even 110° and 111°. The delirium is at first moderately active, the muscles twitching, and the eyes restless; the patient may try to get out of bed. The face is generally dusky red, and the tongue dry and tremulous. When the temperature exceeds 107°, the patient becomes semi-comatose, or even quite comatose, a condition which is always present if the thermometer marks 110°. Under suitable treatment

for reduction of the temperature recovery often takes place; but otherwise the respiration becomes more frequent and shallow, the face more dusky or livid, the pulse rapid and feeble, râles accumulate in the chest, and death ends the scene, often within twelve or twenty-four hours of the first indication of hyperpyrexia.

*Rheumatism in Children.*—This may be in every respect like that already described; but in a great many cases children are found to have evidence of a cardiac lesion without any history of illness, or of pyrexia, or of the joints having been inflamed, or of anything more than vague pains, perhaps called *growing pains*, which have not even confined the child to its bed. The cardiac lesion may be any one of those above described, and may be even in an advanced stage when first recognised. Sometimes a child attacked by the articular form of rheumatism is found to have a cardiac lesion obviously of old date, not dependent upon the present synovitis, but without any history of a preceding synovitis. The relations of *chorea* to rheumatic fever will be discussed later (*see* Chorea); but it is more frequent in children and occurs in association with cardiac lesions,

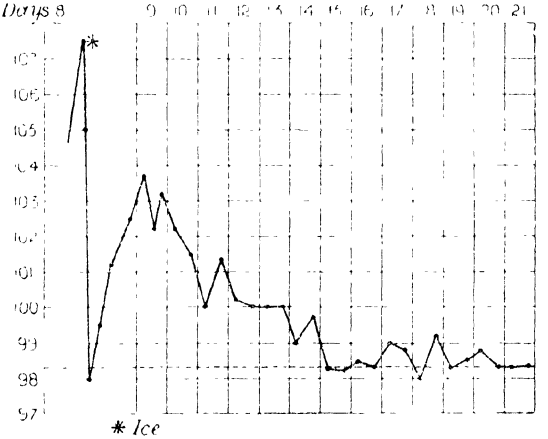



FIG. 5.—Hyperpyrexia in Rheumatic Fever. *Recovery.*

with the subcutaneous nodules described below, and sometimes with, and at other times without, synovial inflammation. If it occurs in a child with no previous evidence of rheumatism, endocarditis or swelling of the joints will sometimes follow.

*Subcutaneous nodules* are also more common in children than in adults, and are found in the neighbourhood of joints, and over bony ridges and prominences elsewhere. They are freely movable under the skin, and slightly on the fibrous structures beneath them; and they consist of spindle-shaped nucleated cells —fibroblasts—and some vessels. They last a variable time, and may disappear in a few weeks.

**Course of Rheumatic Fever.**—If untreated, the symptoms in the joints may last from ten to fourteen days, when they will often subside; if treated by salicylates, the pains and fever are often gone within a week. In any case, however, rheumatism shows a great tendency to *relapse*, the joints being affected in a precisely similar manner after an apyrexial painless interval of from two days to a fortnight. It is possible that in such cases the organisms persist in the joints through the apyrexial periods (Beattie). In such a relapse the patient runs just the same risk in regard to the heart, and to the occurrence of hyperpyrexia, as he does in the first attack. Another relapse may succeed, or irregular affections of now one, now another joint, with or without marked pyrexia.

Occasionally recovery is delayed by the persistence of the inflammation in one joint for weeks or months; pain, swelling, and stiffness are prominent troubles, and the joint has ultimately to be treated, with rest, splints, and local treatment. Another cause of delay in convalescence has already been mentioned—namely, the rapid progress of an endocarditis, so that the patient passes at once from rheumatism into pronounced heart disease, with murmurs of aortic or mitral disease and failing cardiac muscle.

Death takes place in the course of an attack chiefly from the thoracic complications, especially when endocarditis, pericarditis, and pleuritic effusion on one or other side occur simultaneously, more rarely from hyperpyrexia.

**Diagnosis.**—This usually presents no difficulty, the sudden occurrence of joint pains, with redness and swelling, fever and profuse sweating, being mostly decisive, especially if it occurs in young persons with previous good health, or, on the other hand, with a previous history of rheumatic fever or of heart disease. It must be distinguished from *acute anterior poliomyelitis*, in which, however, the pain is only present on movement, and there is no effusion into the joints, and the knee jerks are absent. Where one joint alone is affected, *acute osteomyelitis* must be considered. Here the constitutional symptoms are more severe; there is great tenderness, especially when pressure is exerted on the bone. *Acute pyæmia* with suppurative arthritis is uncommon, and there is usually some obvious focus of infection, such as a septic uterus after childbirth. The joints when attacked take a long time to clear up. *Gonococcal synovitis* is also more persistent than rheumatic fever, and is only rarely accompanied by cardiac complications, and the inflammation particularly tends to involve fasciæ; but in early stages it may be readily mistaken for rheumatism both in men and women, until the presence of a discharge is ascertained on inquiry, or admitted by the patient. Acute multiple arthritis may also occur in many specific infections, *e.g. enteric fever, dysentery*, and *pneumococcal infections, relapsing fever*, etc. The reaction of the patient to salicylates is a valuable indication of acute rheumatism. The diagnosis from *gout* will be given with the description of that disease.

The diagnosis of rheumatism in a child who has not had a multiple arthritis depends on the recognition of sore throat, the cardiac lesion, subcutaneous nodules or chorea, and the inference, perhaps not always justified, that rheumatic infection must be their cause.

**Prognosis.**—The mortality of acute rheumatism is small. Most danger is to be apprehended from the coincident cardiac infection and the rare occurrence of hyperpyrexia. The former is considered later on (p. 322). Hyperpyrexia is dangerous in proportion to the temperature reached before cooling measures are adopted. In any case the possibility of future cardiac disease has to be remembered.

**Prevention.**—The most important point is to treat all possible sources of infection; in this connection the nose and sinuses and throat should be examined with care, especially in children, while in adults dental infection in particular should be looked for, and tonsils and adenoids removed if necessary. A clay soil is usually regarded as predisposing to rheumatism, and low-lying situations in river valleys should be avoided. Children who are rheumatic should change their clothing if it becomes damp. (*See also* Prevention of Heart Disease, p. 347).

An important statistical study has appeared on the subject of tonsillectomy carried out on eighty-five children with various rheumatic manifestations (**6**). In most cases the tonsils had been inflamed more than once, and in all cases the tonsillar lymph glands were enlarged. The cases were followed up over an average of three and a half years after tonsillectomy. The tonsillar lymph glands became impalpable in 59 per cent. of cases. Of cases who had had rheumatic fever, there was no recurrence in 84 per cent. As regards chorea, there was no recurrence in 50 per cent. of cases. As regards myositis and joint pains, there was no recurrence

in 77 per cent. of cases. In a few cases a second operation was necessary to remove the tonsils completely. Starling (7) has obtained very similar results in thirty-five cases.

The following conclusions may be drawn: In rheumatic children complete removal of the tonsils by means of enucleation is desirable when the tonsillar glands are enlarged; the operation should be carried out after an acute infection has subsided, but should be carried out at once in apyrexial attacks of rheumatism and chorea.

**Treatment.**—For the efficient treatment of even mild cases of rheumatism complete rest in bed is absolutely necessary so long as there is any active infection present (*see* p. 347); in severe cases the patient cannot do otherwise than lie still. The joints should be protected from every risk of injury. Sometimes it is desirable to raise the bedclothes from the limbs by a cradle; and some local relief to the pain may be obtained by wrapping them round with cotton wool, upon which, in severe cases, a little anodyne, such as belladonna or opium liniment, may be sprinkled, or methyl salicylate (artificial oil of wintergreen) may be spread on the joint and covered with guttapercha tissue. In the past a strict milk diet has usually been prescribed, but on general principles it is much better to allow a more varied dietary, as in other febrile conditions.

The drug now almost universally employed is sodium salicylate, and sometimes salicin or salicylic acid. When the patient is fully under the influence of one of these drugs, the pains disappear, the redness and swelling of the joints subside, and the temperature falls two or three degrees—it may be to the normal. If the drug is then lessened or discontinued, the pains will most likely return; if the dose is maintained, the rheumatism may be practically cured from that time; but the treatment, both by drug and diet, will have to be continued for ten days or more, at the end of which time some relaxation may be cautiously allowed. An efficient dose of either salicylic acid or its sodium salt is 20 grains, and of salicin 30 grains, every four hours during the first twenty-four or thirty-six hours; but in less severe cases a smaller quantity may suffice. Some give a smaller dose every hour for the first four or five hours, and then diminish the frequency to every two hours. If the attack is very severe, it may be desirable to give a 20-grain dose every two or three hours for the first day. If too much is given, the patient suffers from headache, deafness, tinnitus aurium, and slight delirium, which cease when the drug is withdrawn. Occasionally vomiting, a slow or irregular pulse, albuminuria, epistaxis, or hæmaturia has occurred. As a rule, the earlier toxic symptoms coincide with the subsidence of the pains; but, this having been obtained, the frequency of the dose must be reduced to four times or three times a day, at which rate it should be continued until five or six days have elapsed from the last pain or the last abnormal temperature, when the drug may be stopped altogether. When a milk diet has been given, it is usually increased about this time by the addition of farinaceous food, and after a few days meat broth, fish, and finally meat, are given.

There is no material difference in the effects upon rheumatism of the three drugs under consideration. The sodium salt is generally preferred, and it is frequently combined with an equal quantity of sodium bicarbonate. Sometimes these drugs are not so successful; the pains continue in abated form, or relapses frequently occur. Salicylate of quinine (2 to 6 grains) may then be useful; or recourse may be had to the old alkaline treatment—potassium bicarbonate or acetate, 20 grains every four hours—or to potassium bicarbonate with quinine. Compounds containing salicylic acid, such as salol, salophen, and acetyl salicylic acid (aspirin), have some influence upon the pains of acute rheumatism. The last has been largely used in 10 or 15-grain doses in cachet.

It is not known at present how salicylates act. Many authorities believe that they have no specific action on the infection itself. It has, indeed, been suggested that there is a disadvantage in abolishing the pyrexia and the joint pains,

because an indication of the progress of the infection in the heart is thereby lost, and a certain measure of pyrexia may be a useful defence against the infection.

The treatment of the cardiac complications is described under Diseases of the Heart. The one essential is that the patient should remain in bed for several weeks, or two or three months, to give the valves or cardiac muscle time to recover.

The treatment of hyperpyrexia must be prompt and energetic; it consists in the application of cold externally whenever the temperature is found to be rising above 105° F. Salicylic acid and other antipyretics do not lower the temperature with sufficient rapidity, and the best method of reducing it is by the application of blocks of ice, as described under Heat Stroke (p. 27).

## INFLUENZA

This term, often wrongly applied to any severe nasal catarrh, is the name given to an acute febrile disease which in past times has frequently swept as an epidemic over Europe, but which was practically unknown among us after the violent outbreak of 1847–48 until the winter of 1889–90, when it again appeared. On this occasion it was first observed in Bokhara in the preceding May; it appeared at St. Petersburg in October, and soon invaded Austria, Germany, France, England, and other European countries, as well as the United States of America. A few months later it was conveyed to India, Australia, New Zealand, the African coast, and South America. The disease has again frequently broken out in the British Isles, and of late years has rarely been entirely absent. The last great epidemic, which appeared first of all in Spain, broke out in England in the form of three waves: the first and slightest in point of mortality in June and July, the second and most severe in November, 1918, and the third in February and March, 1919. It is a characteristic of influenza that, while children and elderly people usually escape death, the disease is particularly fatal between twenty and forty years of age.

**Ætiology.**—The true epidemic invasions of influenza have always been characterised by the extraordinary rapidity with which the population has been attacked, especially in crowded towns. Hundreds have been struck down at the same time, or within a few days, and this, among other circumstances, led to the view that the disease was not contagious from man to man, but was borne by the air simultaneously to many people. This feature was especially marked in 1889–90. But that the disease was spread by human intercourse, however sudden the outbreak may have been, has now been amply confirmed.

In 1892 Pfeiffer discovered a minute bacillus, which is present at some time in the sputum of influenza patients and less commonly in the blood. It is called the *Bacillus influenzæ*, and until recently has generally been regarded as the cause of influenza. It is a minute rod, not exceeding 1·5$\mu$ in length and 0·3$\mu$ in thickness. It is negative to Gram's stain, and grows best on media containing hæmoglobin or blood. The bacillus has been cultivated post mortem in 98 per cent. of cases of pneumonic influenza. But it is also present in various parts of the respiratory tract in a smaller proportion of people who have died of quite other diseases (**8**).

However, evidence has recently been forthcoming (**9**) that the cause of the disease is a minute filter-passing organism, similar to that which causes the common cold. It is obtained from the nasal secretions of patients early in the disease, when the *B. influenzæ* is often absent, and influenza has been produced by cultivation of this organism, which has been named the *Bacterium pneumosintes*. The *B. influenzæ* probably plays an important but secondary *rôle* in the disease by producing the various respiratory complications. Infection takes place by inhalation of droplets of sputum or saliva projected into the air during

coughing or speaking. An attack of influenza protects the subject for some months at least, but the acquired immunity is not indefinite. In the last epidemic about 20 per cent. of patients had had previous attacks.

**Morbid Anatomy.**—Influenza is a septicæmia, as indicated by blood cultures, by the prevalence of acute nephritis, and by the changes in the lungs. The appearances of the latter are extremely variable. Tracheitis, bronchitis and bronchiolitis are always present. There may be extreme congestion of the lungs, hæmorrhagic œdema, or diffuse hæmorrhage without consolidation, or on the other hand solid hæmorrhagic areas involving a whole lobe, or occurring in patches like infarcts. There may be broncho-pneumonia or multiple abscesses, usually small and aggregated, or actual gangrene. There may be multiple small areas of collapse or massive collapse. Peribronchitis and interstitial emphysema may occur, and also pleurisy with or without effusion. The bronchial glands are inflamed. In most cases the sphenoidal sinuses and other accessory air sinuses are infected. The primary focus of the infection in the body is probably situated in the nasopharynx. The kidneys contain excess of blood, but otherwise look normal (*see* Acute Nephritis).

**Symptoms.**—The incubation period is from just under forty-eight hours to five days. There is the greatest possible variety in the manifestations of influenza. In a large number of cases the symptoms are those of an acute febrile illness, without special determination to any one organ or system of the body. This may be described as the *simple* type, or *simple febrile* type.

The disease begins suddenly with severe frontal headache, pains at the back of the eyes and muscular aching and pains in the muscles of the loins, thighs, calves, and other parts of the body. Rigors are often absent, but the temperature rises within a few hours to 102°, 103°, or 104°. The other accompaniments of fever are present, such as quick pulse, thirst, and scanty, high-coloured urine. The tongue is flabby, tremulous, indented, and covered with a thick white fur. The fauces and tonsils are red, and the breath is offensive; epistaxis is fairly common. The skin is generally dry, but there are sometimes profuse perspirations. The spleen is sometimes slightly enlarged. The patient is exceedingly ill, restless, sleepless, prostrate and depressed. No other symptoms may appear, and the temperature falls in twenty-four, thirty-six, or forty-eight hours as rapidly as it rose; but the general pains in the limbs continue for some time after the temperature has fallen, and the sense of prostration, which is present from the first, persists for some days after the fever. However, it must be admitted that there is much variety in the course and duration of cases in this group; and that while in some the fever is high, of short duration, and falls rapidly, in others the course is longer, and the fall of temperature more gradual, so that a confusion with other febrile illnesses, such as typhoid fever, is rendered possible. In either case there may be a relapse.

In the *pneumonic* type of the disease, which occurred in about 20 per cent. of cases ill enough to be admitted to hospital at Aldershot during the 1918–19 epidemic, the commencement presents the same features, namely, fever, headache, pains in the limbs, and prostration; but it is soon seen that the respiratory tract is largely involved. There are rapid breathing, pain in the chest and troublesome cough. The signs in the chest are extremely variable: there may be no signs except a few scattered rhonchi in front and some crepitations behind; again, the signs may resemble those of a lobar pneumonia; again, râles may be heard everywhere with no signs at all of consolidation; again, consolidation may appear in one part of the lung to be replaced on the next day by vesicular murmur, but to appear in another part of the lung; again, signs of fluid may be present. The prognosis cannot be gauged at all by the extent of consolidation of the lung. The sputum may be either purulent and abundant or blood-stained, tenacious, frothy, and rather scanty. In the most serious cases there is a uniform heliotrope cyanosis over the face, associated with marked deficiency of oxygen in the

arterial blood. Nephritis occurs in the majority of pneumonic patients. There is no œdema usually, but the urine contains albumin and casts. Nasal catarrh with suffusion of the conjunctivæ is occasionally a condition of influenza, but both the simple and respiratory forms commonly occur without them.

The *abdominal* type is less frequent, but varies with different epidemics. The patient has abdominal pain, diarrhœa, vomiting, and occasionally jaundice. The temperature is often less high than in the preceding forms.

Both the respiratory and the gastro-intestinal symptoms may appear to be rather complications and sequelæ than parts of the original disease; that is, the fever and pains may be present for a few days before either of these systems is manifestly involved. Other systems are also involved more often secondarily or rather late in the history. Sometimes the pulse is irregular or intermittent, due to auriculo-ventricular or sino-auricular heart block; there may be syncopal attacks. Tachycardia also occurs, and the heart may show evidences of dilatation. Hæmorrhages from the different mucous surfaces are sometimes observed.

The nervous system is frequently involved. Drowsiness occurs in early stages, with delirium in severe cases. Later there may be sleeplessness, a persistent neuralgia, or muscular pains. In a large proportion of cases, and without any special localisation of symptoms in the nervous system, there is prolonged weakness of the limbs, inability for physical and mental exertion, and great mental depression lasting for weeks after the beginning of the attack. The skin is occasionally the subject of eruptions in the height of the attack, or a little later. These are mostly in the form of rose-coloured spots, or erythematous rashes like those of measles, scarlatina, or urticaria; alopecia may occur. In addition there is scarcely any local inflammation that may not in some case or other appear as a sequel of influenza; for instance, otitis, which is rather common, orchitis, peripheral neuritis, myositis, phlebitis, parotitis, pericarditis, meningitis, encephalitis, myelitis, conjunctivitis, keratitis, arthritis, and lymphadenitis. Amongst functional nervous troubles not hitherto mentioned are loss of taste and smell and mental breakdown in the form of melancholia or delusional insanity.

Early in the disease the blood shows a polymorphonuclear leucocytosis. This is followed by a leucopenia with a deficiency of polymorphonuclear cells and a relative lymphocytosis (**10**). If pneumonia supervenes there is a leucocytosis with a great increase in polymorphonuclear cells. The hæmorrhagic nature of the pneumonia may be associated with the diminution of blood-platelets (*see* p. 542), which has been observed.

**Diagnosis.**—The great variety that influenza presents will lead to its being diagnosed in the early days of an illness, when further acquaintance with the case may show it to be some other febrile complaint, such as pneumonia, and especially enteric fever (*see* p. 87). The very sudden onset, the local pains, and the short fever are the chief distinguishing points of influenza; but there are slight cases which can only be diagnosed by way of exclusion, by the amount of depression succeeding it, or by its complications and sequelæ. Cases with pulmonary or bronchial lesions may give the opportunity of finding Pfeiffer's bacillus in the sputum.

**Prognosis.**—Death only occurs in a small proportion of cases attacked. Cyanosis in a pneumonic case is always a bad sign, and if the temperature suddenly falls in such a case, it is almost hopeless.

**Prevention.**—There is statistical evidence that prophylactic inoculation with a stock vaccine containing 400 million *B. influenzæ*, 80 million streptococci and 200 million pneumococci prevents the onset of influenza. It is important for all persons in attendance on influenza patients to wear masks consisting of several layers of butter muslin. In the last epidemic such masks were worn with effect by people in crowded places. The evidence in favour of taking

quinine as a prophylactic is not good. The instillation into the nose of Glegg's paraffin and vaseline mixture, so effective against coryza (*see* p. 197), is well worth a trial during an influenza epidemic.

**Treatment.**—The patient should save his strength by at once taking to his bed. In the early stages the severe pains call for treatment, and may be met by sodium salicylate (10 to 15 grains every four or six hours), by aspirin (7 to 10 grains), or by phenacetin (5 to 6 grains). The great tendency to prostration after the illness makes it necessary to give these drugs with caution. Instead of them, salines (10 or 15 grains of potassium citrate or ½ ounce of liquor ammonii acetatis) may be given in the early stages, combined with expectorants, if there be much bronchial complication (ammonium carbonate, 3 to 5 grains, or tincture of senega, ½ to 1 drachm). In cases of cyanosis it is essential to give oxygen practically continuously. (For methods of administration, *see* p. 234.) As the fever subsides a tonic regimen is often given. Quinine and nux vomica are especially useful, and in older patients stimulants are also needed.

The local manifestations of the disease require to be treated as they would be if arising under other circumstances.

## TRENCH FEVER

During the great European war there was observed, at first almost exclusively in men returning from the trenches, but later behind the front, a form of febrile disease which presented new features, and was essentially different from anything formerly recorded.

**Ætiology.**—The virus of trench fever has been experimentally transmitted (McNee, Renshaw, and Brunt) to other men by intravenous injection of blood from a patient. More recently it has been proved conclusively that the disease is conveyed by lice. It has been transmitted by allowing lice that have fed

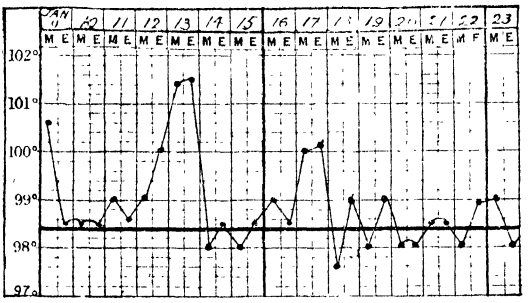



FIG. 6.—Chart of Temperature in Trench Fever.

on patients to feed on healthy men, and also by rubbing the excreta of infected lice into abrasions of the skin. There is good evidence that the virus of trench fever is a minute body, which grows in pairs like a diplococcus, measuring 0·3$\mu$ by 0·3 to 1·5$\mu$, and called a Rickettsia body. Although these bodies are difficult to find in patients, they appear in the body of the louse just when it reaches its most infective stage, *i.e.* five to eight days after feeding on an infected patient.

**Symptoms.**—The incubation period is from five to thirty days. The onset is somewhat sudden, and the patient suffers from headache, giddiness, sometimes nystagmus, shivering, and pains in the back and legs. Occasionally he vomits. There are three characteristic types of temperature chart: (1) The single short bout of fever, making the case resemble one of influenza (Fig. 6). This is

called the *short* form of the disease. The temperature rises in the first day or two to 103° or 104° F., and the pulse is about 100 in the minute. The fever lasts three or four days or even more, the temperature falling sometimes suddenly, at others more gradually, in which case the uniform fall may be interrupted by a small evening rise. During this fever there is no other clinical condition by which the disease can be distinguished. There is some tenderness in the back and legs which are the seat of pain, and there is cutaneous hyperæsthesia over the shins; but there are no spots, and no diarrhœa. There is, however, some constipation. The spleen is enlarged. On the fall of temperature to the normal the patient is perfectly well. (2) The *relapsing* type. The patient has an attack like that just described. After an interval of three or four days he is attacked by the same symptoms, and the temperature again rises, though not quite so high as in the original fever (*see* Fig. 6). Sometimes the illness ends with this relapse; in other cases a second relapse occurs three or four days after the ter-

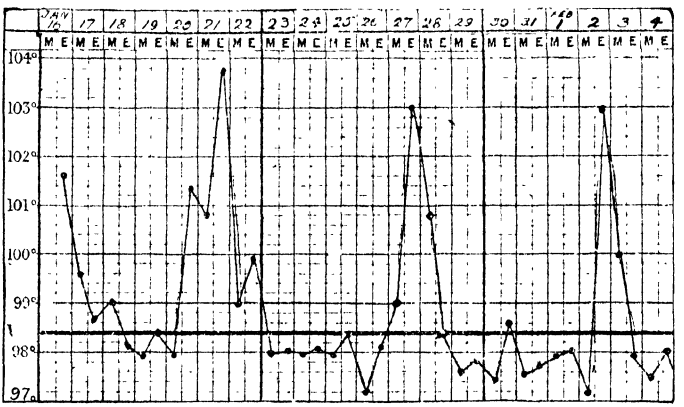



FIG. 7.—Chart of Temperature in Trench Fever.

mination of the last, and even a third or fourth (Fig. 7). In some cases the attacks do not come at such regular intervals, or there may be some irregular temperature in between. It has been noted that, when the relapses have been frequent, the primary fever has been less intense and less prolonged than in cases without relapse or with one relapse only. In the relapses the temperature may not be above the normal for more than forty or forty-eight hours, and thus if the temperature is not taken the relapse may be overlooked. (3) The type with the *prolonged initial fever*, making the case resemble one of typhoid fever; but the symptoms are the same as in the other types.

The disease may last for six or seven weeks, and it is not fatal. Tachycardia frequently develops, the disease being a common cause of effort syndrome in soldiers.

**Diagnosis.**—It is necessary to exclude the enteric fevers, and for this the agglutination test may be used. Malaria and relapsing fever can be detected by an examination of the blood. A confusion with influenza is easy in the short form of the disease. The points against it, enumerated by Rankin and Hunt, are—that catarrh is absent, that the patient is not very ill, that the headache is not severe, that there are no complications, that there is often leucocytosis, and that Pfeiffer's bacillus cannot be found.

**Treatment.**—This can only be conducted on the general principles applicable in other fevers. Quinine and aspirin have been given, and sodium salicylate may relieve pains. Salvarsan is said to be of no use.

## DIPHTHERIA

Diphtheria (from διφθέρα, a prepared hide, piece of leather) is an acute infectious disease, of which the essential clinical feature is a peculiar inflammation of surface tissues resulting in the formation of a so-called "membrane." This commonly affects the mucous membrane of the mouth, pharynx, nose, or larynx, more rarely some other mucous membrane (conjunctiva, vagina), or the abraded skin, or the surface of a wound.

The specific micro-organism of diphtheria is the bacillus described by Klebs and Loeffler. It is a short rod, but varies in length in different circumstances, from 2·5$\mu$ to 6$\mu$, so that short, medium, and long varieties have been described. It is slightly curved, and often clubbed at one end; it is non-motile, and does not form spores. It stains well with aniline dyes, by Gram's method, and with Loeffler's methylene blue, showing "beading." The *B. diphtheriæ* is found for the most part in the deep layers of the diphtherial membrane, but may be present in small numbers in the lymph glands, and in the liver, spleen, and kidneys.

**Ætiology.**—Diphtheria is contagious, being conveyed directly, as in kissing, or by means of droplets of saliva, projected into the air as the result of coughing or talking, as well as by clothes and other objects. The bacillus survives drying, and has been found in the dust of the sick-room, and there is evidence to show that it may be sometimes transmitted over considerable distances of country by the wind. Milk is a well-known channel of infection; but this is due to human contamination, and not to any disease of the cow. Water sewage and sewer gas are not known to be vehicles of infection.

The *diphtheria-carrier* is also a source of contagion. In one-eighth of convalescent cases the bacillus is found in the throat for one or two months after the first symptom, and in a few cases for three or four months (*convalescent carriers*). From 8 to 30 per cent. of those in contact with a case of diphtheria become *contact* carriers (*see* pp. 12, 81); but in the later periods of the infection the bacilli have often lost their virulence, and in any case a carrier is not necessarily an active source of contagion. Diphtheria bacilli have been found in 10 per cent. of Baltimore school children. These were classified as *healthy* carriers, as only a very small proportion had ever been in contact with a case of diphtheria. In the majority of these healthy carriers, the bacilli disappeared in a few weeks without treatment. They were non-virulent, and remained so when inoculated into the throats of five healthy persons. Virulence can be tested by inoculating a guinea-pig, and from these experiments it would seem advisable to make this test before deciding to isolate and treat diphtheria carriers.

Diphtheria sometimes complicates measles and scarlatina (about 2 per cent. of each at the M.A.B. hospitals); it is more frequent in rural than in urban districts, especially in the more exposed parts of the former; and it affects both sexes and all ages, but it is especially frequent in children up to ten or twelve years of age. Its maximum incidence is in October and November.

**Pathology.**—The inflammatory change which is characteristic of diphtheria is the formation of a "false membrane." It is the combined result of necrosis of the superficial tissues and the exudation of fibrin and leucocytes. A membrane which does not extend beyond the base of the epithelial layer is called a "croupous" membrane, and can easily be pulled off without bleeding. This is the case in the trachea in diphtheria, where it consists chiefly of fibrin and leucocytes, and is loosely attached to the surface. In the fauces, on the other hand, the stratified epithelium is infiltrated with fibrin as well as the subepithelial connective tissue, and necrosis takes place, causing the formation of a greyish white or white layer firmly adherent to the deeper tissues. This is called a "diphtheritic" membrane. It is difficult to remove and leaves a bleeding

surface. In the smaller bronchi the exudation is purulent; the lungs often present lobular pneumonia, with occasional hæmorrhages.

Diphtheria toxin has a profound effect: (1) On the *circulation*, causing a fall in blood pressure and concentration of the blood, the percentage of hæmoglobin being greatly increased. The plasma passes out into the tissue spaces. In fatal cases the cells of the suprarenals show chromatolysis (Harding). (2) On *nerve fibres*, causing disintegration of the myelin sheaths and rupture of the axis cylinders. Degeneration of anterior horn cells has also been described. The toxin primarily attacks the nerves locally, and this explains the fact that paralysis of the palate is such a common accompaniment of faucial diphtheria. Similarly in wound diphtheria the paralysis affects the muscles in the neighbourhood of the wound (Walshe). It is possible that it passes up the nerves to the central nervous system, like tetanus toxin and the virus of hydrophobia.

It is remarkable that in *laryngeal diphtheria* this toxic action is not very noticeable. Possibly this is due to the membrane being "croupous" in character, so that toxins are not readily absorbed.

The changes in the various organs are attributable to the influences of the toxins circulating through the body. The heart may be pale, soft and friable; the muscular fibres show a cloudy swelling and fatty changes, and blood is extravasated. The convoluted tubules of the kidney show fatty degeneration, and the epithelium is in many places separated from the basement membrane.

Streptococci and staphylococci are often present in the superficial layers of the diphtherial membrane, and sometimes lead to secondary suppurative lesions.

**Symptoms and Course.**—The *incubation* lasts from two days to four days (limits one to six).

*Faucial Diphtheria.*—The disease, though febrile, begins insidiously; there are generally malaise, loss of appetite, and headache, and there may be nausea, vomiting, or shivering. Sore throat is soon complained of, and it is seen to be inflamed. Within a short time one or more patches of a creamy white deposit form on the inflamed surface. There are *five* areas where such patches occur in diphtheria, between the pillars of the fauces on both sides, *i.e.* over the tonsils (2); the uvula (1); the soft palate on both sides (2). It is characteristic that there is never more than one patch in a particular area, but any number of the areas may be affected at the same time. The patch is raised above the surface of the mucous membrane, and the edges are sharply defined; the colour may be glistening white, bluish, yellow, or grey. Coincidently with the inflammation of the throat the lymphatic glands at the angle of the jaw enlarge, and they can always be felt on one or both sides, according to the lesions within. Sometimes the typical membrane is preceded by a grey mucous secretion. Gangrene occurs occasionally in the severe cases.

The temperature of diphtheria is very variable, and runs no definite course; it may rise to 103°, 104°, or 105°, but is often throughout the whole illness much lower. The pulse is feeble, and the blood pressure falls, and the patient soon becomes pallid and weak. The appetite is lost, and feeding becomes difficult and painful from the condition of the throat. In a large proportion of cases, variously estimated at 25 to 60 per cent., the urine is albuminous, and this occurs, not after the illness, as in scarlatina, but during the height of the throat symptoms. In some cases the specific inflammation spreads to adjacent mucous membranes—those of the nose and the conjunctiva, the Eustachiad tube (causing otitis media), and the larynx and respiratory passages. Severe cases may be hæmorrhagic in type with epistaxis, bleeding from the throat and subcutaneous ecchymoses. In uncomplicated cases death takes place by asthenia, fall of blood pressure and circulatory failure, sometimes with extraordinary suddenness. Occasionally dilatation of the heart can be recognised by physical signs, and the pulse becomes quick, feeble, and irregular or slow from heart block. It may thus happen on the second, third, or fourth day, or later.

*Nasal diphtheria* may occur alone, or as the result of a direct spread from the fauces. There is more or less obstruction to nasal respiration, the mucous membrane is swollen, and a muco-purulent or thin pale brown mucoid secretion runs from the nostrils, reddening or excoriating the alæ and adjacent upper lip. It may be streaked with blood, or decided epistaxis may occur.

*Laryngeal diphtheria* presents the symptoms of laryngitis, and the obstruction, due to the swollen mucous membrane, is increased by the presence of the diphtherial false membrane. It is rare in adults.

The *first stage* is indicated by the presence of a loud, brassy cough and a rough, hoarse voice, and it lasts for at most two days.

In the *second stage* there is respiratory distress with cough and aphonia. Stridor develops owing to narrowing of the glottis, being most marked during inspiration. As obstruction increases the supra-clavicular, supra-sternal, and intercostal spaces are sucked in with each inspiration; and in infants and young children with soft yielding bones the lower end of the sternum, or the three or four lower ribs, are drawn in, showing the extent to which the air is hindered from access to the lungs through the glottis. Slight degrees of obstruction may persist some days without much change, but more often the case gets progressively or rapidly worse. The face, at first flushed, with bright eyes, gets cyanosed. The child is restless, putting its hand to its mouth or throat, as if to remove the impediment. The cough becomes husky, and from time to time there may be spasmodic closure of the glottis, in which violent inspiratory efforts are made, and the cyanosis becomes extreme.

In the *third asphyxial stage*, which only lasts a few hours, the pulse becomes weaker, and the respiratory effort is lessened. The skin is livid, the extremities are cold, the mental faculties are blunted, and stupor supervenes. Sometimes there are convulsions before the end.

As a rule, in laryngeal diphtheria the process is not confined to the larynx; it spreads to the trachea and the bronchi, forming a continuous membrane in the former, which, in the middle-sized and smaller bronchi, is gradually changed into a purulent secretion. These morbid products naturally increase the difficulty of breathing, though it is not always easy to recognise their presence by physical signs; in fact, death not uncommonly occurs from blocking by a plug of desiccated mucus or loose membrane at or below the bifurcation of the bronchi after a successful tracheotomy (Biernacki). Generally a loud and stridulous noise is heard in the chest, caused by the obstruction at the glottis. It may be mixed here and there with mucous râles, and there may be patches of tubular breathing, due to the broncho-pneumonia which is so frequent a result of the spread of diphtheria into the lungs.

Laryngeal diphtheria is often primary, though there is usually some catarrh of the throat, but it may be associated with faucial and nasal diphtheria.

**Complications and Sequelæ.**—Complications are chiefly the extension of the disease to different parts, which have been described.[1] *Pleurisy* may accompany the *pneumonia* (*0·29*), or *broncho-pneumonia* (*1·0*). *Albuminuria* (*23·30*) is rarely more than a symptom, but occasionally a definite nephritis (*0·69*) may persist or occur as a sequela. The lymphatic glands may inflame and suppurate or slough with about one-third the frequency of occurrence in scarlet fever; thus *suppuration* in the acute stage (*0·22*), in convalescence *simple adenitis* (*2·06*), and *suppurative adenitis* (*0·63*).

The most important sequel of diphtheria is the affection of the peripheral nerves, which results in *diphtherial paralysis* (*8·76*). This shows itself first in the soft palate. Some days, or a week, or several weeks, after apparent recovery, the child is noticed to speak with a nasal, twanging voice, and when it swallows liquids a small quantity is regurgitated through the nose. These defects are due

[1] The bracketed percentages in italics are from 6,184 cases in M.A.B. hospitals in 1914.

to paralysis of the soft palate, which fails to shut off the mouth from the nose, as it should during speaking and swallowing. Shortly after this the child is noticed to be weak in the legs, and unable to walk any distance, or the knees give way on standing for a short time. The knee jerk is lost quite early. In older children and in adults failure of accommodation of the eye for near objects is often noticed, due to paralysis of the ciliary muscle; and the extrinsic muscles of the eye may be also affected, producing strabismus or squint. In many cases the paralysis does not proceed beyond this stage, and in a few weeks the muscles recover their power completely. In others the muscular system throughout the body may be affected. The patient lies motionless in bed, respiration is rendered difficult from paralysis of the intercostal muscles or diaphragm, and food given by the mouth is rejected, from inability to swallow it. The paralysis of the diaphragm is often followed by collapse of the lower lobes of the lungs (*see* Lesions of the Phrenic Nerve). The laryngeal muscles are also sometimes affected—one, or many, or all of them. Thus there may be paralysis of one cord, or paralysis of the abductors, or paralysis of all the muscles, with cadaveric position of the cords. The voice in the last case will be lost completely, and variously modified in other cases (*see* Paralysis of the Larynx).

Sensory symptoms may occur, but in children they are frequently not detected. They consist in a feeling of numbness, or formication, or distinct anæsthesia, especially in the extremities. Ataxy has been observed with very little actual paralysis, and rarely transient muscular spasms. Sometimes the muscles or the nerve trunks are tender on pressure. In severe cases electrical reactions are diminished, and some muscular atrophy ensues. Recovery generally takes place within three or four months, and the paralysis rarely, if ever, becomes chronic. Death, however, results sometimes from paralysis of the diaphragm, with gradually increasing accumulation of secretion in the bronchial tubes, and sometimes from cardiac paralysis, shown by a feeble, irregular or intermittent, generally quick, but sometimes slow, pulse, with vomiting and cyanosis.

**Diagnosis.**—*Faucial Diphtheria.*—The cardinal features are—insidious onset; characteristic membrane already described; relatively slight pyrexia; lowered blood pressure and weak pulse; albuminuria; onset of paralysis. The diagnosis can only be positively established by the bacteriological cultivation of the Klebs-Loeffler bacillus from the secretions of the affected part. This is generally done by means of a swab of cotton wool on the end of a piece of wire: the swab is smeared over the fauces or tonsil, inserted in a sterilised glass or metal tube, and sent to the bacteriological laboratory for cultivation It is important to remember that bacilli are sometimes cultivated from the throat, both together with the Klebs-Loeffler bacillus and apart from it, which resemble it closely, but, unlike the *B. diphtheriæ*, are not virulent to guinea-pigs. The most important of these is *Hofmann's bacillus*, which is often found. It is about $2\mu$ in length, and generally arranged in pairs; and it produces alkali, not acid, in milk and glucose media.

In *follicular tonsillitis* small yellow plugs are often seen; they can be easily removed; but there may be white plugs of secretion exactly like the diphtherial deposit. There are often several plugs in the same area, as contrasted with one patch in diphtheria. The temperature is usually high.

*Vincent's angina*, which is described under Diseases of the Throat, resembles diphtheria in its membrane.

In *scarlatina* the tonsils are swollen and are covered with viscid, mucoid, and often yellow secretion; and the occurrence of a definite white patch would suggest a complicating diphtheria. In the earliest stages of throat inflammation it may be impossible to say, unless by cultivations, until the rash of the one disease or the membrane of the other is seen. Influenzal sore throat, herpes, and secondary syphilis must also be distinguished.

*Laryngeal Diphtheria.*—The diagnosis will be easier if there is simultaneous

faucial or nasal diphtheria. It must be distinguished from catarrhal laryngitis, which may be simple, or may herald the onset of measles. Clinically it is often impossible to distinguish the two, so that a swab must be taken at the earliest opportunity. It is safest to assume the case is one of diphtheria until the contrary has been proved. Laryngeal diphtheria must also be distinguished from (*a*) obstruction below the larynx, such as broncho-pneumonia where there may be recession of the ribs, but not aphonia; and pressure from enlarged glands; (*b*) obstruction above the larynx, such as retro-pharyngeal abscess; (*c*) œdema of the glottis, due to sepsis, nephritis, urticaria, and various other conditions.

**Prognosis.**—The mortality from diphtheria has been considerably reduced since the introduction of the treatment by antitoxic serum in 1893. In the hospitals of the Metropolitan Asylums Board during 1891, 1892, 1893, the annual mortality was 30 per cent.; in 1913 to 1915 it averaged 7·17 per cent. The chance of recovery is diminished by every day, or half-day, that the treatment is delayed. Extensive formation of membrane, spread of the disease to the nose, rapid failure of strength, feeble pulse and hæmorrhages, are of unfavourable prognosis. Laryngeal diphtheria is more fatal, because, though laryngeal obstruction may be obviated by tracheotomy, death may occur from purulent bronchitis or broncho-pneumonia, caused by extension to the lungs. In these cases also the mortality has been much diminished by antitoxin. Diphtherial neuritis generally recovers, but is occasionally fatal through paralysis of the diaphragm.

**Prevention.**—It must be remembered that the bacilli may remain in the throat long after the patient is himself quite well, and hence the risk of contagion remains. It is usual to keep a diphtheria patient from contact with others until bacilli can be no longer cultivated from the throat or nasal secretions. Sometimes a period of several weeks elapses before the patient is free; but 50 per cent. lose them at the same time as the membrane, not more than 7 per cent. retain them for one month, and not more than 1 or 2 per cent. for three months (Ledingham and Arkwright). These carriers may be treated as described under Cerebro-spinal Fever. Favourable results have also been obtained with vaccines.

Other measures of prevention are—taking care of the milk supply; removal of susceptible children; periodical swabbing of throats in institutions; prophylactic injection of serum.

Undoubtedly the most important method of prevention is the Schick test (**11**). This test is used for determining whether individuals are susceptible to diphtheria infection or not. A small dose of diphtheria toxin—one-fiftieth of the minimum lethal dose for a 250-gramme guinea-pig in 0·2 c.c. of normal saline—is injected into the arm intradermally, not under the skin. The needle, which is of fine calibre, is passed nearly parallel to the skin within its substance. The injected fluid should appear and feel like a small button in the skin. A control injection of the toxin heated to 75° C. for ten minutes is made into the opposite arm. A positive reaction, which means that the individual is susceptible, is shown by a circumscribed area of redness accompanied by slight infiltration of the skin, measuring from 1 to 2 cm. in diameter. This appears in about twenty-four hours, and reaches a maximum in four or five days. Pseudo-reactions, which are fairly common, appear earlier and the area of redness is not so well defined.

By this test infants are found to be immune, and the percentage of persons susceptible to diphtheria is greatest between the ages of two and five years. This coincides with the age-period when the incidence of clinical diphtheria is greatest.

Active immunisation with toxin-antitoxin has been carried out with success in susceptible people, and this may be of value in the case of doctors and nurses or of children when diphtheria is prevalent in the locality.

**Treatment.**—Immediately upon the diagnosis of diphtheria being known, and even before, if there is a high probability of the suspicion being confirmed by the bacteriological test, *diphtheria antitoxic serum* should be injected.

The methods of preparing the serum vary somewhat in detail, but the principle

is the same. An animal, for instance the horse, is gradually rendered immune by successive injections of increasing quantities of the culture fluid of the diphtheria bacillus, deprived of the organism itself. When the animal is at length completely insusceptible to the diphtheria poison, its blood serum is found to have the power of neutralising the influence of diphtheria cultures inoculated into animals; and hence it appears that this serum contains a substance (*antitoxin*) which antagonises the toxin of the diphtheria bacillus. The serum is standardised by experiment upon animals. The unit adopted by Ehrlich is the amount which, when mixed with a hundred times the fatal dose of toxin, protects a guinea-pig of 250 grammes weight from death within four days. The initial dose required is from 4,000 to 12,000 units, according to the severity of the disease; and 4,000 units may be contained within 20 c.c. of the serum. The dose may be repeated at intervals of twelve or twenty-four hours during the next two days, and the amount must be estimated by the intensity of the disease, and not by the age of the patient. The injection should be made under the skin of the flank, or into the vastus externus (J. D. Rolleston and Macleod), with antiseptic precautions. An effect is very often observed in a few hours either in the fall of the temperature or at least in the arrest of the progress of the symptoms. An urticarial or morbilliform rash with pains in the joints sometimes follows the injections; this is due not to the antitoxin, but to the horse serum containing it. It usually appears from seven to twelve days after the injection.

Treatment in general must be supporting and stimulating. The patient should be confined to bed, and liquid food should be given in small quantities frequently. The fever is not often so high as to require special attention; but if the heart dilates, and the pulse becomes feeble, tincture of digitalis may be given. Wine and brandy will be early required, and in severe cases they must be given freely. Recent experiments on animals suggest that blood transfusion may be of value in the circulatory failure associated with severe toxæmia (Harding).

Local remedies are applied partly as palliatives, partly as antiseptics. The patient may be given ice to suck if the throat is painful. One of the most useful ways of applying remedies is by syringing. The patient lies on his side. The syringe is introduced nearly as far as the tonsil that is resting uppermost, the fluid being collected in a dish. Boric acid (half-saturated solution), or eusol may be employed. Where syringing is difficult swabbing may also be carried out, lotions of permanganate of potassium (2 grains to ℥j), formalin (1 in 200), chinosol (1 in 600), the tincture of ferric chloride (℥ss to ℥j), carbolic acid (2 grains to ℥j), boroglyceride, being applied every four hours with a brush. In somewhat stronger solution they may be used as a spray. A useful solution for the spray consists of carbolic acid, 120 grains; iodine liniment, 2 drachms; rectified spirit, 1 drachm; water to 12 ounces.

For the removal of the offensive and irritating secretions when the nasal mucous membrane is involved the nostrils should be syringed with dilute disinfectant solutions, such as potassium permanganate and carbolic acid, or these may be administered by the nasal douche.

In laryngeal diphtheria the patient should be subjected to an atmosphere saturated with moisture. In a small room it will be sufficient to use a bronchitis kettle, the steam from which may fill the room. Much relief is also sometimes given by a hot bath. If improvement is not apparent in a few hours, intubation or tracheotomy should be performed, and this must be done at once if there is sucking in of the chest, if the patient is drowsy or becoming cyanosed, or if the forehead is cold and clammy. The probability of success is greater the earlier a tube is introduced into the larynx or trachea; and if it is suspected that the obstruction will increase, the operation should be done while the child is strong and of good colour. Generally in diphtheria tracheotomy is to be preferred to intubation. The latter is bloodless, and if it fails can be succeeded by tracheotomy; but it requires special skill in its performance, and the risk is run of push-

ing membrane down into the trachea. Nearly always some improvement follows an operation; the child breathes freely and deeply and sleeps tranquilly; but the danger of broncho-pneumonia still remains, and, as already mentioned, obstruction may take place from a plug of desiccated mucus lower down. To avoid this steam should be used, and oxygen, if given, should be passed through water. If obstruction occurs, spraying through the tracheotomy tube with sodium bicarbonate (10 grains to 1 ounce water) during inspiration may loosen the plug so that it is coughed up. In the last resort forceps should be passed down and the attempt made to get hold of the plug (Biernacki). Diphtheria antitoxin should be given in any case, whether the laryngeal lesion is primary or secondary. Internally expectorants, such as ammonia or ipecacuanha in small doses, may be tried. The tracheotomy tube may often be removed in from one to four days.

If there are any signs of diphtheritic paralysis, the patient must be kept lying down. In the more severe cases, where swallowing becomes difficult, feeding by the nasal tube will be necessary. There will also be special danger in these cases of failure of the circulation. If the paralytic signs persist into convalescence, the patient should only be allowed to get up gradually, when all danger from the heart has disappeared. Strychnine in small doses is often prescribed for paralysis. Later on massage and electricity may be used for the muscles. They practically always recover their function completely. In diaphragmatic paralysis oxygen gas may be inhaled.

## PNEUMOCOCCAL INFECTIONS

The micro-organism first found by Sternberg in the saliva, and subsequently recognised by Fraenkel as the causative agent of acute lobar pneumonia, was called by him the *Diplococcus pneumoniæ;* and it is otherwise known as the *Diplococcus lanceolatus*, and more generally as the *Pneumococcus.* It consists of oval gram-positive cocci, of about 1$\mu$ in diameter, often united in pairs or in short chains of five or six. Each is surrounded by a distinct halo or capsule of colourless homogeneous material.

This organism is responsible for acute and severe inflammations in many organs and parts of the body, and is chiefly known in connection with acute lobar or croupous pneumonia, of which it is the cause in from 80 to 90 per cent. of the cases. However, it also attacks other organs. Thus there occur pneumococcal pleurisy, empyema, peritonitis, meningitis, arthritis, enteritis, endocarditis, pericarditis, nephritis, endometritis, and subcutaneous, intramuscular, and intravisceral abscesses. It is rather characteristic of pneumococcal infections that pus formation takes place relatively late, after a copious exudation of serous fluid. The pus, too, when formed often sinks to the bottom, leaving a fairly clear layer of serous fluid on top.

Four types of pneumococci have been isolated, which resemble one another morphologically and culturally, but have different biological characteristics. Types I. and II. account for two-thirds of pneumonia cases in this country and America, and anti-sera have been prepared which have been used successfully in treatment. Type III. occurs in the most severe cases, and the corresponding anti-serum is useless. Type IV. resembles the pneumococci found normally in the mouth. It is of low virulence, and the resulting pneumonia is very mild.

There is no doubt that in many cases in which a severe local lesion, such as inflammation of the lung, is the principal feature of the case the organisms are to be found in the blood, and there is indeed a *pneumococcal septicæmia;* and rarely a few cases have been reported in which this general septicæmia has been fatal without any local inflammation. But in the vast majority of instances the local disease is prominent, and for many reasons it seems to be preferable to describe the lesions which it causes in separate organs in the section devoted to the disease of such organs.

Thus the reader will find in the appropriate places accounts of pneumonia, broncho-pneumonia, pneumococcal bronchitis, pneumococcal peritonitis, pneumococcal meningitis, pneumococcal arthritis, and nephritis.

## GONOCOCCAL INFECTIONS

The acute inflammation of the generative organs, male and female, known as *gonorrhœa*, is an infective disease, of which the micro-organism is the *Micrococcus gonorrhœæ* or *gonococcus*. The gonococci are found in the pus discharged from the urethra, and are largely contained within the leucocytes. They are decolorised by Gram's method.

The course of the disease, which is very variable, may be classified under five heads :

(1) *The Primary Focus.*—Infection is, as a rule, by direct contact, and involves usually the urethra in the male, and the urethra, cervical canal, and, to a less extent, the vagina in the female. Gonorrhœa of the female genital organs is a fertile source of gonococcal conjunctivitis and other ocular lesions in the new-born infant.

(2) *Area of Local Spread.*—The infection may spread along the lymphatics to the passages and organs in the neighbourhood of the primary focus, and may cause inguinal bubo, vesiculitis, epididymitis, orchitis, cystitis, prostatis, pyelitis, endometritis, salpingitis and pelvic cellulitis and peritonitis.

(3) *Gonococcal Septicæmia, i.e.* a general blood infection without focal lesions. This is a rare condition, and in this sense the gonococcus resembles the staphylococcus, which rarely produces a pure septicæmia.

(4) *Metastatic Gonococcal Infection*, or focal sepsis. The spread of the infection from the primary focus to other parts of the body takes place by the blood stream. In severe cases there is also a general blood infection (septicæmia), as may be found in the more severe cases of gonococcal arthritis, or gonococcal endocarditis, which is one form of malignant endocarditis. However, in most cases the metastatic infection is not accompanied by a septicæmia.

The commonest infection of this type is the so-called *gonorrhœal rheumatism*, which includes arthritis, fibrositis, teno-synovitis, bursitis, myositis and perineuritis. Periostitis and osteo-myelitis are rare. The eye is not uncommonly affected by metastatic infection, the principal lesions being iritis and conjunctivitis. It is probable that some cases of " simple " mitral disease are secondary to gonorrhœa. There is a gonococcal pleurisy.

**Gonorrhœal rheumatism,** from its resemblance to acute rheumatism, requires separate notice. It begins at an interval of fourteen days or three or four weeks from the commencement of the urethral discharge, sometimes while the discharge is still purulent, more often during the subsequent stage of gleet.

**Morbid Anatomy.**—There are serous effusion into the joint, infiltration and œdema of the tissues around it, and in severe cases suppuration, erosion of the cartilages, disorganisation of the joint, and ankylosis. The synovial membrane is primarily affected in the acute cases, and the surrounding fibrous tissues are chiefly and first involved in the subacute forms.

**Pathology.**—The gonococcus has frequently been found in the fluid of the inflamed joints and of the sheaths of the tendons when they are affected. If suppuration takes place, pyogenic organisms may also be present. In more chronic cases the gonococcus is not found. It may be tucked away in the recesses of the joint. Another suggestion is that the inflammation is due to the gonococcus toxin.

**Symptoms.**—In *acute* forms of gonorrhœal rheumatism several joints are at first affected with pain and swelling, but the disease often, after a time, localises itself in one only, which may be the elbow, knee, ankle, wrist, or foot. There is very extensive redness, with swelling, pain and tenderness. The redness often spreads up the limb far beyond the joint, and the tissues are infiltrated to a corresponding

extent. This infiltration may be actually mistaken for abscess, and it may also have a closer resemblance to gout than to rheumatic fever. The pain is very severe on the slightest movement; the fever is not high. The inflammation only slowly subsides, and leaves a good deal of stiffness behind; but the joint does not often suppurate. Cardiac complications are only occasionally observed. The late N. C. Davies-Colley stated that this form of gonococcal arthritis was as common in women as in men.

In other less acute or *subacute* cases the resemblance to a mild rheumatic fever is in some respects closer; the joints are swollen, not so red, and less generally infiltrated. All the joints in the body may be affected; but the knees, ankles, and wrists are most often involved. Not infrequently there is much pain in the fasciæ, especially in the plantar fascia, and the sheaths of tendons may be involved. Spondylitis occurs in a certain proportion of the cases. As in the acuter forms, the inflammation tends to be persistent and does not readily subside and come again, as it does in ordinary rheumatic fever. It lasts two, three, or more weeks, and leaves a great deal of stiffness or even fibrous ankylosis.

**Diagnosis.**—The disease is most likely to be mistaken for *rheumatic fever* until it is discovered that the patient has a discharge, or until the persistence of the arthritis in a few joints makes one suspect the nature of the case. The resemblance may be increased by a history of previous attacks, for, though gonococcal synovitis does not itself recur after long intervals like rheumatic fever, other attacks are often induced by fresh infection. The acuter forms of the disease may closely resemble *erysipelas*, *abscess*, or *acute gout*. The age of the patient and the position of the inflammation will generally exclude the latter. *Pyæmia* may be a cause of multiple synovitis after gonorrhœa; but in this case the illness is generally more severe, with rigors and such serious complications as pericarditis, endocarditis, pneumonia, or pleurisy.

An investigation of the common valvo-vaginitis of children up to 15 years, has shown that 36 per cent. are gonorrhœal, and 64 per cent. are non-specific and due to staphylococcus, see etc. (**29**).

**Treatment.**—It is, no doubt, desirable to cure the urethral discharge as soon as possible. For the arthritis alkalies and iodide of potassium have been largely employed, the latter in full doses; but it is probably better to give plenty of good food, with cod-liver oil and iron or cinchona. Vaccine treatment is also employed. Autogenous vaccines are obtained by cultivating the gonococcus from the patient's discharges, and may be injected repeatedly. But the cure often takes five or six weeks. Stock vaccines are prepared from a number of different strains, and not from the patient's own organisms. Recently sensitised and detoxicated vaccines have been used. The former is a mixture of a vaccine with antigonococcal serum. The latter is a vaccine with the virus much attenuated, either by treating the organisms with *caustic soda* or by prolonged cultivation on *artificial media* outside the body. Locally the joints may be painted with iodine. In acute cases the limb should be kept completely at rest by means of a plaster of Paris splint; and anodyne applications, especially the compound mercury ointment, with extract of belladonna, may be used. As soon as the inflammation has subsided the tendency to fixation must be met by friction, massage, and passive movements.

## TYPHUS FEVER

*(Jail Fever, Typhus Exanthematicus)*

Typhus fever is an acute specific contagious disease, lasting two or three weeks, and producing a typical eruption on the skin.

**Ætiology.**—Typhus occurs, for the most part, in epidemics, which may last for some months, and then gradually subside. These epidemics commonly break out in large towns, in prisons, and in armies in the field. It is now comparatively

rare in England, but it raged extensively in Serbia in the early part of the recent European war, and in German war prisons (Wittenberg). The disease called Brill's disease in America is at least a variety of typhus, if not identical with it.

It attacks persons of all ages, and males and females equally. Those who have already gone through an attack are, with rare exceptions, protected from another. It is not much affected by season or weather, except in so far as they may determine overcrowding; but it is confined to temperate and cold climates and is particularly liable to occur in famine areas.

Nicolle and Conseil, of the Institut Pasteur in Tunis, showed that the infection was conveyed by body lice. They successfully inoculated monkeys with blood from typhus patients, and then, by means of lice feeding upon the first monkeys, produced the disease in others. Wilder from the study of typhus in Mexico confirms this view, and believes that the virus proliferates inside the insect. There is now little doubt from experience in different theatres of war from 1914 to 1918 that the incidence of the disease depends entirely on conditions which favour the presence of infected body lice and their transmission from one person to another. Thus in hot climates the disease dies out entirely during the hot summer months, when the temperature becomes sufficiently high to destroy the lice. It would appear very doubtful whether factors such as overcrowding or poverty predispose individuals to infection, except in so far as these conditions are associated with the presence of lice. The cause of the disease is now thought to be an organism known as the *Rickettsia prowazeki*. It has been found in the bodies of infected lice and in the lesions of man; it has been grown in tissue plasma cultures. It appears to be closely associated in the circulating blood with the blood-platelets. Injections of platelets from a human case has produced the disease in guinea-pigs. Further, an injection of infective platelets into human lice per diem has caused a profuse growth of the Rickettsia in these insects. It is probable that human infection takes place by the rubbing in of the excreta of lice through abrasions in the skin, rather than directly by means of insect bites.

**Morbid Anatomy.**—The post-mortem appearances are scarcely distinctive, but are such as are characteristic of severe fever. The *rigor mortis* is imperfect, decomposition sets in early, and there is much post-mortem discoloration; the blood is more than usually liquid, coagulating rapidly but imperfectly. The voluntary muscles are soft and friable, and may undergo Zenker's degeneration (*see* p. 22); the muscular tissue of the heart is also soft, and affected with fatty or granular degeneration. The bases of the lungs are in a state of hypostatic congestion; they are dark red or purple, congested, airless, and friable, yielding blood and serum on section, and without the granular surface of pneumonic hepatisation. This last condition is, however, also present in some cases. The spleen and liver are soft and somewhat enlarged, and may present ecchymoses on the surface. The kidneys are often soft and large, though sometimes quite normal. The enlargement of the liver and kidneys is associated with cloudy swelling and parenchymatous degeneration. The characteristic lesions are the spots on the skin, and nodules in the brain and viscera. These are formed by the endothelial cells of the capillaries, which act as phagocytes and then become necrosed and surrounded by lymphocytes and plasma cells.

**Symptoms and Course.**—The period of *incubation* is variable; in most cases it is from five to twelve or fourteen days. The disease begins, like many other fevers, with headache, loss of appetite, and a general feeling of illness, with perhaps some chilliness or actual rigor. The headache at the outset is frequently extremely severe, more so than with the other fevers with which the disease is liable to be confused. Very marked weakness is also a prominent symptom, and the knee jerks are almost invariably lost early. In severe cases there are sharp rigors, with nausea or sickness. During the next two or three days, while yet there is nothing distinctive of the disease, all the symptoms of severe fever are rapidly developed. The temperature rises to 103° or 104°; the

pulse and breathing are proportionately quickened; there are furred tongue, continued headache, flushed face, and suffused eyes, pains in the back and limbs, anorexia, scanty high-coloured urine, and constipation. By the third or fourth day the patient is generally so ill as to be obliged to take to his bed. On the fourth or fifth day, sometimes as early as the third, sometimes on the sixth or even the seventh, appears the characteristic eruption, or *mulberry* rash, of typhus. It comes out on the abdomen and chest and on the backs of the hands and wrists, and in the course of two or three days covers the trunk, and perhaps also the arms and legs. The face and neck are mostly free. It consists of two portions: one, a dusky red mottling, fading on pressure, not giving rise to any elevation of the surface, and often described as "subcuticular"; the other, a rash, consisting of numerous paler or darker pink or red prominences or papules of any size up to 3 mm. in diameter, scattered irregularly over the surface, at intervals of $\frac{1}{4}$ to $\frac{1}{2}$ inch from each other. These at first fade on pressure, but after a day or two they become more dusky, and later some of them become petechial from extravasation of blood, which persists under the pressure of the finger. The rash gradually fades during the second week, by the end of which it

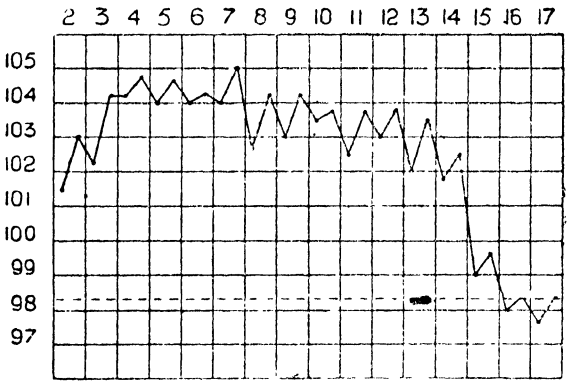



FIG. 8.—Temperature in a Case of Typhus Fever.

is generally gone. In rare cases the mulberry rash is preceded by a diffused red rash, or *roseola*, not unlike the eruption of scarlatina; but this disappears entirely before the mulberry rash comes out.

By the time the eruption is developed—that is, at the end of the first week—the fever has made progress. The patient lies on his back in bed, with a dull, heavy, stupid look, the face flushed, the conjunctivæ injected, and the pupils contracted. The lips and tongue are tremulous early in the disease. The temperature, pulse, and respiration are, of course, above the normal; the tongue continues furred. He may still complain of headaches, but is mostly apathetic and listless, and only towards night has a little rambling delirium. In the second week the symptoms are aggravated. The headache, indeed, is no more complained of, but the delirium becomes constant day and night; and, though generally low and muttering, it is sometimes noisy and raving, so that the patient may become violent, may start from his bed, and even jump out of the window. But more often his muscular weakness is extreme; he sinks down in the bed, and is unable to raise or turn himself; the limbs are tremulous, and subsultus tendinum is observed. Later on the patient becomes completely comatose, the fæces are passed unconsciously, and the bladder becomes distended from retention of the urine. The pulse is rapid, reaching 110, 120, or more, and with the progress of the illness it becomes feebler and

smaller. It is dicrotic, and in severe cases may be irregular and intermittent. The sounds of the heart are faint, and in the worst cases the first sound is almost inaudible and the pulse imperceptible. The breathing is rapid, reaching thirty or forty per minute, and there may be some bronchitic rhonchi over the chest. In the later stages there is generally evidence of congestion of the bases of the lungs —viz. dulness for three or four fingers' breadths from the base of the chest, deficient breath sounds, and râles over the same area, while the breath sounds under the clavicles are supplementary. The tongue, which is furred at the commencement of the illness, soon becomes dry, brown, and cracked; and the tongue, teeth, and lips become covered with a brown or black crust of sordes. The bowels are often constipated, but there may be diarrhœa; and in either case the motions are generally dark. The urine continues dark and scanty, the chlorides are reduced to a very small amount, and in many cases a trace of albumin appears, especially towards the end of the illness. Corresponding to these findings there is an increase of the urea in the blood in more than half the cases. The nervous symptoms have recently been ascribed to uræmia. An examination of the blood shows that the red corpuscles are somewhat diminished in number; there is a very slight leucocytosis, with a relative increase of lymphocytes and a diminution of the polymorphonuclear corpuscles (*see* Diseases of the Blood). The nervous symptoms are accompanied by changes in the cerebrospinal fluid indicative of meningitis. It may be clear or turbid, or in severe cases yellow and even coagulated. There is an intense cellular reaction.

After the initial rise of temperature there is often a slight remission on the third or fourth day, followed by a considerable rise with the appearance of the rash. It then continues elevated and reaches a maximum of 104·5°, 105°, or even 106°, about the end of the first week. It remains for some days about the same level, with perhaps slight morning remissions, but is commonly a little lower in the second week, towards the end of which it shows more decided morning remissions, and finally, in cases that recover, undergoes the rapid fall which constitutes the *crisis* of the disease. This happens in the majority of cases about the end of the second week, or from the thirteenth to the sixteenth day (Fig. 8), the temperature falling four or five degrees in the course of twenty-four to thirty-six hours, from 103°, it may be, or higher, to 99°, 98°, or 97°. In some cases the fall is more gradual (*lysis*). At the same time the pulse and respiration become slower, and the crisis is not uncommonly accompanied by other indications of change in the condition of the patient—viz. profuse sweating an or attack of diarrhœa. From this time the improvement of the patient is rapid: he soon regains consciousness, the tongue cleans, the dusky hue of the face subsides, and thirst is replaced by appetite. Convalescence is usually protracted. Tachycardia even on slight exertion is often a prominent feature. The patient frequently loses a great deal of hair and may become entirely bald. As a rule at least three months elapse before convalescence is complete.

But the termination is often unfavourable; in fatal cases death takes place commonly towards the end of the second week from cardiac failure, or from congestion of the lungs or pneumonia, or from increasing coma. It is occasionally preceded by a rapid rise of temperature.

Among the **Complications and Sequelæ** are pneumonia and bronchopneumonia, which often arise during the second week, and persist into the period of convalescence, delaying recovery, and sometimes going on to gangrene; bed sores; tender toes; gangrene of the fingers, toes, nose, or pudenda, probably from embolism; suppuration of the joints; otitis media; inflammation and suppuration of the parotid, submaxillary and sublingual glands; and erysipelas of the face, cellulitis and subcutaneous abscesses. Thrombosis of the femoral vein, with resulting œdema of the leg, may occur, but is less frequent than in enteric fever. Meningitis has been found in a few instances *post mortem*, and uræmic convulsions sometimes occur in association with renal disease and albu-

minuria, which are either of old date or have been set up by the typhus fever itself. During convalescence there may be syncopal attacks with cardiac bruits suggesting that the myocardium has been affected; peripheral neuritis, indicated by pain and tenderness of the limbs; and mental disturbances.

**Diagnosis.**—At the outset of the disease it may be impossible to distinguish it from other febrile illnesses, unless it is known that the patient has been exposed to contagion. When the eruption appears it may be mistaken for *measles*, but the rash of measles generally comes out first on the face, near the scalp; the spots are brighter red, more raised, more irregular in shape, and perhaps arranged in crescentic forms, and their appearance is preceded by catarrh. The eruption should be sufficient to distinguish it from *acute meningitis* and from *acute pneumonia*, with which typhus may be mistaken, on account of pronounced cerebral symptoms in some cases, or of respiratory distress and lividity in others. Pneumonia, however, should be recognised by its physical signs, but may, of course, be secondary to typhus. In its early stages, typhus may be mistaken for *enteric fever*. The papules of typhus may resemble those of typhoid, but their early appearance, and their occurrence on the forearms and wrists, should prevent errors, as well as the general mottling, if pronounced, and the petechiæ in later stages. Other points of difference are the more sudden onset of illness in typhus; the absence of diarrhœa in typhus as contrasted with the loose, pea-soup, offensive stools which so often occur in enteric fever; and the early stupor and delirium of typhus. Typhus also spreads rapidly, so that many members of a family may be affected at the same time. Exceptionally a case of *malignant endocarditis* with petechial eruption may closely resemble typhus. If the Widal test be employed (*see* p. 16), the serum will fail to agglutinate typhoid bacilli, or paratyphoid bacilli, or the micrococci of Mediterranean fever. During recent years it has been found that the serum of patients suffering from typhus agglutinates in high dilutions a form of *Bacillus proteus*. This agglutinating power is obtained as early as the sixth or seventh day of the disease, and is usually at its maximum by about the fourteenth day. Undoubted cases of typhus have, however, been recorded which have failed to give a definite agglutination reaction. As a rule, however, the clinical diagnosis is clear by the seventh day; and therefore this agglutination test, known as the Weil-Felix test, is not as important clinically as it would otherwise be. In a fairly high proportion of cases the Wassermann reaction is positive.

**Prognosis.**—The mortality of typhus varies from 6 to 30 or 40 per cent.: in 2,000 cases occurring in Serbia it was 15 per cent. (Davy and Brown). It increases with the age of the patient; thus in children under ten years of age it is about 5 per cent., and in people over sixty years of age it is as much as 66 per cent. The chances of recovery are lessened by previous intemperance and by deficiency of bodily vigour from any cause, whether it be overwork, starvation, or overcrowding; even the attempt to keep about during the first days of the attack renders the case less promising. The symptoms of the illness itself, which suggest an unfavourable prognosis, are abundant rash, very high fever, very rapid pulse, early development of cerebral symptoms, great weakness of the circulation, severe pulmonary complications, and convulsions. The determination of the urea in the blood is a valuable aid to prognosis. When the value is below 0·05 per cent. recovery is the rule. Above 0·09 per cent. the outlook is unfavourable, while above 0·2 per cent. it is hopeless.

**Prevention.**—As the disease can only be acquired through the bites of infected lice, epidemics can be avoided by routine inspections for lice and their destruction in clothes, blankets, etc., by heat or soaking in disinfectants, such as cresol. To avoid infection of nurses or medical attendants, the latter should wear tightly fitting overalls with gum boots. On admission of patients suspected of the disease the head and hairy parts should be shaved and the body thoroughly disinfected, and infected clothing dealt with. Once the lice are destroyed,

there is no danger of infection. Prophylactic inoculations have been carried out as follows: Defibrinated blood from a patient in the acute stage of the disease is inactivated by heating to 58° C. for half an hour and injected in doses of 1, 2 and 5 c.c.

**Treatment.**—Recent attempts have been made to cut short the disease by injections of 0·008 gm. of mercury cyanide and salicylate daily. Others have used turpentine inhalations by placing in the nose and frequently renewing small plugs of cotton wool moistened with oil of turpentine. Injection of 20–50 c.c. serum taken from typhus patients, whose temperatures have been normal for 4–11 days, have mitigated the disease. The object of general treatment is to maintain the strength of the patient, so as to bring him safely through his illness, by following the principles laid down under the head of Pyrexia (*see* p. 24). Rest in bed, adequate nursing, cleanliness, prevention of bedsores, attention to the mouth, and in later stages to the bladder, suitable food frequently given, are the main considerations. In severe cases sedative treatment is to be preferred to stimulants, especially if there is weakness of the heart. Potassium bromide in 5 grain doses may be given in the early stages. Headache and sleeplessness may be relieved by applying ice to the head, by cold bath or sponging, or by the subcutaneous injection of a quarter of a grain of morphia. Chloral in a dose of 10 or 15 grains is more suitable when the delirium is maniacal, and it may be combined with the same quantity of potassium bromide. For sleeplessness paraldehyde (℥ss to ℥j in water) or sulphonal (20 grains) may also be used.

## THE ENTERIC FEVERS

In the early part of the nineteenth century, the fevers, excluding the exanthemata, which were characterised by a definite cutaneous eruption, were divided into continuous and intermittent. The intermittent were those now known as malarial. When the continuous fevers were further differentiated they included typhus, typhoid, and relapsing fevers. Though the distinction between typhus and typhoid was convincingly demonstrated by Sir William Jenner in 1849 to 1851, the absence of characteristic features in individual cases led to difficulties, and until comparatively recently these two fevers were still in Germany included under the common name *typhus*, the former being called typhus exanthematicus, from the prominence of its eruption in typical cases, and the latter typhus abdominalis, from the presence of intestinal lesions with their corresponding symptoms. In English practice for many years the term *enteric fever* has also been in use both to represent this characteristic feature and to accentuate the difference from the pathologically distinct typhus. This distinction was confirmed when, in cases of typhoid or enteric, Eberth discovered a bacillus, now known as *B. typhosus*. In 1901, however, it was found that in some of these cases, which clinically could not be regarded as other than enteric, the causative bacillus differed in certain particulars from Eberth's organism. Two such organisms have been identified which have been called *B. paratyphosus A* and *B. paratyphosus B;* and it is found, as a rule, with certain exceptions to be mentioned hereafter, that the serum of patients suffering from typhoid fever will agglutinate (*see* p. 16) only the *B. typhosus*, while the serum of a patient with paratyphoid fever, whether *A* or *B*, will agglutinate the paratyphoid bacillus, whether *A* or *B*, which is the cause of his illness.

At present, therefore, it is convenient to speak of all these forms as Enteric Fevers, or forms of enteric fever, and to distinguish them as

Typhoid Fever, due to the *B. typhosus;*
Paratyphoid Fever *A*, due to *B. paratyphosus A;*
Paratyphoid Fever *B*, due to *B. paratyphosus B.*

Typhoid fever will be first described, and subsequently the differences which are presented by the fevers caused by paratyphoid organisms.

## TYPHOID FEVER

*Typhoid fever is infectious* chiefly through the excretions. It has a febrile period of about three weeks' duration, and occasionally one or more relapses of the same length. The distinctive pathological lesion is inflammation and ulceration of Peyer's patches in the small intestine.

The specific micro-organism discovered by Eberth is a bacillus, 2–3$\mu$ in length, with round ends, and provided with from eight to twelve fine flagella of about twice its length. It bears a close resemblance to the *B. coli communis*, but can be distinguished from it by bacteriological tests. Eberth's bacillus has been found during life in the stools, in the blood, in the urine, in the sputum, and in the pus of abscesses resulting from periostitis and other similar lesions months and even years after the attack. After death it has been found in Peyer's patches, in the mesenteric glands, spleen (abundantly), liver, gall bladder, kidneys, meninges, bone marrow, and, rarely, in the lungs and testicles.

**Ætiology.**—Enteric fever shows little preference for either sex; but age has a marked influence, and the disease is much more frequent amongst young people. The quinquennial period which presents the highest percentage of cases (viz. 27 per cent.) is that between fifteen and twenty years; nearly 50 per cent. of the cases occur between fifteen and twenty-five, and more than 84 per cent. between five years and thirty (Corfield). The disease does, nevertheless, occur (1 or 2 per cent.) in people over sixty-five years of age. It is more prevalent in the latter part of the year—that is, in the four months August to November inclusive—and cases are more numerous during hot and dry weather than under the opposite condition. It is not affected by overcrowding, poverty, and uncleanliness, in the same way as typhus and relapsing fever, which are transmitted by external parasites. As a rule, doctors, nurses, and students in hospitals do not take enteric fever directly from the patients. The agent of transmission is, in the vast majority of cases, the fæces; and in those not very common instances in which nurses have contracted the disease from their patients it was probably by direct contact with linen or bedclothes soiled with the fæcal discharges. But bacilli are found in the urine in some cases, and are constantly present in the pus from the bone lesions (*e.g.* periostitis) which sometimes follow typhoid fever; both these secretions may therefore be the means of transmitting the disease.

When more than one member of a household has contracted the disease, or where the members of a group or a community are concerned, it is either because they have been exposed to the same common risk, or because infection has been conveyed indirectly from the fæces of the first sufferer in the ways here indicated. Thus a frequent cause of the spread of typhoid fever in a town or a country district is the contamination of its water supply by the stools of a single case. The opportunity arises from the imperfect means so often employed for the disposal of sewage. In the country, wells used for drinking water may be poisoned in consequence of the soil being saturated with sewage which has leaked from a neighbouring privy or imperfectly constructed cesspool. In one case a well was contaminated by the slops from a laundress's house leaking into it; enteric fever broke out in the house supplied by the well shortly after the laundress had received some linen soiled by the discharges from a patient with this disease. Where the drinking water is conveyed by pipes, the disease may find an entrance if the pipes by any chance are defective, and if they lie in a porous soil sufficiently close to any collection of sewage, imperfectly confined, which has received any enteric stools; and a whole reservoir may be infected in the same way. Drinking water is, however, not the only source of danger. Epidemics of enteric fever have been traced to the milk supply, the probability being that the milk itself has been first infected by being stored in vessels washed with water exposed to contamination by typhoid sewage. Typhoid has also been traced to ice-creams sold in the street, and to oysters, cockles, mussels, and clams, supplied from

breeding areas exposed to sewage contamination, and eaten uncooked. Watercress or celery may be an agent in a similar way.

Contamination of food by flies, which have had access to the excreta of enteric patients, may spread infection. This fact was proved by experience in the Spanish-American war in 1898 and in the South African war. Experimentally it has been shown that flies which had been in the neighbourhood of cases of enteric fever could infect sterile tubes of media from which on incubation typhoid organisms were isolated.

But the transmission of the disease by the bacillus is not confined to the period during which the patient is suffering from the fever. Months or years after recovery, when apparently in perfect health, he may still be harbouring the bacillus; and indeed the organisms may exist in those who are not known to have had typhoid fever at any time, and yet may be the cause of infection to persons coming into contact with them. These are all called *typhoid carriers*, and are divided into groups. *Convalescent* or *temporary carriers* are those who have had the disease within two or three months; *chronic carriers* are those in whom the bacillus persists for months or years; *healthy carriers* are those who are not known to have had the fever, and yet are infecting others; and *early carriers* are certain persons, also healthy at the time when bacilli are found in their fæces, who only subsequently develop typhoid fever. The test of a carrier is that the bacilli should be found in the fæces or in the urine; in a large proportion the blood gives the Widal reaction. That the bacillus of typhoid fever is constantly found in the gall-bladder is well known, and in a large proportion of chronic carriers gall stones are formed and give rise to the usual difficulties. The carriers infect others by direct contact, or by unconsciously conveying the bacilli to water, milk, or things they handle.

**Morbid Anatomy.**—The essential lesions of enteric fever occur in the *Peyer's patches* and *solitary follicles* of the small intestine. These become infiltrated with lymph corpuscles, and a Peyer's patch so affected swells, and projects one or two lines upon the inner surface of the intestine; it is grey, fawn-coloured or pink, but the surrounding mucous membrane may have its natural colour. The lymph corpuscles at first multiply in the follicles, but subsequently infiltrate the mucous membrane above and the deeper structures below. As the patches become larger they acquire a creamy-white colour, and about the tenth day or a little later they begin to ulcerate or slough, presenting at first a superficial abrasion at one point of the surface, which becomes deeper and deeper until a great part of the gland is removed; or a whole patch may slough at once. When the slough is still adherent, it is often stained yellow by bile pigment. By these processes the muscular coat or the peritoneal covering may be exposed in the floor of the ulcer, and finally the peritoneum may slough, ulcerate, or tear, so that the contents of the bowel escape into the peritoneal cavity and set up intense peritonitis. The stage of ulceration generally occupies part of the third week, and towards the end of that time, in favourable cases, the process of healing by cicatrisation begins. Ulceration does not necessarily occur: in mild cases the inflammatory swelling subsides without any destructive change. The number of Peyer's patches affected is very variable, and though the cases with severe diarrhœa generally have extensive inflammation of the bowel, there is no necessary correspondence between the extent of ulceration and the severity of the other symptoms. The patches near the ileo-cæcal valve are those first attacked, and the process spreads upwards. The change in the solitary follicles of the lower end of the ileum is of the same kind, and in some cases the lymphoid follicles of the large intestine (mostly the cæcum) are also enlarged and ulcerated. Coincidently with these lymphatic structures of the intestines the *mesenteric glands* are inflamed; they are enlarged, fleshy, pink, red, or purplish, and their histological changes resemble those of the Peyer's patches. Sometimes they soften into one or more small collections of fluid resembling pus, which in rare

cases burst into the peritoneum; but they may become cheesy or even calcareous. Quite exceptionally fatal cases have occurred in which no intestinal lesion could be found. The *spleen* is commonly enlarged, dark in colour, and, in later stages of the disease, softened. The *liver* is often hyperæmic, and softer than natural; the *kidneys* are congested, and in both these organs the gland cells are granular. The *heart* is often soft and flabby, its muscular fibres being in a state of fatty and granular degeneration. Zenker's degeneration (*see* p. 22) occurs in the voluntary muscles, and was indeed first described in connection with enteric fever. The *lungs* are either œdematous, or congested at the bases; or, in occasional cases, there is actual pneumonia.

**Symptoms and Course.**—The period of *incubation* of enteric fever is variable; but many cases in which it has been ascertained have shown it to be about a fortnight, or between ten and fifteen days. In exceptional cases it may be as short as five or as long as twenty-two days. The beginning of the disease is often very little marked. The patient feels ill, depressed, unfit for work; he has headache, pains in the limbs and back, loss of appetite, and perhaps nausea. These may come upon him so that he scarcely knows when they began, but he can frequently fix a day on which he says he first fell ill. Often the headache is severe, and forms the most prominent complaint. There may be diarrhœa in the first few days. Constipation, however, is also extremely common. Sometimes on the first feeling of illness a purgative is taken, and the bowels continue loose. The patient may go about, struggling to do his work, for five or six days, but generally towards the end of the week he is obliged to give up and take to his bed. The temperature has been stated, in the first four or five days of enteric fever, to rise two degrees each evening, and to fall one degree each morning, so that at the end of that period it will have reached 103° or 104°. So many cases escape accurate observation in the early days that it is not always easy to confirm this, but it is certain that in some cases the thermometer may rise on the first evening of illness to 103° or higher. The high level of 103° to 104° once reached, the temperature commonly remains at nearly the same level till the tenth to the fourteenth day, oscillating, however, between morning temperatures of 102° to 103° and evening temperatures of 103° to 104·5°. The pulse is quick, full, soft, and markedly dicrotic. Though in some cases very rapid, it is generally, in relation to the temperature, much slower than in typhus and many other febrile conditions; it may never exceed 100, and a pulse of eighty may co-exist with a temperature of 102° or 103°. The respirations are increased in frequency, and there is very frequently slight bronchitis, indicated by sibilant rhonchi, and accompanied, it may be, by mucous expectoration. About the seventh to the tenth day the patient commonly begins to present the characteristic appearance of enteric fever. He is dull, listless, apathetic, but not so dull and stupid as in typhus; the eyes brighter, the pupils often dilated; the face pale, with flushed cheeks and dark lips; the tongue dry, with a band of dry white fur on each side, the sides, tip, and middle clean and red. As the disease progresses, or in severe cases, the tongue tends to become thickly furred and dry all over its surface. Occasionally profuse perspiration occurs, or bleeding from the nose. At the end of the first week, or later—that is, from the sixth to the twelfth day—appears the characteristic *rose rash* of enteric fever; it consists of rose-pink spots, circular, slightly raised above the surface, flat, convex, but not pointed, so that they are often described as *lenticular*, from 2 to 4 mm. in diameter, disappearing under firm pressure with the finger, and never petechial like the typhus rash. They are seen first on the abdomen and front of the chest, and may be confined to these parts; but they also occur on the sides, back, and the upper arms and thighs. In number they vary from half a dozen to twenty or thirty, but they may be much more numerous, and in a certain number of cases (10 to 20 per cent.) are entirely absent. Each spot has a limited duration, gradually fading in three or four days; but spots continue to come out day after day until the end of the

third week, or in some cases even later. They are not visible after death. In the second week also the *intestinal symptoms* become prominent. The abdomen is generally full, even distended, and resonant on percussion; and there may be both tenderness and pain, but the former is more common than the latter. Pressure in the right iliac fossa, over the seat of the cæcum and lower end of the ileum, often elicits a little pain. *Diarrhœa* is a familiar symptom of enteric fever, but it is very variable in duration and in severity. Often there is a sharp attack of diarrhœa in the first week, and after this the bowels are confined; sometimes (up to 40 or 50 per cent. of the cases in some epidemics) there is *constipation* throughout. In other cases diarrhœa is constant, and the motions number three, four, or five or more daily. The stools, moreover, are distinctive in being liquid, of the colour of pea-soup, and of a peculiar offensive odour. They commonly contain particles of undigested food, intestinal epithelium, bile pigment, micrococci and bacilli, crystals of ammonium-magnesium phosphate, and after a time shreds of sloughs from the diseased Peyer's patches. They are alkaline and ammoniacal. The intestinal lesions further show themselves occasionally by the occurrence of *hæmorrhage*. This often happens in the stage of separation of the sloughs or of ulceration, and large quantities of bright red blood are discharged from the bowel, so as to cause severe collapse, with pallor and depression of temperature; but the bleeding may be quite slight, and this more often in the

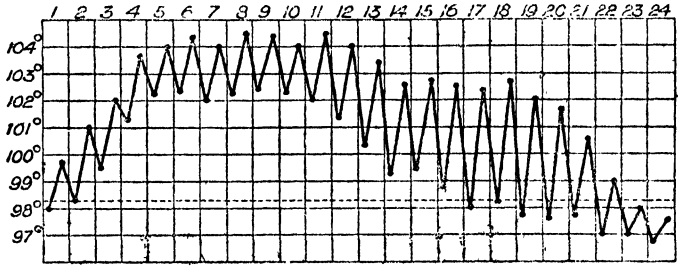



FIG. 9.—Temperature in Enteric Fever.

earlier stages of the illness. The *spleen* is generally enlarged; this may be manifest only from the results of percussion; but in most cases the organ can be felt on deep inspiration 1 or 2 inches below the costal margin. The *urine* is scanty, dark, and of high specific gravity; the urea and uric acid are increased, but the sodium chloride is much diminished. Late in the illness albumin is found in a small proportion of cases. But for the headache and some giddiness the cerebral functions may be very little disturbed in mild cases; the headache rarely lasts beyond the tenth day, and there may be then only a little drowsiness or tendency to wander at night. A temporary deafness is not uncommonly noticed. Such mild cases reach their acme at the end of the second week—the tenth to the fourteenth day. The temperature then takes a characteristic course; hitherto standing always at a high level, it now falls every morning quickly lower and lower, while the evening temperatures, though also falling, come down much less rapidly. Thus the morning temperature in four or five days reaches 99° or 98°, while the evening temperature stands at 102° or 101°. This may be called the *remittent* stage. From this point to the end of the illness the fever has for three or four days an *intermittent* character; it is about normal in the morning, but rises to 101° or more in the evening. Then rather suddenly the evening fever ceases, the temperature remains normal or subnormal, and convalescence has commenced (Fig. 9). During this falling temperature spots may continue to come out, the spleen is still perceptible, and there may be a little diarrhœa; but the mental condition of the patient generally improves, and he often acquires an appetite some days before the fever has entirely left him.

On the other hand, the graver cases are mostly accompanied by an increase in the intensity of the nervous symptoms, to which the symptoms of cardiac failure, or severe abdominal troubles, may be added; more or less continuous delirium may supervene, with drowsiness or even coma, extreme muscular prostration, subsultus tendinum, and plucking at the bedclothes. The face becomes dusky, the tongue dry, sordes collect on the lips and teeth, the pulse is rapid, soft and dicrotic, the heart sounds are feeble, and the bases of the lungs are congested, as shown by râles and a very feeble respiratory murmur. The urine may be retained, or both fæces and urine are passed unconsciously. The condition resembles that described under Typhus Fever; the patient is indeed in a truly *typhoid* state. The delirium is less often violent than in typhus, but occasionally patients get out of bed, or refuse food. Cardiac failure shows itself by the feebleness of the heart's beat, the indistinctness of the sounds, the small, rapid pulse, and by venous congestion of the face and extremities, and of the

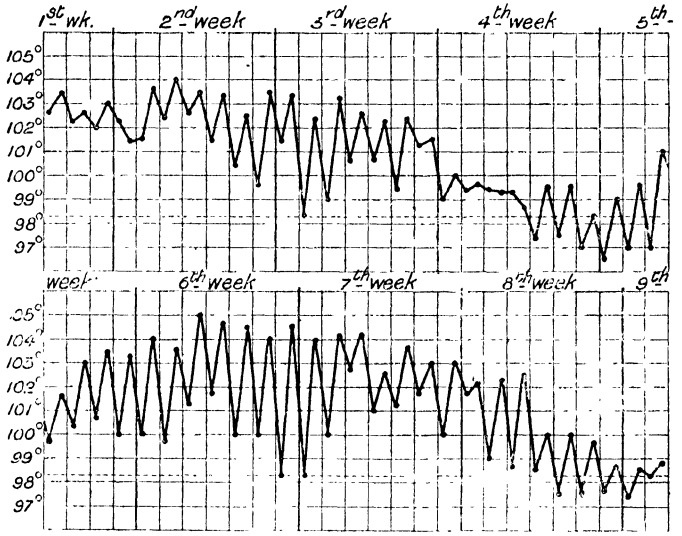



FIG. 10.—Temperature in a Case of Enteric Fever with Relapse

bases of the lungs. Sometimes there is evidence of cardiac dilatation in displacement of the impulse outwards, and the pulse may be irregular or intermittent. With the increase of the nervous symptoms the abdominal troubles are often prominent, the diarrhœa becomes profuse, and the abdomen is much distended, tense and tender; in this stage the ulcerated bowel may give way, and peritonitis may result from the escape of fæcal matter into the cavity of the abdomen. In a few cases severe bronchitis is the main feature of the disease; the face is livid, râles and rhonchi are heard over the whole chest, and breathing is seriously obstructed. Under these various circumstances death may occur almost at any time after the tenth or twelfth day; but recovery occurs after lengthened periods of coma and other severe symptoms, the temperature slowly returning to the normal, and convalescence being very protracted.

**Relapses.**—A true relapse of enteric fever occurs in a certain proportion of cases, which have been found by different observers to be from 3 to 10 or 11 per cent.[1] It consists of a repetition of all the phenomena of the illness: ulceration of Peyer's patches, fever, diarrhœa, and rose spots; and it occurs after an interval

[1] At the Metropolitan Asylums Board's hospitals in twelve years from 1900 to 1911 the percentage was 10·3. In 1914 in 316 cases the percentage of relapses was 6·96.

which may be as long as eleven days from the termination of the original fever, but is often much less. Sometimes, indeed, there is no interval of actual apyrexia, and the relapse seems to be continuous with the primary fever. Its duration is often quite as long as that of the first attack (Fig. 10), and, as a rule, it is somewhat milder. Death may, indeed, take place in the relapse, but this is more often from complications, such as perforation of the intestine and peritonitis, or hæmorrhage, than from the severity of the pyrexia or toxæmia alone. Occasionally a second relapse occurs after another interval of apyrexia; and even third and fourth relapses have been observed, though very rarely.

**Complications.**—The complications are numerous and varied, but a large proportion of patients escape them. As might be expected, the most important are those connected with the intestinal lesions.[1] *Hæmorrhage* (*6·01*) has been already mentioned (*see* p. 83). *Peritonitis* is a frequent cause of death. It arises most commonly from perforation of the floor of one of the ulcerated Peyer's patches, through which the contents of the bowel are extravasated into the peritoneal cavity; it occasionally happens from extension of inflammation through the peritoneal coat without any perforation being discovered; and it has also been caused in rare cases by the softening of inflamed mesenteric glands and infarctions in the spleen, and by rupture of the gall bladder. *Perforation* of the bowel takes place in more than 30 per cent. of fatal cases of typhoid fever. In more than two-thirds of the cases it occurs in the second, third, or fourth week, but it is rare before the ninth day. The perforation almost always occurs in the last 2 feet of the ileum, and not infrequently there may be two or more perforations. Its onset is often accompanied by acute pain, collapse, and perhaps vomiting or rigors; the abdomen is tender, sometimes flat and rigid, at others distended, but in both cases moving scarcely at all on respiration; the pulse is small and rapid, and the temperature sometimes falls. But its advent may only be marked by collapse and increased distension; and in very severe cases, with much distension of the bowel as well as coma and delirium, there may be no certain signs to indicate peritonitis, so that perforation and peritonitis are occasionally found post mortem when not suspected during life. A progressive increase in the total leucocyte count from hour to hour is a valuable diagnostic point in doubtful cases. So long as the ulcer remains unhealed there is a possibility of a rupture taking place; and such a rupture may be induced by any disturbance of the bowel, as by vomiting, defæcation, the exertion of sitting up, or the administration internally of indigestible food or of aperients; and thus even cases which are running a mild course may be fatal from this cause. *Ulcerations of the pharynx* have been observed. They are mostly superficial, situate upon the pillars of the fauces, and sometimes accompanied by swelling of the lymphatic glands; they may occur early in the illness, and have been wrongly attributed to syphilis or diphtheria.

A slight amount of *bronchitis* is frequent in enteric fever, but occasionally it is so severe as to constitute a very serious complication. The face may be quite livid, and a more or less venous tinge may be given to the whole surface; the chest is filled with râles and rhonchi, and there is expectoration of mucus or muco-pus. *Ulceration of the larynx* occurs sometimes in severe cases. The ulcer is situate commonly over the arytenoid cartilage, and this may be even exposed and in a state of necrosis. Sometimes an abscess forms around the cartilage in consequence of *perichondritis*. As results of these laryngeal complications there may be hoarseness or complete aphonia; subcutaneous emphysema, from air being forced during expiratory efforts from the larynx into the

[1] The bracketed figures in italics represent the percentage occurrence of each complication among 316 cases in the Metropolitan Asylums Board's hospitals in 1914; they differ from those representing the complications in 9,462 cases in the twelve years 1900 to 1911 given in the tenth edition of this book, p. 112, in the less frequency of hæmorrhage, and the greater frequency of broncho-pneumonia.

connective tissues; and cicatricial stricture of the glottis in cases that recover. A temporary aphonia may occur without any evidence of ulceration. *Pneumonia* sometimes becoming gangrenous, *broncho-pneumonia* (*5·37*) and *pleurisy* (*0·9*), both serous and purulent, occasionally occur, and much more rarely *pneumothorax*. *Jaundice* is of rather rare occurrence; it may be due to hepatitis or to the well-established affinity of the bacillus for the gall bladder. The stools are not necessarily deprived of bile pigment, and recovery may take place without any further indications. In some cases local diseases independent of enteric fever cause the jaundice. *Cholecystitis*, or inflammation of the gall bladder, is a result of direct infection of that organ. *Acute nephritis* (*1·26*), sometimes with abundant albuminuria or hæmaturia, may occur. In about one-fourth of the cases of enteric fever the bacilli are found in the urine, especially in the third week; sometimes they are so abundant as to cause a visible deposit (*bacilluria*), and sometimes they give rise to *pyelitis* or *cystitis*. They may persist there for years, as has been stated in connection with typhoid-carriers (*see* p. 81). *Otitis* (*3·16*) and *otorrhœa* may occur during or after the fever, and may lead to deafness, or to the more serious conditions of *septicæmia* and *meningitis*. Nevertheless meningitis is quite rare as a complication of enteric fever, and the cerebral symptoms commonly occurring are independent of cerebral inflammation. Meningitis from the typhoid bacillus has been recorded as occurring without intestinal lesions. Double *optic neuritis* is sometimes seen, but it is rare. Other local inflammations occasionally occur either during the fever or during convalescence, and may considerably delay recovery, such as *parotitis* (*0·31*), which may be followed by suppuration, or extensive infiltration of the neck; *orchitis; myositis; cancrum oris;* abscesses (*2·21*), boils (*0·63*) and facial *erysipelas*. *Periostitis* (*1·26*) occurs especially on the tibiæ, but also in other bones, such as the ulna or metacarpals; and *perichondritis* of the costal cartilages may occur. Pain in the lumbo-sacral region aggravated by walking, and persisting for a long time, has been called *typhoid spine;* in some cases the Röntgen rays have shown ostitis, periostitis and perichondritis about the lumbar or lower dorsal vertebræ. Another condition is described as *tender toes;* in this the toes and soles of the feet are painful on pressure during attempts at walking. In severe cases *bed-sores* may form, in spite of careful nursing. *Thrombosis* (*2.21*) of the femoral vein, generally on the left side, may occur during early convalescence, leading to œdema of the foot and leg, and tenderness in the course of the vein. It mostly subsides without much trouble, but the thrombosis may extend into the large abdominal veins, or portions of clot may be detached, and lead to pulmonary embolism and death. *Rigors* are of rare occurrence. They may be due to complications, such as constipation or pneumonia; but they sometimes happen without recognisable cause. Among the nervous sequelæ, besides meningitis, are *encephalitis* (rarely); *mental disturbances; peripheral neuritis* (*0·31*); and, rarely, localised muscular atrophy.

**Varieties of Typhoid Fever.**—There are few diseases more variable than typhoid fever. Though its duration is characteristically three weeks, it may be as short as ten days or as long as five or six weeks; and though short attacks may sometimes be fairly represented as abortive attacks, they may be followed by a relapse of precisely the same nature and duration. Sometimes the temperature begins to fall in the manner described (*see* p. 82), and then, before reaching the normal, persists in its remittent type, oscillating between 100° (morning) and 102° (evening) for eight or ten days, so that the fever is prolonged into the fifth week, although the patient is feeling better every day, and has no obvious complications. In other cases the prolongation of the fever corresponds with a continuance of the high temperature characteristic of the second week, and these are generally severe cases. In some cases the illness is so slight that patients go about their ordinary occupations until, perhaps, an indiscretion in diet or the use of aperients, given in ignorance, leads to a fatal perforation.

Cases so mild as this in their general symptoms, and yet so dangerous from their possible termination, have been called *ambulatory typhoid*. Ataxic and adynamic forms have been described, but these terms simply indicate the predominance of symptoms in one or other system of the body. Very rarely a *hæmorrhagic* form occurs, in which there are purpuric eruptions on the skin, bleeding from the mucous membranes, epistaxis, hæmoptysis, hæmatemesis, and hæmorrhage into the muscles and internal organs. (Compare Measles and Small-pox.) Typhoid fever is very often mild in children, often of short duration, and associated with less extensive disease of Peyer's patches than in the average of adult cases. The remissions of temperature, which are well marked in the latter half of the illness in adults, are often still more marked in children, and the "infantile remittent fever" of older writers was undoubtedly enteric fever. In elderly persons also the rose spots and enlarged spleen are often absent.

**Diagnosis.**—In every case of pyrexia which is in any way suggestive of enteric fever, an attempt should be made at the earliest possible moment to isolate the organism by blood culture. Blood is withdrawn from a vein under strictly aseptic precautions; enough sterile sodium citrate is added to make a 0·5 per cent. solution. This is to prevent clotting. The blood is then mixed with 5 to 10 times its volume of sterile broth and incubated at 37° C. At least 5 c.c. of blood should be withdrawn and several culture tubes prepared. Blood culture gives a high proportion of positive results if performed within the first seven days of the disease. It may, however, be positive very much longer, and, at whatever stage of the disease the patient is seen, it should always be attempted. In cases of over seven days' duration a specimen of blood should also be taken in order to test the agglutinating power of the serum against the enteric group of micro-organisms (Widal's reaction). It is advisable as a routine measure to send specimens of fæces and urine for cultivation. Positive results, however, cannot be secured at all stages of the illness, and negative reactions must not be hastily regarded as excluding the disease. It is therefore necessary to give full consideration to all the clinical features of the illness, as well as to the several points in the ætiology and history of the case. The Widal reaction is not usually positive until about the fourteenth day of the disease, but may remain so for many months after convalescence. The value of the reaction in the diagnosis of the paratyphoid fevers and in persons who have been inoculated is discussed later.

A great number of diseases may be confounded with enteric fever, from the variety of forms which it assumes, and from the frequency with which its own typical symptoms are absent or badly marked; but it may be briefly stated that the characters which are the most constant and the most suggestive of the enteric fevers are *headache*, *persistent fever*, *rose spots*, and *enlarged spleen*.

In early stages it is distinguished from the exanthems by the absence of characteristic eruption. By the fifth day of the illness the rash of typhus, small-pox, or scarlet fever would have developed; the appearance of rose spots a few days later confirms the diagnosis of enteric fever. Severe joint pains may lead to a suspicion of rheumatic fever. A prolonged febrile complaint which has come on insidiously, and presents no obvious local lesions, should always make one think of enteric fever; but the great prevalence of *influenza* gives rise to frequent mistakes. For though influenza is often a much more sudden and quickly prostrating disease, it presents so much variety that almost any illness beginning with headache, backache, and fever is liable to be mistaken for it. If typhoid fever is present, the temperature remains high, or even rises, and the diagnosis may be soon confirmed by diarrhœa, enlargement of the spleen, or rose spots. Tenderness over the gall bladder and muscular resistance in the right hypochondrium are said to be early signs of typhoid infection, but of course they may be due to local inflammatory lesions.

Later stages present a resemblance to different diseases according as the head, chest, or abdomen shows the most prominent disturbance. Thus the early

headache of typhoid and the subsequent delirium may suggest *meningitis*, and the two diseases are frequently confounded together. Lumbar puncture and the examination of the cerebro-spinal fluid for cells and protein will decide the diagnosis. Sometimes it is impossible to distinguish them clinically until later stages, when optic neuritis or a local paralysis, squinting or convulsion, or the obstinately retracted abdomen, may decide for tuberculous meningitis, or, on the other hand, the increase of abdominal symptoms, with the presence of spots, may prove it to be typhoid fever. In this latter, headache rarely continues beyond the tenth day. When pulmonary symptoms are marked, *acute general tuberculosis* may be simulated by the abundant bronchitic râles and crepitations accompanied by a remitting fever. The abdominal diseases which may be confounded with typhoid fever are especially *tuberculous peritonitis* and *appendicitis*. In both there may be high fever, abdominal distension and tenderness; and in tuberculous peritonitis the stools may be frequent and yellow from accompanying tuberculous ulceration. Appendicitis is generally distinguished from typhoid fever by localised pain, rigor, and vomiting, neither of which occurs, as a rule, in typhoid; but, on the one hand, they are sometimes present in typhoid, and conversely an appendicitis may develop without causing these familiar evidences of acute local inflammation. The *pyæmic* or *septicæmic* condition associated with abscess or suppuration in other parts of the abdomen, such as hepatic abscess and perinephritis, may also give rise to confusion; and the rare disease *suppurative pylephlebitis*, in which local evidence of the liver being involved may be little or none, must not be forgotten. In most of these conditions *leucocytosis* is present. An allied condition, *infective* or *malignant endocarditis*, is not infrequently mistaken for typhoid fever. The symptoms in favour of endocarditis are the existence of a murmur, or of irregular action of the heart, hæmorrhages under the skin, or the evidences of emboli, such as obliteration of the pulse at the wrist or ankle, abundant albuminuria, or retinal hæmorrhages; rigors may be present, and the temperature often oscillates freely. Trichinosis, the disease caused by the multiplication of the *Trichina spiralis* within the body, has been mistaken for typhoid fever; it is distinguished by severe muscular pains, œdema of the eyelids, and sometimes of the whole body; and one finds neither rose spots nor enlargement of the spleen.

*Mediterranean fever* presents some resemblances to typhoid fever, and should be thought of when the illness has been contracted in places where the former disease is prevalent. Certain types of malarial infection, especially malignant tertian, may almost exactly simulate typhoid fever. They may be distinguished by the demonstration of parasites in the blood and the reaction of the pyrexia to quinine. It must not, however, be forgotten that malarial infection and typhoid fever not infrequently co-exist in the same patient. It is especially in a disease like this, and in others where positive symptoms other than the continued pyrexia are infrequent or inconspicuous, that the Widal test and the search for the actual organisms are likely to be of value. And such methods are of increasing importance as it is by them alone that the differential diagnosis can be made between typhoid fever and its paratyphoid relatives.

An examination of the blood (*see* Diseases of the Blood) may give some help in diagnosis. In all but the earliest stages of typhoid fever there is a reduction of the neutrophil leucocytes, which reach their minimum in the period of declining pyrexia. The lymphocytes are also diminished at first, but increase again at the end of the stage of continuous pyrexia, and remain abundant throughout the fever, and for some weeks into convalescence. Eosinophils disappear at first, and reappear with the increase of the lymphocytes (Nägeli). Secondary infections, or other complications, may increase the leucocytes again, especially the polymorphonuclear cells.

Marris has found that in the enteric group of fevers the heart's action is not quickened by atropine to the same extent as it is in healthy persons. This

difference is observed about the tenth day of the fever, though sometimes earlier; and the normal response to atropine may be resumed at any time after the fourteenth day, though it may persist much longer, and will vary with relapses. In persons over fifty years of age, and in those suffering from heart disease or from arterio-sclerosis, the failure to quicken may be due to pre-existing cardio-vascular changes, and not to the typhoid poison.

The pulse rate is taken and recorded minute by minute till it is steady. One thirty-third of a grain of atropine sulphate is then injected subcutaneously, best over the triceps. After twenty-five minutes the pulse is again recorded minute by minute till it is clear that its rate has reached its highest point, and is now falling.

Marris concludes that an increase of the pulse rate by twenty or more beats in the minute after atropine may be accepted as an indication that the patient is probably not suffering from typhoid fever or one of the paratyphoid series. An increase of less than ten beats is suggestive of infection by one of these diseases. Readings between ten and twenty are uncertain.

**Prognosis.**—The mortality of typhoid fever varies in different epidemics from 5 to 20 per cent. Complications contribute largely to the deaths, and their occurrence will modify the prognosis at any time. Apart from them, the intensity of the fever is an important guide. If the temperature is, although high at the end of the first week, subsequently never above 103°, the case is favourable; if the temperature is maintained at 104° or higher throughout the second week, it is much more dangerous. Some cases sink rapidly by the twelfth, eleventh, and tenth days, or even before this. Perforation is almost certainly fatal unless it is promptly treated by surgical methods. Hæmorrhage is less dangerous, but may be responsible for about one-fifth of the deaths; and the mortality amongst cases with free hæmorrhage is much above the average. A severe hæmorrhage, even if not fatal, renders the patient very anæmic, and considerably prolongs convalescence. Much abdominal distension, profuse diarrhœa, incontinence of urine and fæces, severe general bronchitis, and a feeble and irregular heart, are all unfavourable.

**Prevention.**—Typhoid fever is spread mainly by the fæcal discharges infecting the water supply, and the greatest possible care must be taken to prevent any such contamination when a case is first recognised. The more perfect the sanitary arrangements, with reference especially to water supply, drainage, and the removal of sewage, the less likely is this to take place. Typhoid discharges cast into sewers without previous disinfection render the contents infectious, and may poison drinking-water when the drains, sewers, or wells are faulty. The same discharges soiling bed-linen, clothes, towels, and similar things, may directly, or by infecting washing-water, be the means of conveying infection to remote parts. The above indicates the direction in which one should act: disinfection of the discharges and of everything soiled with them, and constant watchfulness to avoid contamination of water, as well as air and food. Cleanliness in every way helps in this respect.

*Preventive Inoculation.*—But even if the risk of exposure to infection cannot be avoided it is possible to diminish the susceptibility of the individual by the inoculation of a vaccine consisting of dead cultures of typhoid bacilli (*see* p. 15). This has been done as a routine measure in the army for some years. A vaccine containing 500 millions of bacilli is first injected, and a second injection of 1,000 millions is made ten days later. A certain amount of local and general reaction takes place, but soon subsides. It is estimated that the liability to the disease was thereby reduced to one-eighth, and the mortality among those who had the disease to one-half; but still more striking figures have been published. Moreover, both larger and more frequent doses have been inoculated. An inoculation of 1,000 millions should be repeated at about yearly intervals.

The treatment of typhoid-carriers presents many difficulties. Intestinal

antiseptics are valueless; and the gall bladder has been drained, and even removed, without notable success. The use of vaccines has been tried in doses of several millions of sterilised typhoid bacilli at intervals of two or three weeks; but even if the organisms disappear for some time from the urine and fæces the permanence of the cure cannot be guaranteed. The only efficient means of meeting the difficulty of the typhoid-carrier is to keep him under observation and ensure disinfection of his excreta until he is shown to be permanently free. Particular care must be taken that his employment does not in any way necessitate his handling or preparing food for other persons. In purely urinary cases urotropine will reduce the number of bacilli as long as it is being taken.

**Treatment.**—The patient should be in bed in a well-ventilated apartment, and the same rules should be carried out as to nursing as in the case of other infectious fevers. The special dangers of perforation and hæmorrhage from the ulcerated bowel should never be lost sight of. Rest should be absolute; the patient should be allowed no exertion, and a bedpan should be used when he wishes to pass his motions or urine. The diet should be fairly liberal, and varies considerably with the patient's appetite and his power of assimilating food. Milk forms the greater part of the diet, but may in many cases be supplemented by soft solid food, such as custards, junkets, bread and butter without crusts, mashed potatoes, eggs, either raw or soft-boiled, and milk chocolate. Plenty of carbohydrate should be given, such as dextrose, milk sugar, or ordinary sugar dissolved in milk. Feeds should be small in quantity and frequently administered. When milk is taken badly, or if the stools show, by the presence of curds, that it is imperfectly digested, it may be peptonised by the use of Benger's liquor pancreaticus. The patient should be encouraged to drink large quantities of water, barley water or lemonade. This causes a polyuria, and helps to combat the toxæmia. Sugar should be added to all fluids as far as possible.

As to medicinal treatment, in mild cases little or none is wanted. A small dose of dilute mineral acid, or of a saline diaphoretic like the acetate of ammonium, may be grateful to the patient; and the body may frequently be sponged with tepid water. Stimulants are often unnecessary. If required, they may be given on the principles laid down (*see* p. 26).

Special symptoms of complications may have to be met, such as bronchitis, by small doses of expectorants, or persistent headache by phenacetin, 5 to 10 grains, or aspirin, 5 to 10 grains. If the bowels are not opened more than four times in the twenty-four hours, no treatment is required; but it is generally desirable to check diarrhœa if it exceeds this limit, and this is best done by the use of a starch enema with 15 or 20 minims of tincture of opium; and bismuth carbonate or salicylate, or the vegetable astringents, may be given internally. Any linen that is soiled by fæces or urine should be at once removed, not only for the sake of keeping the patient clean and free from the risk of bedsores, but also to prevent the possibility of the attendants being infected.

If constipation occurs, the bowels may be left for two or three days without harm, and it is then safest to use a soap enema from time to time as required. In cases where meteorism is marked, this symptom may be relieved by injection of 1 c.c. of pituitary extract.

This purely expectant line of treatment suffices in many cases, especially those of milder type; the patient is cared for while the disease runs its course. The methods of treatment by which it has been sought to influence directly the progress and prevent the accidents of the fever are those by (1) antipyretics, (2) antiseptics, and (3) antitoxic serum or vaccine.

The theory of the *antipyretic* method was that the continued high temperature to which the tissues and organs of the body are subjected is the chief cause of their granular degeneration, and that their ultimate failure, or the occurrence of complications, is so much more probable the higher the fever. Hence our object should be to bring down the *mean* temperature, by lowering the tempera-

ture 3° or 4°, at more or less regular intervals. This theory can scarcely be maintained now, with our modern knowledge of bacteria and toxins. Nevertheless the practice is sometimes of value in typhoid fever, though it does not cut short the illness.

The methods employed have been already described under the general treatment of fevers (*see* p. 24). Of these the external application of cold gives the best results. The influence of a single bath upon the immediate condition of the patient is generally pronounced. Headache, delirium, stupor, thirst, are at once diminished, the tongue becomes clean, the pulse slower and firmer, and the patient feels altogether relieved. But this effect is only temporary, and by the next observation the temperature may be as high as before. When the system is thoroughly carried out by frequent baths at low temperatures, or even by continuous immersion, it is claimed that the mortality has been reduced by 5 or 6 per cent., and even in its modified forms a decided improvement has been noted. The contra-indications are extreme collapse of the patient, hæmorrhage already established, and severe conditions of pulmonary congestion.

Cold sponging, wet packs, the application of ice-bags or ice compresses to the surface of the body, and the ice-cradle (the patient lying under a bed-cradle within which are hung small vessels containing ice) are less efficient than baths, but they give less trouble, and are often of real service.

Antipyretic drugs have been fully tried, but the disadvantages in the collapse and cardiac failure are generally felt to outweigh their apparent advantages.

The *antiseptic* treatment consists in the use, internally or by enema, of such drugs as carbolic acid, sulphurous acid, naphthol, hydronaphthol, naphthalene, bismuth salicylate, salol, oil of cinnamon and chlorine. They are said to diminish diarrhœa and tympanites, and to make the stools less offensive; but they have little or no influence on the changes in the bowel, or on the duration of the pyrexia, nor do they prevent relapse. The doses employed for adults have been of β-naphthol 3 to 5 grains, suspended in mucilage, every four hours, of hydronaphthol 2 to 3 grains every two to four hours, of sulphurous acid 20 to 30 minims, of salol 5 to 7 grains, and of oil of cinnamon 3 to 5 minims every two hours.

The treatment of enteric fever by a *serum* is yet in its infancy. The recognition that the poison of the typhoid bacillus was mainly an endotoxin, and that little exotoxin was formed, suggested that the bacterial cell juices should be injected into the horse in order to produce an *anti-endotoxic* serum. Such a serum, employed in the treatment of typhoid fever, has awakened hopes that it may influence the course of the disease (Macfadyen, Hewlett). Recent attempts at treatment by *vaccine* have not been encouraging, and there was a suspicion that the incidence of hæmorrhage was increased thereby.

For hæmorrhage from the bowels opium internally or morphia by hypodermic injection is probably the best treatment. Acetate of lead, tannic acid, oil of turpentine (10 minims), ergot, and adrenalin chloride have at different times been used, and more recently chloride of calcium in 10-grain doses every three or four hours.

Tympanites may be relieved by the application of ice, in small lumps, between two pieces of flannel, and by the use of turpentine enemata.

If perforation is recognised, laparotomy should be performed at once, the abdomen should be washed out, and the ulcer closed. Without operation the condition is practically invariably fatal. With operation the recovery rate is about 20 per cent. It has been shown that the prognosis is very much better if laparotomy is performed within twelve hours of perforation occurring.

Both for the treatment of bacilluria and cystitis when they occur, and for the prevention of infection in others, urotropine (10 grains three times a day) or helmitol should be used during the fever and for three weeks of convalescence.

During convalescence purgatives must be carefully avoided, or used only in

the form of enema. Even in favourable cases without complications or sequelæ the bodily and mental vigour returns with remarkable slowness, and the patient should not be too early allowed to exert himself. Rarely is he fit for work under three months from the commencement of the illness, and in the graver forms, or in case of relapse or of complications, this period may be prolonged to five or six months. Specimens of fæces should be examined bacteriologically on three separate occasions during convalescence in order that typhoid carriers may be detected.

## PARATYPHOID FEVERS

The organisms which cause paratyphoid fever are two in number, distinguished as *Bacillus paratyphosus A* and *B. paratyphosus B*. They differ from *B. typhosus* in cultural characters such as their fermenting action on different sugars, their action on milk, and their agglutinative reactions.

These two have had a somewhat different geographical distribution, the *B. paratyphosus A* having been found in many parts of the world, including Germany and India, but rarely in England, whereas the *B. paratyphosus B* was found more commonly in England, but also in Germany and America. But both forms have been present in the enteric fevers prevalent in the Dardanelles and in Egypt during the Great War, and there the cases of paratyphoid outnumbered those due to *B. typhosus*.

**Ætiology.**—The ætiological factors in the spread of the paratyphoid fevers appear to be the same as those which favour typhoid itself. The diseases are spread chiefly through the fæces of those who are ill, and of the different groups of *carriers*, but also by clothing, bedclothes, etc. Drinking-water, milk, flies, dust, shellfish and other agents are concerned in different cases (*see* p. 80).

**Symptoms and Course.**—The symptoms and pathology of the two forms are identical, and they can only be distinguished from one another by agglutination tests or by isolation of the bacillus from the blood or fæces. They are also not materially different from the symptoms and the pathology of typhoid itself, and the following are the chief conditions which have been observed to be more or less frequent than in typhoid. Thus it is stated that a sudden onset is more common in the paratyphoids; that the rose spots are redder and more profuse; that the spleen is more often or more considerably enlarged; that the pulse is unusually slow, as compared with the elevation of temperature, so slow even as fifty in the minute; that abdominal pain is not infrequent, and hence a risk of confusion with appendicitis, and if there is much diarrhœa with dysentery; but the great variations in the symptoms of typhoid fever must not be forgotten.

The duration of the pyrexia may be as little as ten days, but it may be as much as eighteen or twenty-one days, the longer period more often in paratyphoid A than in paratyphoid B. It oscillates often between 100° and 102°, and is rarely above 103°.

The spots are as variable in number as in typhoid, and in the shorter forms may not appear till near the end of the pyrexia.

In the paratyphoid fevers complications occur similar to those seen in typhoid fever; that is, there are inflammatory lesions of other organs besides the intestine, due either to the paratyphoid bacillus or to other infections. And sometimes these occur so early in the disorder as to distract attention from the main disease unless the specific tests are applied. By Dr. C. H. Miller several forms of paratyphoid fever are recognised, determined by the organs or systems most severely or earliest attacked in different cases. Thus he describes dysenteric, biliary, nephritic or urinary, pulmonary, rheumatic, influenzal and septicæmic varieties. Accordingly during paratyphoid epidemics the possibility of this infection must always be remembered in cases of jaundice, dysenteric diarrhœa, acute bronchitis, pneumonia, or nephritis.

The mortality among paratyphoid cases has generally been regarded as slight,

namely, from 1 to 3 per cent.; but in cases occurring among soldiers it has sometimes been as high as 5 or 6 per cent. (Miller).

Post mortem in some cases no intestinal lesions have been found, in others ulceration of Peyer's patches, or of the solitary follicles in the ileum, cæcum and colon. Enlargement of the spleen and of the mesenteric glands is also found.

**Diagnosis.**—The diagnosis between paratyphoids A and B and typhoid infection can only be made with any certainty by bacteriological examination and agglutination reactions. A positive finding from culture of the blood, urine, or fæces will decide the diagnosis at once. Failing this, the serum of the patient must be tested for agglutinating power against the various organisms. If the patient has never received any prophylactic inoculations against the enteric group of organisms, diagnostic results may be obtained by an agglutination test on one occasion only. If, however, he has at any time been inoculated with typhoid organisms or with a mixed vaccine of typhoid and the paratyphoids, the agglutination reactions are markedly affected. In such cases, to be of any diagnostic value the tests must be performed by the quantitative macroscopic method introduced by Dreyer. He has shown that there is a gradual rise followed by a fall in the agglutinating power of the serum towards the organism that is causing the infection. In order to establish this agglutination curve at least three tests must be carried out at intervals of about four days. Thus, for example, in a case of paratyphoid A who has been inoculated with all three organisms it will be found that the serum agglutinates all three, but that there is a rise of agglutinating power for paratyphoid A, followed later by a fall as convalescence is established.

**Prevention.**—The known ætiological factors, drinking-water, soiled clothes, flies, dust, etc., have all to be considered; and preventive inoculation by means of vaccines prepared from the two kinds of bacilli can be employed where, as in the case of a campaign, the risk of infection is almost certain to be incurred.

**Treatment.**—This is the same as is required in typhoid fever.

### PSITTACOSIS

This is a condition of septicæmia which occurs among parrots (ψιττακός, a parrot), and in which enteritis, sometimes hæmorrhagic, is a prominent symptom. It is due to a bacillus, belonging to the typhoid group, which is found in the spleen, the bone marrow, and the blood. The disease is communicable to other small animals and birds, and also to man, probably by the feathers soiled with fæcal matter. The incubation in man is about ten days; and then occur fever, headache, anorexia, restlessness, delirium, vomiting, diarrhœa and albuminuria. Broncho-pneumonia is observed in many cases, and the mortality is about 30 per cent. The organism has been isolated from the blood of the heart.

## CEREBRO-SPINAL FEVER

*(Cerebro-spinal Meningitis, Spotted Fever)*

This disease was first recognised at Geneva in 1805. Since 1860 it has been prevalent in the United States and in Germany. In 1846 it appeared in Ireland, and again in a severe form in 1866–68; and in 1906–8 some hundreds of cases occurred in Glasgow and in other towns in Scotland, and a few in London. The disease is endemic, sporadic cases occurring, especially in infants. In the latter the disease used to be described as *posterior basal meningitis*. Every now and then the disease assumes epidemic form. This occurred in this country in 1915 and 1916.

**Ætiology.**—The specific organism is a diplococcus (*D. meningitidis intracellularis* of Weichselbaum or *meningococcus*), which is seen in the polymorpho-

nuclear leucocytes of the meningeal exudations, but also lying free between the cells; it is also sometimes found in the blood, in pus from the joints, in pneumonic foci in the lungs, and in the mucus of the naso-pharynx. Meningococci when tested by their agglutination reactions fall into two main groups; they are sometimes called meningococci and parameningococci. Each group is again divisible, also by agglutination, into a number of different strains. As far as their cultural characteristics are concerned, the different groups cannot be separated from one another, and there is not much evidence that they produce different symptoms. Meningococci may be found in the naso-pharynx of perfectly healthy people, who are known as *carriers*. It is possible that the meningococci latent there assume a virulent form from time to time so that an epidemic breaks out. It is noteworthy that carriers do not very often themselves contract the disease; but they disseminate the organisms by projection of their saliva in talking and coughing, so that susceptible people become infected by inhalation and get the disease. Normally about 5 per cent. of the population are carriers; when the number reaches 20 per cent. an epidemic may break out. An epidemic does not sweep through the community like influenza; it is only people here and there who contract the disease. Previously the young have most often been sufferers, 80 per cent. of cases being under sixteen, and the most susceptible age of all being from birth to five years. This tendency was not shown in the 1915–16 epidemic. As a rule the sexes are attacked nearly equally. The disease is usually more prevalent in the winter months; it is probable that overcrowding, such as occurred among the troops at the beginning of the War, and the presence of catarrhal throat affections, owing to sneezing and coughing, tend to its dissemination.

**Pathology.**—Much discussion has taken place as to how the meningococcus obtains access to the meninges. The older view that it spread direct to the brain from the naso-pharynx *viâ* the sinuses or the cribriform plate has received some support from the finding of pus in the sinuses at post-mortem. However, in many cases suppuration of this kind has not been found although carefully looked for. On the other hand, the meningococcus was isolated from the blood and from skin lesions before the onset of meningitis in cases during the last epidemic, so that, as in other septicæmias, the meninges were probably infected *viâ* the blood stream, the naso-pharynx being the primary focus.

A third view has been recently expressed (**13**), viz., that infection passes from the blood to the choroid plexuses and then to the intraventricular fluid. The normal passage of the fluid carries it through the roof of the fourth ventricle out to the subarachnoid space, while there is a special tendency for the inflammatory products to accumulate at the periphery where the fluid is normally absorbed, *i.e.* at the superior longitudinal sinus, and in the spinal canal. The evidence for this is: (1) That in fulminating cases meningococci are found in the ventricles alone, while there is no perceptible inflammation of the meninges, but there is acute inflammation of the choroid flexuses; (2) In less acute cases the fluid from ventricular puncture, though it has the same composition as fluid from lumbar puncture, always contains fewer pus cells, suggesting that these have accumulated at the periphery. In favour of this view, it has been shown in dogs that Indian ink, when injected into the ventricles, travels from the fourth ventricle upwards in the subarachnoid space along the sulci, resembling the distribution of pus in meningitis.

**Morbid Anatomy.**—There is an acute lepto-meningitis of the brain and spinal cord. The pus and lymph are abundant at the base of the brain, in the quadrilateral space and on the convexity, along the larger blood vessels and in the fissures. In the spinal cord the posterior surface is more affected than the anterior, and the lumbar region more than the other parts. The ventricles of the brain contain turbid serum or pus. In the early stages they are reduced in size, owing to œdema of the brain substance. Punctiform hæmorrhages, accu-

mulations of leucocytes, or actual abscesses are found in the cortex of the brain. Other changes found are congestion of the lungs, liver, spleen, and kidneys, fatty degeneration of the renal epithelium, and granular degeneration of the voluntary muscular fibres, sometimes ecchymosis of the pericardium and pleura and suppuration of the joints. In fulminant cases there is often hæmorrhage into the adrenals. In cases of long duration pronounced hydrocephalus is present, and the pia-arachnoid has an opaque appearance, as if flour had been dusted on its surface. This is due to increased connective tissue formation, the result of the previous inflammation. The hydrocephalus is due to the sealing up of the outlets of the cerebro-spinal fluid round the base of the brain (foramina of Majendie and Luschka), because during the acute stage pressure is exerted at this spot by the swollen brain being forced down into the rigid foramen magnum. In infants suppurative meningitis is sometimes found on the vertex of the brain as well as at the base, while at other times the vertex is free and the base and spinal cord alone affected. It is this latter condition that has caused the disease to be known as "posterior basic meningitis."

**Symptoms and Course.**—The incubation period may be as short as one or two days, but it may also be much longer. The disease itself is extremely variable, so that many different clinical forms have been described. There are, however, two chief phases: (1) the general blood infection; (2) the localisation in the meninges, which usually follow one another in this order. The following is Rolleston's classification:—

1. The *fulminating* type; this is usually fatal within forty-eight hours. It may begin with maniacal symptoms, or the patient may suddenly become unconscious. Again, from the outset there may be collapse, vomiting, and severe headache, and sometimes diarrhœa and a purpuric eruption with large hæmorrhagic areas. There may be no meningitic symptoms at all, the patient remaining sensible to the end. In other cases coma rapidly comes on.

2. The *acute* form begins suddenly like an attack of influenza with headache, fever, vomiting, and, especially in children, with convulsions. Meningitic symptoms appear within a few hours; there is stiffness of the muscles of the back of the neck, which may be shown by attempting to bend the head forwards, and often the head is drawn back by the contraction of the deep muscles; the dorsal and lumbar muscles may be similarly affected, so that the back is kept straight (*orthotonus*), or even arched with the concavity backwards (*opisthotonus*); and sometimes the legs and arms are flexed in tonic spasm. Pains frequently extend down into the muscles of the lower extremities, and cutaneous hyperæsthesia may be also present. The knee jerks are often active, but may be absent; Kernig's sign is practically constant, but Babinski's sign, often limited to one side, is less often observed (*9*)[1] (*see* pp. 732, 734). In addition to these symptoms, referable to irritation of the roots of the spinal nerves, there are others due to the implication of the cranial nerves. These may be, in different cases, ptosis (*3·6*) or strabismus (*25*); usually dilatation, or inequality of the pupils; or contraction of the facial muscles; but trismus is rare. Optic neuritis (*10*), and conjunctivitis (*5·6*), and panophthalmitis (*1·4*) occur. Pain in the ear, tinnitus and defective hearing are not uncommon, and suppuration of the labyrinth or of the tympanum may occur. Deficiency of the sense of smell has been noted. Drowsiness, delirium, and coma, sometimes with Cheyne-Stokes breathing, or convulsions, supervene in due course; and death takes place with varying rapidity in different cases. Fever is present from the first, but it runs no regular course; it is remittent or intermittent, perhaps normal for a day or two, and then rising to 102° or 103°, seldom exceeding 104°. Occasionally the fatal termination is preceded by a temperature of 108° or 109°. With recovery the temperature falls slowly and irregularly. The pulse is variable.

[1] The bracketed numbers in italics indicate roughly the percentage of cases in which the particular symptom occurs. These percentages vary greatly in different epidemics.

An important feature of epidemic meningitis is the occurrence of cutaneous eruptions; the earliest to appear are rose spots, papules, or petechiæ, which are due to emboli in the septicæmic form of the disease; herpes facialis, which is usually regarded as the commonest rash (*50*), occurs about the fourth day; herpes zoster is much rarer. The rashes may be present together. Sometimes the joints are inflamed, hot, red, painful, and swollen, due to synovitis (*10*)—a condition which generally subsides, but may go on to suppuration. The abdomen is often retracted; the spleen is not often enlarged. The urine may contain a little albumin, or a trace of sugar. Epididymitis and orchitis, bronchitis, pneumonia, pleurisy, endocarditis, and latent pericarditis sometimes occur. A leucocytosis of from 15,000 to 60,000 is constant, and is nearly always polymorphonuclear; only occasionally in infants and young children is there a lymphocytosis. If a lumbar puncture be performed (*see* p. 736) the fluid withdrawn may be clear in the early stages, but is usually turbid and may be purulent; it comes out under an increased pressure of from 150 mm. to 500 or 600 mm., and its quantity may reach 20 or 30 c.c. It contains increased quantities of albumin and globulin, and shows in acute stages polymorphonuclear leucocytes, which contain the *meningococcus*. The organisms may also be free in the cerebro-spinal fluid. In chronic cases lymphocytes may sometimes be present in excess.

3. The *abortive* forms are of two kinds: (A) those cases which do not progress further than the septicæmic stage and then get better within twenty-four or forty-eight hours before the meningeal invasion; (B) mild cases with both kinds of symptoms. Relapses or recrudescences are apt to occur in abortive cases.

4. The *chronic* forms: (1) Purely septicæmic, and not often recognised, may last for weeks with periodic rises of temperature resembling malaria, joint pains, orchitis and various skin rashes; sometimes it follows a definite meningitic attack. (2) *Encysted and loculated meningitis*, due to the shutting off by adhesions of some of the space containing cerebro-spinal fluid. This type occurs, particularly in infants, as a sporadic disease, and was known previously as *posterior basic meningitis*. It is distinguished from most adult forms by—(*a*) the chronicity of the disease; (*b*) the rarity of skin eruptions; (*c*) loss of vision quite early in the disease without visible retinal changes or optic neuritis; (*d*) the rarity of deafness: there is often hyperacuity, the child screaming at the slightest noise; (*e*) the prominence of opisthotonus owing to the flexibility of the spine; (*f*) the bulging of the fontanelle from increased intracranial pressure; (*g*) Kernig's sign is rather inconstant at first, but it usually occurs late in the disease. The course is most variable; the child may lie semi-conscious for weeks, at first well nourished, but later it begins to waste. Complete recovery may take place, or hydrocephalus, cerebral diplegia, mental deficiency, or deaf-mutism may result.

**Diagnosis.**—This is not difficult in the course of an epidemic. The characteristic features are the sudden onset, the headache, vomiting, pain in the back and limbs, stiff neck, and the herpes labialis. The purpuric eruption also seems to distinguish it from tuberculous and other forms of suppurative meningitis, *e.g.* pneumococcal, influenzal, streptococcal, etc., which must always be carefully considered. Obscure toxic conditions, such as those arising from some kinds of food poisoning, have been mistaken for the disease. It is desirable in all cases to perform lumbar puncture, and prove the nature of the disease by microscopic examination, or by culture, of the meningococcus. In the later stages of the posterior basal form, the organisms may not be seen in the fluid, and lymphocytes may replace the polymorphonuclear leucocytes. The organism may also be cultivated from the blood in the early stages. Intra-ventricular puncture has also recently been used in diagnosis (**13**). In infants fluid may be obtained through the anterior fontanelle. In adults Goetze's channel borer is used for piercing the cranium on the vertex either 1 c.m. or 4–6 cm. from the middle line.

Many diseases have been at different times confounded with cerebro-spinal fever, especially in early stages, such as pneumonia, influenza, and measles. Acute poliomyelitis, in its so-called meningitic form, may closely resemble it; and the diagnosis may be only possible after a lumbar puncture.

**Prognosis.**—The mortality has varied from 20 to 90 per cent. in different epidemics, but is rarely below 50 per cent. in untreated cases. The disease is most fatal during epidemics among infants, and in the fulminating cases. With efficient serum treatment the mortality has been diminished to 18 per cent. The impression was gained from the last epidemic that, apart from deafness, the patient, if he survived, was not likely to suffer from permanent disability.

**Prevention.**—Certain general measures should be taken, as will be obvious from the ætiology of the disease. In the case of the military and naval forces the treatment of carriers was undertaken from among the persons who were in contact with a sufferer from the disease (so-called *contacts*). The carrier was diagnosed by applying a swab, on a wire, passed up behind the nose, through a glass tube, curved at the end, so as to protect the swab from the mouth and tongue during withdrawal. Any one proved to be a carrier was treated by spraying the nose and throat with an antiseptic, such as pot. permang. 1 in 1,000, or by insufflation of the same into the nose. Carriers were effectually disinfected by the use of a new antiseptic, chloramine-T. A 2 per cent. solution of this was sprayed into a chamber, or inhalatorium, and the carrier was placed in this room for a period not exceeding twenty minutes. He was then found to be free from the micro-organisms, and no longer infective. Spraying with dichloramine-T in oily solutions was also of value. A case has been made out for protective inoculation with a stock vaccine during an epidemic.

**Treatment.**—Apart from general treatment applicable to any acute febrile illness, the specific treatment consists in injecting antimeningococcal serum. It is essential to give the serum corresponding to the particular strain of organism, and when this is not known, as, for instance, at the beginning of the attack, a multivalent serum may be given. The sooner the serum is administered, the better is the prognosis. At the very beginning of the attack in the septicæmic stage doses of 20 to 40 c.c. are injected intravenously until 200 to 600 c.c. have been given in all. In the meningitic stage the serum is given intrathecally. The cerebro-spinal fluid which is under pressure is allowed to run out through the lumbar puncture needle, and the serum should be allowed to run in slowly in less amount than the amount of fluid removed. Twenty to thirty cubic centimetres are given twice daily for three or four days, and the foot of the bed is raised to allow it to run down towards the brain. Lumbar puncture and injection should be carried out under a general anæsthetic or after a hypodermic injection of scopolamine $\frac{1}{100}$ grain, morphine $\frac{1}{6}$ grain, atropine $\frac{1}{100}$ grain. When there is a recrudescence or a relapse the injections must be repeated, but the patient must be desensitised (*see* Anaphylaxis). Irrigation of the theca with normal saline or 3·8 per cent. sodium citrate has been of value in preventing adhesions. Where adhesions have formed and the intracranial contents are shut off, the lateral ventricles may be tapped and serum injected. In children this can be carried out through the anterior fontanelle. In posterior basic meningitis intrathecal injections of serum are also given in suitable doses.

## ACUTE POLIOMYELITIS

(*Acute Anterior Poliomyelitis, Acute Polioencephalitis, Heine-Medin's Disease*)

For years it has been known that children were liable to suffer from paralysis and atrophy of one or more limbs, or part of a limb; and that this paralysis was due to an acute inflammation of the anterior grey cornua of the spinal cord. Such cases were called *infantile paralysis*, and later *acute anterior poliomyelitis* (πολιός,

grey). These cases occurred sporadically, but they were seen to affect two or more members of one family, and sometimes larger groups of cases or epidemics occurred. Within recent years, however, there have been extensive outbreaks, as, for instance, in Norway and Sweden, Holland, Westphalia, New York, and Melbourne, and epidemics have occurred in different counties of England, so that the cases notified have numbered 823, 736, 500 and 408 in the four successive years 1912 to 1915. Although the disease attacks the spinal cord most frequently, it may also attack any other part of the central nervous system, and probably the peripheral nerves. The term *polioencephalitis* has been used for certain of these cases; but it is best avoided, so as to prevent confusion with acute encephalitis, which is closely related, but is a different disease. Acute poliomyelitis is to be regarded as one of the acute specific fevers.

**Ætiology.**—Whether sporadic or epidemic, the disease attacks infants or children with much greater frequency than adolescents or adults; for instance, in a large epidemic two-thirds of the cases were under six years of age, and five-sixths under ten years. It is most frequent in the summer and autumn months. The micro-organisms causing the disease are minute globoid bodies arranged in pairs, measuring 0·15$\mu$ to 0·3$\mu$ in diameter; these are so small that they readily pass through a porcelain filter; they can be cultivated anaerobically on a special medium, and cultures reproduce the disease by inoculation into monkeys, from which animals the organism can be again recovered in pure culture (Flexner and Noguchi, 1913). Washings from the nose, mouth, pharynx, upper air passages, and small intestines of patients when filtered produced the disease in monkeys, and there is evidence that healthy people may act as *carriers*. The disease may also be spread by patients who have the *abortive* form of the disease. There is considerable doubt as to whether the virus is conveyed by flies, dust, or external objects. It is believed to pass to the central nervous system along the lymphatics of the peripheral nerves. Paralyses may occur in domestic animals, particularly during epidemics; but they are probably not due to the same disease.

**Morbid Anatomy.**—The essential lesion is an acute inflammation of the interstitial tissue of the central nervous system. In the spinal cases, which form the majority, the disease involves the grey matter of the spinal cord, but also to a less extent the white matter, and the meninges. The pia mater is œdematous and infiltrated with mononuclear cells; there is œdema of the affected part of the cord, proliferation of cells in the sheaths of the blood vessels both in grey and white matter and in the meninges, and cell infiltration of the substance of the grey matter. The nerve cells, especially those of the anterior cornua, present various degrees of degeneration up to complete disappearance; and they may be seen infiltrated with polymorphonuclear and mononuclear cells (*neuronophagia*). The lesion of the anterior cornua is followed by secondary degeneration of the nerve fibres of the motor roots, and atrophy of the muscles supplied by them. The same acute processes may occur in patches in the medulla, pons, cerebrum, cerebellum, and meninges.

In some fatal cases, in addition to the lesions of the nervous structures, the lymphoid tissues of the small intestine, the thymus and the spleen have been enlarged, and there has been some degeneration of the gland cells of the liver.

In cases dying after years of permanent atrophy of one or more limbs, the cord presents changes obvious to the naked eye. The motor nerve roots, coming from the part presumably affected, are diminished in size and number. On a transverse section the cord is smaller on the affected side, and the anterior cornu is shrunken. Under the microscope there is an almost entire absence of motor nerve cells and axis cylinders; the few nerve cells that remain are smaller than normal, shrunken, fusiform, and wanting in processes, and lie in a dense felt-like overgrowth of neuroglia. The motor nerve roots, both in and beyond the cord, show the destruction of axis cylinders, and are obviously degenerated.

The muscles in physiological connection with the damaged parts undergo

fibrous or fatty degeneration, which may be partial or complete. They are pale pink, watery in appearance, and present under the microscope the changes described as the result of lesions of motor nerves (*see* p. 735).

**Symptoms.**—For the sake of convenience, a number of clinical forms of the disease have been described depending on the part of the central nervous system attacked. It must be remembered that individual cases are often a mixture of two or more forms. Certain *general symptoms* common to all forms may first be described. The period of *incubation* is from four to twelve days. The onset is rapid: there are feverishness with headache, and drowsiness, and severe pains in one or more limbs, suggesting acute rheumatism. The pain is increased on movement. Convulsions, vomiting and diarrhœa sometimes occur. While drowsy the patient may pass urine and fæces involuntarily. There may be a diffuse erythema, or a vesicular rash, and occasionally herpes zoster. Tingling or formication may accompany the pain in the early stages, but there is never any considerable loss of sensation, and the above symptoms pass away in a few days.

The cerebro-spinal fluid in about 80 per cent. of the cases shows an increased cell count in the first week of the disease; in the earliest stages this may consist largely of polymorphs, but after the first two days lymphocytes predominate, and at the end of the first week polymorphs are seldom found. By the end of the second week the cell count is usually normal. The protein almost always shows a slight increase; this occurs in the second week of the illness, and often persists after the cell count has returned to normal. The sugar and chlorides are not diminished.

1. *Spinal* Form.—This accounts for about three quarters of the cases. It usually shows itself as a paralysis of the limb muscles; but the muscles of the trunk, abdomen, and neck are also sometimes involved.

In the course of twenty-four or forty-eight hours it is found that there is weakness or definite paralysis of certain muscles, or a child may go to bed well and be found to be paralysed in the morning. It frequently happens that three or four limbs are paralysed at first, and recovery quickly takes place in two or three, leaving the others permanently affected; in other cases, certain limbs are affected from the first and remain so. The paralysis, however, need not involve the whole of a limb, but, it may be, only a part of it, or even one muscle group. If paralysis affects both legs, or the arm and leg on one side, it is not distributed uniformly, as in some other forms of paralysis. The affected muscles rapidly undergo atrophy, lose bulk, and become flaccid; when tested electrically some days after the onset they show the reaction of degeneration (*see* p. 733) or in severe cases do not respond at all to either current. All the deep reflexes are lost in the most affected parts, and generally the superficial reflexes as well.

Although the disease in most cases attacks principally the grey matter of the spinal cord, a complete transverse lesion is occasionally seen; in the cervical region such a case shows loss of power and wasting of the muscles of the hands and arms and spastic paralysis of the legs. Mention must also be made of certain "jump" cases. The disease may, for instance, involve the legs and remain stationary for some days, when a more widespread paralysis affecting the muscles of the trunk or upper limbs suddenly takes place, or this extension may take place gradually. It may pass downwards along the cord as well as upwards. Some of these cases closely resemble Landry's paralysis. In addition, *relapses* may take place after some weeks' interval.

At the end of the febrile period the muscles which remain paralysed after the first partial recovery will themselves improve only very slowly after weeks and months. The amount of impairment in the use of the limbs will depend on the number of muscles atrophied; but after a time, in many cases, lost movements are restored by fresh combinations among the muscles which have been spared. Atrophy is, in almost all cases, a prominent feature, hollowing out the rounded part of the forearm, or reducing the upper arm or the leg to a mere stick. Sometimes,

however, the loss of muscle may be entirely concealed by the presence of fat; the flabby condition of the muscle even then can be generally recognised. Associated with the atrophic condition of the muscles is generally a change in the vascularity of the limb; it is cold, shrunken, and blue or livid from retarded circulation. The nutrition of the bones and other parts is also involved, so that a limb paralysed in infancy or early childhood does not grow with the same rapidity as its fellow, and may be shorter by ½ inch, 1 inch, or more. Lastly, deformities may occur besides those directly due to loss of muscular substance, owing to faulty treatment. Some are the simple result of failing muscular support; thus, from atrophy of the deltoid, the humerus falls from the glenoid cavity. Others consist of permanent changes in the position of the limbs, such as talipes equinus, which so often results from paralysis of the anterior tibial muscles.

2. *Bulbar, Pontine, and Mid-brain* Forms.—Any of the cranial nerve nuclei may be attacked as well as the tracts passing through this part of the brain (*see* p. 703). The seventh nerve is the one most commonly affected. However, if a case were seen with only a seventh nerve paralysis it would certainly be due to Bell's palsy, and not acute poliomyelitis.

3. *Cerebral* Form.—Hemiplegia is the commonest manifestation of this form of the disease; but it is not a common cause of hemiplegia in children, accounting for at most 10 per cent. of the cases. Athetosis is rare.

4. *Cerebellar* Form is indicated by the occurrence of cerebellar ataxy (*see* p. 839).

5. *Meningitic* Form.—Symptoms of meningitis are not at all uncommon in acute poliomyelitis. After severe headache the patient may become comatose with rigidity of the muscles at the back of the neck, with convulsions, and with typical changes in the cerebro-spinal fluid.

6. *The Abortive* Form is only recognised where there are epidemics of acute poliomyelitis. There are fever, headache, pains in the limbs, and general weakness, but no paralysis.

**Diagnosis.**—The symptoms of fever, headache, vomiting, convulsions and pain may be caused by many acute illnesses, such as meningitis or pneumonia. The muscular power, the loss of which is the distinctive feature, should be critically examined from the first, in view of the fact that young children will not volunteer the information. After some days the diagnosis is confirmed by the rapid atrophy, by the loss of reaction to the faradic current, and the changed reaction to galvanism. The pain which is sometimes present may suggest *rheumatism*, but it is situate rather in bone and muscle than in the joints; in infants *scurvy* must be thought of. The disease may also be confounded with *Landry's paralysis*, with *multiple neuritis*, due to some other organism, to alcohol or other poison, or with acute myelitis, due to some other infection. Pain and tenderness are not usual in Landry's paralysis, but some cases described as Landry's paralysis have certainly been actually poliomyelitis. In the early stage, and in abortive cases, the diagnosis may often be established by examination of the cerebro-spinal fluid (*see* p. 98). The normal chloride content in poliomyelitis is a useful point in the differential diagnosis from tuberculous meningitis. A method of *serum diagnosis* is also available. This depends on the fact that a 5 per cent. emulsion of spinal cord containing the virus will cause the disease in monkeys if injected intracerebrally; but if mixed with an equal volume of serum from a patient who has recovered, it will be inert. By such means it would be possible to find out definitely whether certain suspected cases had really had the disease, which would be valuable from the public health point of view.

**Prognosis.**—In recent epidemics of this disease the mortality has varied from 10 to 20 per cent., a much higher mortality than was recognised in sporadic cases, in which life was not generally regarded as endangered. On the other hand, complete recoveries appear to be more frequent in epidemics. Death is due generally to respiratory paralysis or to broncho-pneumonia if the respiratory muscles are interfered with; it rarely occurs if the fourteenth day is safely

reached. Not more than about 15 per cent. of cases show complete recovery. In the remainder most of the muscles originally paralysed eventually recover, but one or two groups remain permanently damaged. Most improvement takes place during the first six months, and it continues slowly for two years. After this time very little further improvement is likely to occur. Second attacks are extremely rare.

**Prevention.**—Patients and contacts should have their nasal and buccal cavities frequently washed out with a solution of potassium permanganate of 1 to 2,000, to destroy the virus. The period of quarantine for contacts is fourteen days. The patient should be isolated for at least three weeks, and as long as there is any nasal discharge he should still be considered infectious. He may, like a case of typhoid, be treated in a general hospital ward.

**Treatment.**—During the acute stage of the disease, the patient must remain at rest in bed and immobilised as far as possible by means of splints or other form of retentive apparatus, *i.e.* plaster of Paris, etc. Other requisites are a nutritious, easily digested diet, aspirin or sodium salicylate at least in older children to relieve pain, or even morphia in extreme cases, in doses appropriate to the age. Hexamine may be given in full doses, as being an antiseptic which is secreted into the cerebro-spinal fluid; but estimates as to its value vary. But more active measures have been advised, *e.g.* lumbar puncture, which relieves pressure, and with this the intraspinal injection of adrenalin chloride solution (1 to 1,000) in doses of 1 or 2 c.c. every six hours, or the intraspinal injection of an immune serum, that is, serum obtained from the blood of persons who have suffered from the disease months or years previously, in doses of from 10 to 30 c.c. daily (Netter). The cases suitable for this treatment are (1) those in which the diagnosis can be made in the pre-paralytic stage; (2) those with symptoms of meningitis; (3) those in which the paralysis is extending. The treatment is useless for muscles which already show paralysis. In four to six weeks' time, after the pain and tenderness have disappeared and some of the muscles are regaining their power, more freedom of movement may be allowed. The patient may begin to sit up and move the limbs.

The *immediate* treatment of the affected muscles consists in *rest*, in such a position as will prevent the usually occurring deformities. This position may be maintained by celluloid splints, the splint being moulded on an accurate cast of the limb. The correct position of the upper limb is with the thumb adducted, the fingers slightly flexed, the wrist dorsiflexed and supinated, the elbow slightly flexed and the shoulder abducted from the side at a right angle. The lower limb should be placed with the foot at right angles to the leg and the knee and the hip straight. When one leg is affected the other one should also be kept in splints to prevent asymmetry of the pelvis. If both legs and trunk muscles are paralysed, some degree of lordosis should be brought about by supporting the back in the lumbar region, as in this position the patient could eventually walk with the aid of retentive apparatus. In three or four weeks' time it will become obvious what special muscle groups are paralysed, and then the position of the limbs and body should be altered, so as to allow complete relaxation of the paralysed muscles, with a consequent overstretching of the healthy antagonists.

*Re-education* of paralysed muscles should begin as soon as the temperature has subsided and the pain has gone. This consists in getting the patient to use the muscles at stated intervals each day; the effect of gravity should be diminished or abolished, and the amount of work the muscle is called upon to do should be minimal to begin with. For instance, with a weakened quadriceps the knee is at first flexed to a small extent with the patient lying on his back, and he is told to straighten it. The amount of flexion is gradually increased. It is valuable sometimes to carry out these movements in a bath, as the water gives some support. Between the exercises the splints must be kept on. The re-education of the trunk and abdominal muscles can be best carried out by

the patient walking about in the erect position. This can be done when the patient's paralysed legs have been splinted, if he uses a walking machine. Massage, electricity, and baths may also be useful as subsidiary measures. Later, surgical measures may be required.

It must be recognised that if nerve cells are completely destroyed the muscular fibres dependent upon them will be irretrievably atrophied.

## HYDROPHOBIA

(*Rabies, Lyssa*)

This is an infectious disease, which is invariably caught from animals, usually as the result of a bite. Any warm-blooded animal is capable of contracting the disease, but in the British Isles dogs are by far the most important infecting agents. In India wolves, jackals and monkeys are also important. The disease in animals is called rabies, in man hydrophobia; but, in spite of difference of nomenclature, it is the same disease. Rabies in animals is a fatal disease: it occurs in two forms. In the first, or *furious rabies*, the dog is to begin with low-spirited, timorous, and unwilling to move; he then becomes suspicious and irritable, with a strong tendency to bite, and often with a peculiar howl. He refuses his ordinary food, and will eat straw, earth, hair, clothes, bits of wood, etc. Paralysis supervenes, the lower jaw drooping, the limbs failing, so that the animal can no longer stand, and finally death takes place with asphyxia. In *dumb rabies* there is no maniacal stage; the paralytic symptoms appear early, and the dog may be unable to swallow.

**Ætiology.**—The virus of hydrophobia is unknown; it is contained in the saliva, and may be present there for a week before symptoms of rabies develop. Man is infected from the saliva through a bite. Infection may also occur if the dog licks an abraded surface or a healthy mucous membrane. In only about half the cases of bite by mad dogs does hydrophobia afterwards develop, and it is more likely to be the case if the bite is on an exposed part, such as the face or hand. A portion of clothes driven in by the tooth may protect from infection. By inoculation experiments the virus is shown to be distributed throughout the central nervous system, and in the secretion of certain glands (salivary, lachrymal, mammary, and pancreas), but not in the blood. It has also been shown that the virus after inoculation spreads up the peripheral nerves to the central nervous system.

**Morbid Anatomy.**—There are but few macroscopic changes in the organs; the cerebro-spinal fluid is often increased in quantity. The microscopic changes found in the nervous system, especially in the cortex of the brain, in the spinal cord, and most abundantly in the medulla oblongata, consist of dilatation of vessels, collections of small cells round the vessels and in the tissues, clots in the vessels, and small hæmorrhages. Leucocytic infiltration has been also seen in the salivary glands and in the kidneys. In 1903 Negri found certain bodies in the nervous system of animals dying of hydrophobia, and these *Negri bodies* have since been found with great constancy by others. They are seen in the nerve cells of certain parts of the brain, especially the hippocampus major. They appear to be essential to the disease.

**Symptoms.**—It takes a fortnight before symptoms appear in an animal which has had the virus implanted on the surface of its brain. In man the virus has to travel up from the wound in addition, so that the *incubation period* must anyhow be longer than a fortnight. It is usually between one and three months, and rarely more than six months. The more severe the wound and the nearer it is situated to the brain, the shorter is the incubation period. The most characteristic early symptoms are a rise of temperature, a feeling of unrest, an appearance of excitement, and inability to swallow solids or fluids. Patients

may complain of this last symptom while they are still feeling quite well. The first definite sign is often an uneasy sensation of pain in the scar of the wound. This pain may be very severe, and the scar may be slightly reddened or tender. But these indications may be entirely absent. Then appear the spasms which are so characteristic of the disease; they are excited by the attempt to drink, by the sight of water or the vessel containing it, or by the suggestion of those around that some fluid should be taken. Later on they are induced by almost any external impression—a breath of air, a flash of light, or a loud noise. The spasms involve the muscles of deglutition, but the most obvious are those affecting the muscles of respiration—a sudden deep inspiration, like a sob or sigh, is made; the shoulders are raised, the chest expanded, and the sterno-mastoids or platysmas contracted. If water is forced upon the patient, more voluntary efforts to reject it are made, and an aspect of fright or terror is assumed. After a time the contractions extend to other muscles of the body, resembling tetanus. The difficulty of deglutition is shown in another way, for the saliva is not swallowed, but is constantly being collected in white frothy pellets, and is expectorated in all directions. With the increasing severity of these spasms, the patient becomes excitable, talkative, delirious, or wildly maniacal, with delusions and hallucinations. The temperature is raised; the face is flushed; all attempts to give food may be futile—at most a small quantity of milk or other nutriment may be gulped down in a moment of greater control. Emaciation is remarkably rapid in the short time the disease lasts, and exhaustion necessarily follows. Not infrequently, towards the end, the spasms cease entirely, and the patient may even take good quantities of food; but even if this is so, it does not avail to prevent the fatal end, which may be preceded by paralysis and coma. In a few cases, the final symptoms are paralytic; motion, sensation, and reflexes are lost in the legs, arms, and other parts.

The disease lasts from two to four days; a period of ten days seems to be the longest known. Death is inevitable in the developed disease.

**Diagnosis.**—This is not generally difficult, especially if the fact of infection is well authenticated. There is but little real resemblance to tetanus, in which the permanent rigidity of muscle and the absence of mental disturbance are distinctive. Hysterical conditions may simulate hydrophobia, and may occur where the mind has been much directed to the possibility of hydrophobia coming on. They are called "spurious hydrophobia" or *lyssaphobia*.

The presence of the disease in any animal inflicting a bite can be shown by microscopical examination of sections and smears from its hippocampus major, and by the inoculation under the dura mater of a rabbit of an emulsion of the medulla oblongata.

**Prognosis.**—The mortality is under 0·5 per cent. if antirabic treatment is used, provided the case is treated in time.

**Prevention.**—The prevention of rabies in dogs in England is carried out—(1) by the muzzling order; (2) by restricting the movements of dogs into or from an infected area; (3) by a strict quarantine for all imported dogs.

In a *rabies-infected area* all bites from dogs should receive the local treatment detailed below. The dog should not be at once destroyed, but should be kept to see if the disease develops. If this does not happen, inoculation need not be carried out. But if a person is severely bitten on the face, head or neck, inoculation should be carried out at once; it is unsafe to wait; the same applies to less serious bites, if the dog has disappeared.

**Treatment.**—No remedy is known to have any influence upon the disease when once it is developed. Temporary relief may be given by morphia injections or chloroform inhalations.

*Local Treatment.*—The wound must be cauterised: for this purpose it is best to swab it repeatedly with undiluted carbolic acid; it may also be excised. If no antiseptic is available, washing with water is better than nothing at all.

The wound may also be sucked, but there is a little danger to the person doing this.

*Antirabic Vaccine Treatment.*—This must always be used after a bite by a rabid animal. The principle of the treatment is to immunise the patient against the disease before the virus implanted in the wound reaches the nerve centres. In Pasteur's *dry cord method* a rabbit is trephined and inoculated under the dura mater from the spinal cord of a rabid dog; the rabbit becomes rabid after fifteen days' incubation. A second rabbit is inoculated from the first, a third from the second, and so on until the period of incubation, which grows shorter with successive inoculations, is reduced to the minimum of seven days. The spinal cords of these rabbits contain the *virus fixé*, and the virulence is at a maximum. For the purposes of preventive inoculation a number of fragments of these virulent spinal cords are kept in separate bottles of dry air for periods varying from three to fourteen days. At the end of three days the virulence is unaltered, but with every succeeding day it becomes attenuated, until it has quite disappeared by fourteen days. Patients are inoculated subcutaneously with 1 cm. of cord ground up in 3 c.c. of sterile normal saline, the cord used being of gradually increasing virulence. This treatment extends over twenty-one days; but it may be *intensified* in serious cases, the course lasting only for a fortnight. The most recent method of treatment is by means of a *dead rabies vaccine* (Semple). This is prepared from the rabbit's brain, medulla, and spinal cord, containing the *virus fixé*, by diluting it with normal saline containing 1 per cent. carbolic acid and keeping it at 37° C. for twenty-four hours. After further diluting with the same volume of saline it is ready for use, and the immunising properties remain unimpaired for at least three months. It can be sent by post, and so is readily available for treatment.

Antirabic sera have also been used, but only in conjunction with vaccine treatment, so that it is impossible to judge of their efficacy.

## TETANUS

In this disease, of which the name is derived from τείνω, I stretch, the essential condition is the occurrence of tonic contractions of most of the muscles of the body, with paroxysms of increased contraction from time to time. It is due to a bacillus (*B. tetani*), which exists in different forms of earth or garden mould, and which will cause tetanus in animals when such earth is inoculated under the skin.

The bacillus measures 4$\mu$ to 5$\mu$ in length, and 0·4$\mu$ in thickness, is flagellated, and stains with the usual dyes and with Gram's method. It produces spores which are developed at one end, and, having a diameter larger than that of the bacillus, give it the appearance of a drumstick. It grows anaerobically.

**Ætiology.**—The disease occurs in quite young infants (*tetanus neonatorum*), and after that age at all periods of life from five years upwards. It is more common in hot countries, and the dark-skinned races seem especially liable to it. A very frequent antecedent is injury (*traumatic tetanus*), by which an entrance is provided for the bacillus. This may be of any kind, from a mere scratch with a pin or nail to the most serious compound fracture or lacerated wound; but infection is especially liable to take place when the wound has been contaminated by contact with earth, dirt from the road, garden mould, stable straw, or similar materials. In fact, a suspension of washed tetanus bacilli or their spores can be injected into animals without harm; but if calcium chloride or hydrated soluble silica, both of them common constituents of soil, be injected at the same time, the organisms grow and produce the disease. Tetanus has followed the subcutaneous injection of gelatin for aneurysms and of quinine for malaria. In new-born infants the organism enters by the cut surface of the umbilical cord. Probably, in all cases formerly called *idiopathic*, some means of

local infection was overlooked. For instance, a stableman with otorrhœa acquired tetanus, no doubt, because he infected the meatus and tympanum with his finger soiled with stable dirt. The disease is sometimes epidemic.

**Morbid Anatomy.**—Many cases present after death no pathological lesions whatever. The organs most commonly affected are the lungs, which may be the seat of pneumonia, bronchitis, œdema, or hæmorrhages. The central nervous system, as a rule, looks normal to the naked eye, or at most shows some hyperæmia of the grey matter. Microscopical examination may also show slight degenerative changes in the nerve cells. Both these conditions are referable to the action of toxins, or to the vascular disturbance during the spasms. The muscles of the trunk, especially the abdominal muscles, are sometimes ruptured or the seat of hæmorrhages. In traumatic cases the state of the wound bears no relation to the final result—it may be healing, or healed, or suppurating, or sloughing.

**Pathology.**—The bacillus multiplies chiefly in the neighbourhood of the wound, and produces poisons which have an affinity for the central nervous system, especially the spinal cord. They have, however, been found in the lymphatic glands.

Experiments on animals also show that such toxins are absorbed into the blood; but they are also taken up by the end plates of the nerves in the muscles, and are transmitted by the axis cylinders of motor nerve fibres, or by the lymphatic vessels accompanying them, to the cells of the corresponding anterior cornu; and this appears to be the chief means by which the nerve centres are infected. If the toxins are sufficient, they are carried to the opposite cornu, and to other parts of the cord. Nevertheless in unprotected man the muscles first attacked by spasms are not generally determined by the seat of injury, but are usually those of the back of the neck and of the jaw. Tetanus has also been transmitted from man to animals by the inoculation of materials from the wound, and by the injection of urine which contains the toxin.

**Symptoms.**—There are two distinct clinical types of tetanus: (1) the classical type of "pre-serum" days, characterised by trismus being the initial symptom; (2) modified tetanus, a discovery of the recent war, which may occur in patients who have had a prophylactic injection of anti-tetanus serum.

*The Classical or Generalised Type.*—The period of *incubation* between the infliction of the wound and the onset of symptoms is from one to thirty days, about half the total number of cases developing during the second week and one-third during the first week. It is found that the incubation period is delayed in prophylactically immunised patients who contract this type of disease. The patient feels stiffness first in the jaws, so that he is unable to open his mouth wide, or to masticate properly. There is also stiffness at the back of the neck. He may continue like this for a day or two, or may rapidly pass on to the next stage, in which there is rigidity of the muscles of the trunk and to a less extent of those of the extremities. The back becomes rigid, and is slightly arched, with the concavity backwards (*opisthotonus*); the muscles of the trunk and abdomen become quite hard from constant contraction; the movements of the chest are limited from the same cause; the legs are generally rigid, but the arms are only rather stiff about the shoulders and elbows, and the fingers may be moved freely. By this time the jaw is generally firmly fixed by contraction of the masseters, and the teeth cannot be separated for more than ¼ inch (*trismus*, or lockjaw, by which last name the disease itself is popularly known); the angles of the mouth are drawn outwards, and the lips are slightly separated; the eyebrows are drawn up by the frontal muscles, and together by the corrugators, so that the facial expression is that of a painful grin, known as the *risus sardonicus*. When this stage has been reached, the so-called "spasms" or paroxysms of increased and even violent muscular action begin. These consist of sudden contractions involving the whole of the muscles hitherto in tonic rigidity. The teeth are clenched more

violently, the *risus* becomes more marked, the head is thrown back and the back arched more strongly, the chest is fixed, and the respiratory process is checked; a groan may escape from the patient, either elicited by pain or the result of expiratory spasm. The paroxysm is often of momentary duration, scarcely to be counted in seconds, and the patient relapses into his former condition of tonic contraction, or it may last several seconds, the face and hands become more and more livid and swollen, and there is imminent danger to life from the hindrance of respiration. It is always intensely painful; it is brought on by external impulses, by touching the patient, jerking his bed, by passing a catheter or giving a subcutaneous injection. The paroxysms occur at first at intervals of half an hour, an hour, or more, but as the disease progresses unfavourably they become more violent, and occur at shorter and shorter intervals. Between the spasms there is still some pain from tonic contraction, respiration is not entirely free, and the voice is feeble. The reflexes are increased. The pulse is small and quick, and becomes quicker during the paroxysms. The temperature generally at first remains normal, and may continue so to the end, though it sometimes rises a little before death; sometimes the temperature is constantly above normal; in other cases a hyperpyrexia of 107° or 108° occurs just before death, and the temperature has been observed to continue rising even after death to 110°. The urine is often retained, so as to require the use of the catheter. Sensation is generally unaffected, and the cerebral functions are mostly quite normal until near the end, when delirium may occur. In a great number of cases the disease progresses to a fatal termination in from one to twelve days: the paroxysms become more violent and frequent; and death takes place from exhaustion, or from spasm of the glottis, or from fixation of the respiratory muscles; or pneumonia or bronchitis adds its influence against the patient. As happens both in fatal chorea and in hydrophobia, the muscular contractions sometimes entirely cease for eighteen or twenty-four hours before death. In a few cases life is prolonged to the third or fourth week. On the other hand, recovery may take place: the spasms, having reached their height, gradually become less frequent; the constant rigidity of the muscles subsides, and the patient is convalescent in from three to six or eight weeks. Occasionally a case runs its whole course to a fatal termination with general rigidity, but without any paroxysms in addition; and very rarely there are paroxysms without the continuous spasm.

*Modified or Local Tetanus.* In this form the incubation period is lengthened, being over three weeks in about half the cases. The disease is limited to the muscles which are close to the wound. Thus one arm may be affected, or one or both legs, or the abdominal muscles when the wound is in the abdomen. The affected muscles may show tonic or clonic spasms, or there may be rigidity and hardness of the muscles, with inability to move them. The contractions may be extremely painful. Local tetanus may spread after some days. Thus if one limb is affected the disease may later on involve the other one. It may also become generalised, causing trismus and reproducing the classical form of the disease.

Some special types of local tetanus are described. Thus the name *splanchnic* tetanus has been given to cases which arise after lesions of the viscera, such as penetrating wounds of the abdomen or thorax. It is nearly always rapidly fatal: the spasms are confined to the muscles of deglutition and respiration; and the difficulty of swallowing may be so great that the case closely resembles one of hydrophobia in the liability to pharyngeal spasms at the sight, or even mention, of a glass of water. This is usually accompanied by dyspnœa.

The name *cephalic* tetanus is given to cases which arise from injuries to the head or face. Of this variety four forms are described: (*a*) one in which there is no paralysis, but in which dysphagia and dyspnœa may be present, and the dysphagia may reach such a degree as to resemble closely hydrophobia; (*b*) another, ophthalmic tetanus, in which the oculomotor nerve is involved,

causing ptosis, or paralysis of the ocular muscles, extrinsic or intrinsic; (*c*) a third rare form, in which the hypoglossal nerve is concerned; and (*d*) the fourth, in which the facial nerve is affected, facial paralysis occurs, and spasms take place in the paralysed muscles.

**Diagnosis.**—Tetanus may have to be distinguished from strychnine poisoning, hydrophobia, spinal meningitis, tetany, muscular rheumatism, and hysteria. In *strychnine poisoning* the extremities are involved to a much greater extent than in tetanus, and the paroxysms are excited by external stimuli; but in the intervals the muscles are relaxed. The symptoms develop very rapidly, but do not begin with trismus. In *hydrophobia* there is no continuous rigidity; the spasms involve the respiratory muscles, and are excited by the attempt to drink or the sight of fluids. Mental agitation or even maniacal excitement is generally present. In *spinal meningitis*, again, trismus is not an early symptom, nor is there constant rigidity; muscular spasms are excited by attempts to move, and the temperature is high from the first. The early occurrence of cerebral symptoms would be opposed to tetanus. The peculiar distribution of the spasm in *tetany* makes it easy to distinguish it from tetanus. *Muscular rheumatism* may cause stiffness of the back of the neck, which might, under certain circumstances, cause alarm; but trismus is never present. In severe forms of *hysteria* opisthotonus is often a prominent feature, but it occurs as part of a series of convulsive movements, which cannot be mistaken. The local form of tetanus affecting one limb must be distinguished from a hysterical paralysi

**Prognosis.**—The shorter the incubation period the greater the mortality. In pre-serum days the mortality was between 80 and 90 per cent. of all cases, as judged by several independent series of observations. During the late war the average mortality of all the English cases was 50·8 per cent., this reduced rate being accompanied by a considerable increase in the incubation period. This was due to the introduction of prophylactic serum injections. Analysis of the whole series of cases indicated that there was no evidence that curative serum injections had any effect in reducing the mortality (Golla). The mortality of purely local tetanus was practically *nil*.

**Prevention.**—All wounds should be promptly excised or cleansed, especially when contaminated with mud or earth; antiseptics, even strong carbolic acid, may be usefully employed to diminish the chance of infection. In warfare and in other conditions when tetanus is rife, 500 units of tetanus antitoxin should be injected subcutaneously as early as possible: and as the protection conferred by this dose seems not to last more than ten days, a second injection should be given after seven days in cases of septic wounds, and in exceptional cases a third or even fourth at the same interval. The antitoxin has been applied locally to wounds in similar circumstances. In the event of secondary operations having to be performed, Leishman and Smallman recommend injections of antitoxin around the site of the operation, if possible, forty-eight hours before the operation, and to the extent of about 1,500 units. They suggest also injection into the sheaths of the prominent nerves.

**Treatment.**—The patient should be kept at rest, and is best placed in a darkened and perfectly quiet room, so as to avoid all impressions of sight and sound. Nourishment should be given freely, in fluid form; but the closure of the jaws may necessitate its being given by a nasal tube. If a wound is present, it should not be actively interfered with until the tetanic symptoms have subsided, as fresh toxin may be liberated.

The curative action of serum has been much discussed. Statistics suggest that serum is not of much use; but these are not very satisfactory, as the matter is complicated by the very favourable influence prophylactic injection has upon the mortality. Experimental evidence points strongly to the fact that serum may do good if given early, and further that the correct route is by the spinal canal. Thus Sherrington found that there were fourteen recoveries out of twenty-

five monkeys which had been injected with tetanus toxin and received an intrathecal injection of antitoxin forty-seven to seventy-eight hours later, *i.e.* after the first symptoms of tetanus had shown themselves. When the same dose of antitoxin was given subcutaneously the recovery rate was two out of twenty-five; when given intramuscularly it was three out of twenty-five; when given intravenously it was seven out of twenty-five; when given subdurally through the skull it was none out of ten. On the other hand, when it was given intrathecally through the atlanto-occipital membrane the recovery rate was thirteen out of twenty. In view of the uncertainty attaching to the results of serum treatment in the war, it will be safest to take these experiments as the guide to treatment. They point to the importance of intrathecal injection of serum in the first place, and of intravenous injection in the second place. These must be carried out at the earliest possible moment, and a general anæsthetic must be given. The serum should be allowed to run slowly into the theca under gravity through a funnel after withdrawing some cerebro-spinal fluid; at least 10,000 units should be given, if possible. In a few cases 40,000 units have been injected when the serum has been of very high potency. At least 3,000 units should be given intravenously. The question of anaphylaxis must be considered, but the risks are much less if a general anæsthetic is used. Intrathecal injections of magnesium sulphate (1 c.c. of a 25 per cent. solution for every 10 kilogrammes of body weight) have also been used. The contractions may be abolished, but it is doubtful if the injections reduce the mortality.

If necessary, the patient should receive sedatives, *i.e.* morphia hypodermically in ¼-grain doses four-hourly; chloral hydrate, 30 to 60 grains by mouth; chloretone, 10 to 15 grains by mouth or 30 to 40 grains dissolved in olive oil *per rectum*.

If respiration ceases owing to spasm of the respiratory muscles, intratracheal insufflation with oxygen may be tried.

## GAS GANGRENE

This is an infection of the tissues by certain gas-forming anaerobic bacteria.

**Ætiology.**—Gas gangrene was common in the pre-antiseptic days. It became very rare in hospitals after antisepsis and asepsis were firmly established, but was met with again frequently during the recent war, where the bacteria which are often present in dirt gained access to the tissues through gunshot and, particularly, shell wounds. Several different bacteria are found, but the most important are *Bacillus perfringens*, which is the same as the *B. aerogenes capsulatus* of Welch, *B. œdematiens* and *Vibrion septique*.

**Pathology.**—The primary infection takes place in the muscles, which are changed from the normal purple to a brick-red colour in the early stages. They become very friable, and the gas can be pushed about from place to place between the fibres. Later on the colour becomes olive-green and the consistence like putty (Wallace). The bacteria grow on the surface of the muscle fibres, destroying them, and the gas accumulates between the fibre and the sarcolemma or sheath. The infection spreads chiefly up and down the fibres, and a single muscle may be affected at first. At a later stage it also spreads to the neighbouring muscles and subcutaneous tissues, and, by metastases, to other parts of the body. A low antitryptic power in the blood favours the growth of these organisms (Wright), and they produce acid, which causes a lowering of the alkaline reserve of the blood. Growth only occurs when the organisms gain access to damaged tissues.

**Symptoms.**—In the early stages the skin looks normal. Then the limb swells and the skin becomes tense. As gas accumulates a resonant note is obtained on percussion and there are crepitations. Areas of purple staining appear and

coalesce, and there are blebs containing fluid, stained by altered blood. Finally, the purple gives place to a dark yellow-green tint.

**Diagnosis.**—X-rays provide the earliest means of diagnosis. Clear areas corresponding to collections of gas are seen in radiograms. They tend to be arranged along the muscle fibres.

**Prognosis.**—This is practically hopeless, unless early energetic treatment is adopted.

**Prevention.**—This consists in the excision of contaminated wounds, and in the prophylactic injection of specific anti-serum.

**Treatment.**—This is chiefly surgical, and consists in the excision of the affected muscles or amputation. Good results have been obtained experimentally with serum injections (Bull).

## SYPHILIS

Syphilis, or The Pox, is a specific infectious disease, conveyed by inoculation, and producing successively a lesion at the seat of inoculation (*primary* lesion); lesions of the skin, mucous membranes, and other parts after an interval of a month or more (*secondary* lesions); and, after one or more years, deeper lesions of the skin, bones, muscles, viscera and arteries (*tertiary* lesions). While the *primary* stage of syphilis means that the infection is localised in the neighbourhood of the site of inoculation, the separation of the *secondary* from the *tertiary* stage is distinctly artificial, as both are due to essentially the same pathological processes attacking different parts of the body after generalisation of the infection. However, the distinction is useful in the clinical description, and so it will be adhered to. Syphilis is also the cause of locomotor ataxy and general paralysis of the insane, two diseases of the central nervous system that are sometimes classified together under the term *parenchymatous syphilis*.

Syphilis can be transmitted from parents to children, and then takes on forms which differ in some particulars from the disease acquired in the usual way (*congenital* syphilis).

The micro-organism of syphilis is the *Spironema pallidum* (formerly called *Spirochæta pallida*), first described in 1905 by Schaudinn and Hoffman. It is a long thin filament, of spiral or corkscrew shape, with from six to fourteen coils, and tapering at the ends to a sharp point. The length is from 4$\mu$ to 20$\mu$, the breadth about 0·27$\mu$, and it is stained a rose-pink with Giemsa's stain. It is regarded as belonging to the class of protozoa, and is probably allied to the trypanosomes. It has been found at the seat of infection before there is any evidence of a sore, in chancres, in the lymphatic glands associated with them, in the skin papules of primary and secondary syphilis, in mucous patches and condylomata, and in the blood and spleen. In the gumma of tertiary syphilis, the spirochætes often occupy the peripheral parts. In congenital syphilis, the organisms are found in great numbers in the blood, and in the internal organs, viz. the liver, spleen, lungs, and suprarenal bodies. Recently also Noguchi and others have found spirochætes in the cortex of the brain of general paralytics. In the case of superficial lesions the *S. pallidum* is frequently accompanied by an allied organism, *Spironema refringens*.

### ACQUIRED SYPHILIS

**Infection.**—Syphilis is, as a rule, communicated during sexual intercourse, the delicate epithelium of the mucous membranes brought into contact allowing of the easy transmission of the virus. Cracks or abrasions of the mucous membrane do not seem to be necessary for the reception of the virus, though they must undoubtedly favour it. Inoculation by sexual intercourse takes place commonly in the *male* on the glans penis, prepuce, in the sulcus behind the glans or on the side of the frenum, and occasionally on the scrotum or in the pubic

area, but rarely at the urinary meatus. In the *female* inoculation most commonly occurs on the labia, the fourchette, clitoris and the urinary meatus, and occasionally on the cervix uteri, but rarely on the vaginal wall. Syphilis may also be transmitted in other ways -for instance, in the act of kissing, by smoking pipes previously used by syphilitic persons, or by contact of syphilitic sores or secretions with the abraded finger of the medical man. After inoculation there is usually a period of *incubation*, varying from three to five weeks; the extreme limits are from ten days to three months.

**Primary Lesion.**—The first sign is a small red itching papule, which gradually enlarges in all directions like a flat button, and becomes very hard. The surface is dry, or scaly, or superficially ulcerated and covered with a crust of dry secretion. This condition of induration, which is most important, is reached in a week or ten days from the first appearance of the papule; and the lesion is known as the *hard*, *indurated*, or *Hunterian chancre*. On the mucous membrane the lesion may be scarcely so well marked; it begins as a vesicle with a red base; the vesicle breaks, and forms a shallow ulcer, the floor of which becomes indurated. In the course of time, and it may be some months, the induration gradually disappears, the ulcer heals, and a patch of pigment is left behind for a while. During the first stage of syphilis the poison is transmitted to the *glands* of the groin; and from seven to fourteen days after the appearance of the hard chancre a single large indurated gland, the *primary bubo*, may be felt, with occasional small shotty glands beside it. These remain freely movable upon one another, without adhesion to or reddening of the skin, and they do not suppurate.

Extragenital sores mostly occur on the face, eyelid, lips, tongue, finger, or breast. They are usually larger than genital chancres, and the primary bubo is always large and hard. On the lip or tongue ulceration begins early, and the base is often covered with a thick pseudo-diphtheritic membrane.

**Secondary Lesions.**—The appearance of these lesions from five to eight weeks after the appearance of the primary sore indicates the generalisation of the infection throughout the body. Such lesions may continue to appear at any time up to twelve months or more. The most constant are certain eruptions on the skin (*syphilodermia*, *syphilide*), faucial inflammation, and enlargement or induration of lymphatic glands; others are febrile reaction, anæmia, pains in the temples, back, or limbs, swelling of the joints, iritis, and falling of the hair. Albuminuria may occur.

*Eruptions.*—There are four main characteristics in secondary syphilitic skin eruptions:—

1. Lesions of different types are present together (*polymorphism*), but the size of the spots does not vary much. Large diffuse lesions are not seen.
2. The eruption is widely spread and copious.
3. Itching is uncommon; but this feature is not constant, and there may be concomitant lesions, such as scabies or pediculosis.
4. The shape of the lesions tends to be round, and the spots may be grouped or in rings. Except in the case of the macular eruption, the colour resembles raw ham.

The different types of secondary lesion and their differential diagnosis are as follows:—

1. The *macular* syphilide, or *roseola*, which forms rose-coloured round or oval spots about the size of a threepenny bit, rather thickly grouped. The trunk, neck, limbs, palms and soles are affected. There is no infiltration or scaling. It lasts from three weeks to two months, and may recur. It does not itch. It must be distinguished from measles and rubella and *pityriasis rosea*. The latter consists of pink spots of varying size covered with fine scales, and there is frequently a history of a "herald" spot, seen first of all, which is of larger size than the other lesions, and the patient often complains of itching.
2. A *follicular* syphilide, consisting of small dull red papular elevations, with

a hair in the centre of each; they are often capped with a dry scale, and sometimes become pustular. They must be distinguished from *acne vulgaris*, which usually has a long history.

3. The *papular* syphilide consists of hard elevations, flat or hemispherical, or more prominent still, so as to form nodules or tubercles, which come out in crops irregularly over the whole body, like the macular syphilide, or grouped in clusters; the papules are red or ham-coloured, and have a shiny surface, often with a ring of fine scales round the edge; they may occur on the rose spots of the macular syphilide. This variety must be distinguished from *lichen planus*, where the lesions are polygonal, flat-topped and shiny, with lilac or violet tint, and usually itch. In half the cases of lichen planus there are white papules, streaks or patches in the mouth; but there is no ulceration of the fauces or enlargement of the lymphatic glands, as in syphilis.

4. The *squamous* syphilide consists of infiltrated coppery or ham-coloured papules capped with scales, and is common in the flexures. It must be distinguished from *psoriasis*, where the lesion is not infiltrated, and the scales are of a bright silvery type. Further, psoriasis occurs particularly on the extensor surfaces of the knees and elbows. *Seborrhœic* lesions must also be distinguished. These consist of round spots or circinate lesions with greasy scales; the scalp and margin of the forehead are commonly affected, and they also occur particularly in the middle line over the sternum, between the scapulæ and in the submammary regions. Itching is common.

5. *Rupia* is a comparatively rare lesion, occurring in debilitated subjects. It consists of a round or oval ulcer with a purplish edge and soft base, exuding blood-stained pus, which dries to form a characteristic limpet-shell crust. On healing it forms a deep scar. It must be distinguished from neglected patches of *psoriasis* which are covered with masses of brown scales. In the latter case on removing the scale a number of bleeding points are seen, but no ulcer, as in rupia.

Other forms are—(6) *syphilitic keratodermia*, in which there is thickening of the horny layer of the epidermis of the palms and soles, which may be mistaken for chronic eczema or psoriasis.

7. *Syphilitic alopecia*, which may take the form of general thinning or small patches of baldness about the size of a sixpence. The latter must be distinguished from *alopecia areata*, where the bald areas are round or ovoid and quite smooth, with club-shaped hairs at the spreading edge.

8. The *pigmentary syphilide* on the sides of the neck, consisting of an ill-defined area studded with white spots, occurring almost exclusively in women during the first two years of the disease. Arsenical pigmentation resembles it closely; but the latter is found on covered parts, *i.e.* on the trunk.

9. *Mucous Patches.*—These form flat, wart-like growths, and occur commonly about the genitals, perineum, and anus, in the axillæ, groins, and under the breasts, and at the angles of the mouth—in any place, indeed, where the skin is thin and constantly moist. They are often rather extensive, with well-defined edge, moist surface, and dirty grey secretion. They are to be distinguished from venereal warts (*see* p. 962).

*Sore Throat.* Coincidentally with the rash, or even before it, the throat becomes affected; there is a diffused redness of the fauces, with enlargement and excoriation of the follicles; but the most characteristic feature is the swelling and symmetrical ulceration of the tonsils. The ulcers are often kidney-shaped, superficial, with grey borders, painless, and not of very long duration. Sometimes, however, the tonsillar ulcers are much more persistent, extend to the soft palate and uvula, have bright red edges, and are covered with a yellowish-grey secretion, the removal of which is followed by bleeding. Other changes in the mouth in the secondary stage are white spots, like those produced by the application of nitrate of silver, mucous patches on the tongue or cheeks, bald patches on the tongue from the destruction of the papillæ, or enlargement of the tongue.

which is of bright red colour, with hypertrophied papillæ, or irregular prominences and deep sulci between them; this last condition is aggravated, or in part caused, by excessive smoking. The lesions in the mouth must be distinguished from aphthæ, which are rounded yellow, painful superficial lesions, from herpes, which is also painful, and occasionally from erythema multiforme with extensive lesions in the mouth.

The *lymphatic glands* are enlarged, especially in the groins, above the inner condyles, and at the back of the neck. The *fever* of secondary syphilis is often entirely absent, or it is represented by no more than a slight malaise or indisposition preceding or during the outbreak on the skin. In a small number of cases there is very distinct intermittent or remittent *pyrexia*, the temperature highest in the evening; and it may last for some weeks. The *periostitis* of secondary syphilis is slight; pains and tenderness are felt in the tibiæ, skull bones or clavicles, but they are of short duration, and nodes do not generally form, as in the tertiary stage. The *joints* are not often affected; but there may be synovial effusion, which is sometimes excessive (hydrarthrosis), and is liable to vary from time to time in the same joint.

The most common affection of the eye is *iritis;* it usually affects one eye before the other; the symptoms are photophobia and pain, with ciliary congestion, irregular pupil, obscured iris, and, in severer cases, nodules of rust-coloured lymph and blocking of the pupils. Iritis occurs from three to six months after contagion; at a later period still, but within the limits of secondary symptoms, there may be diffuse retinitis or choroiditis.

Various nervous affections, especially myelitis, are apt to occur within a few months or a year of infection, and thus fall within the category of secondary results. Such disorders show a considerable proportion of recoveries under antisyphilitic treatment.

Some other lesions occur at a time which is intermediate between the second and tertiary periods, such as scaly or peeling patches on the palms of the hands (*psoriasis palmaris*); enlargement of the testis with perhaps nodular deposit in the epididymis; choroiditis and retinitis; and transitory visceral changes not due to gumma—for instance, enlargement or tenderness of the liver and spleen, with failure in the blood-making process, slight and temporary albuminuria, and symptoms of impending pulmonary mischief. And, indeed, no hard-and-fast line can be drawn between the end of the secondary and the beginning of the tertiary stage.

**Tertiary or Late Lesions.**—These are first observed from one to two years after contagion, and may recur at intervals for ten or fifteen years, or more. They are certain eruptions on the skin, periostitis and nodes on the bones, and growths in the subcutaneous tissue, muscles, meninges, liver, spleen, testis, and other viscera.

*Late Syphilides.*—The skin rashes of the tertiary period are variable, and may consist of maculæ or scaly patches. But the most characteristic is a dusky red, infiltrated patch, forming a circle or broad band curved in a half-circle or horse-shoe; part of the surface is covered with a brown or greenish scab, beneath which are deep ulcers with sharply cut edges. The lesion spreads in serpiginous lines by the formation of fresh infiltrations or nodules, which in turn ulcerate, while the old ulcers heal and leave scars surrounded by deeply pigmented skin. Sometimes such nodules will subside and leave scars even without ulceration, and altogether there is a general resemblance to lupus. Ultimately large, irregular patches, of several inches in diameter, may form, and they are frequent on the knee, thigh, shoulder, forearm, face, and neck. Sometimes much deeper infiltrations of the subcutaneous tissues occur.

*Gummas.*—The lesion in the viscera and other parts which is so characteristic of the later stages of syphilis is known as a *gumma*. This is a mass of granulation tissue which may very closely resemble tuberculous granulation tissue; but

giant cells are not so commonly seen, and the smaller arteries often show *endarteritis*. In the early stages it is grey, gelatinous and transparent; but the cells undergo fatty changes, and caseation takes place, so that the centre becomes yellow, and the circumference develops into fibrous tissue, which contracts like that of a scar. Sometimes gummas break down completely, and suppuration, with destruction of the tissue in which they are situated, takes place; thus caries and necrosis not infrequently follow nodes on the bones. In the liver gummas form large, more or less uniform, yellow nodules; or a yellow caseous mass lies in the centre of a fibrous cicatrix; or nothing is left but the fibrous cicatrix, with consequent depression and puckering of the organ. In the testis gummas also occur; but this organ is often enlarged by effusion into its substance generally, and may afterwards atrophy from the formation of a dense fibrous tissue without any local nodular growth. For the clinical results of these lesions the reader is referred to the diseases of different organs. It will be sufficient here to say that gummatous periostitis, or nodes, occur especially along the anterior surface of the tibia, on the frontal and parietal bones, and on the clavicles. The patient suffers from pains, which are worse at night, and there may be found on the affected part flat, round prominences, from ½ inch to 1 inch in diameter, soft, or even fluctuating, and very tender. This is not necessarily a sign of pus being present, as quite distinctly fluctuating nodes may entirely disappear under treatment. Gummas are sometimes found involving the synovial or perisynovial tissues of joints. A very definite *pyrexia* with the temperature rising to 101° or 102° in the evening, and falling to 98° or 99° in the morning, may accompany a gumma apart from suppuration. Syphilis attacks arteries, producing in the case of the larger vessels *arteritis* and *atheroma*, which may lead on to aneurysm, and in the case of the smaller ones syphilitic *endarteritis* (arteritis obliterans). Several disorders of the nervous system are referable to syphilis; some, like hemiplegia, are due to syphilitic endarteritis, leading to thrombosis and consequent softening of the nervous tissue: when this occurs in the brain, it gives rise to hemiplegia, or in the spinal cord to acute or chronic paraplegia, including Erb's *syphilitic spastic paraplegia*; another is probably a true infective myelitis; others, like localised paralyses and convulsions, are due to gummas or meningeal thickenings on the surface of the brain and the roots of nerves. The mucous membranes are affected with deep-seated destructive ulcerations, such as are seen in the mouth, destroying the uvula and soft palate, with adhesion of the remainder to the pharynx, or in the trachea, bronchus or rectum, leading to stricture or stenosis of these passages. Late syphilis is also one of the causes of the lardaceous degeneration, even without the existence of any suppuration.

**Parenchymatous Syphilis.**—The latest results of this disease are certain pathological conditions, with accompanying symptoms, the association of which with syphilis was first determined by statistical evidence, and not by anything characteristic in the tissue changes themselves. These affections are locomotor ataxy and general paralysis of the insane, in which it is found that a history of syphilis is obtained in 70 to 80 per cent. of cases; while some forms of ophthalmoplegia, optic atrophy, loss of pupil light reflex, show similar relations. This view is supported by the results of investigation of such cases by the Wassermann test, which is positive in nearly 100 per cent. of cases of general paralysis and somewhat less in tabes (*see* p. 738), and by the discovery of spirochætes in the brains of general paralytics (*see* p. 862). These diseases are much less amenable to treatment than other forms of syphilis.

**Course and Termination.**—In all stages of syphilis there is a tendency for lesions to appear at sites of injury. As examples may be cited the mucous patches and glossitis of smokers, squamous syphilides on the palms of manual workers, and gumma of the frontal bone in Mohammedans, arising from the practice of striking the forehead during religious worship. Thorough treatment in the early stages may entirely prevent the occurrence of late symptoms, and the secondary

symptoms may be avoided or rendered extremely mild by diligent treatment when the primary lesion is first recognised. The disease has no fatal tendency in the first two stages, but in late syphilis the gumma may act like other tumours, and cause death by direct interference with function, especially in the brain and meninges. Death also results from syphilitic disease of the arteries, from bronchial, tracheal, or rectal stenosis, from periostitis with necrosis of bone and pyæmia from lardaceous disease of the liver, spleen and kidneys, and from the disorders classed as parenchymatous syphilis.

**Diagnosis.**—In the *primary* stage it is important to make the diagnosis as soon as possible, so that treatment may be carried out before generalisation of the infection has occurred. The chancre is distinguished by its induration, and by the presence of the hard primary bubo from: (1) *soft sore*, which is usually multiple and causes soft and tender enlargement of the glands; (2) *herpes genitalis* where the lesions are superficial; (3) very rarely *lichen planus*; (4) traumatic ulceration. In addition, when the lesion has been untreated spirochætæ should be looked for. Some serum is obtained from the lesion, and is mixed on a slide with a few drops of normal saline. The slide is examined with a $\frac{1}{6}$-inch objective by the dark ground illumination. The spirochætæ, which appear white, are seen moving in the field.

From a medical point of view it is the recognition of the late syphilitic lesions that is most frequently required, and help is commonly sought in the former history of the patient. The points likely to be remembered are the occurrence of a definite sore other than mere gonorrhœa, the rash, the sore throat and the falling of the hair. Whether the sore was of the hard or soft variety may be unknown to the patient. The patient may be able to give consistent accounts of the rash, or of the ulcerated sore throat. In married women much reliance is often placed upon the previous occurrence of miscarriages; but miscarriages are frequent under quite different circumstances, and one miscarriage, from whatever cause, is liable to be followed by others.

Search should be made for scars of the original sores on the penis in men, for scars of tertiary lesions on the skin and in the throat, for nodes on the tibiæ and skull, for hardness or atrophy of the testes, and for evidences of lardaceous disease, in the size of the liver and spleen and in the existence of albuminuria.

The diagnosis is sometimes assisted or confirmed by therapeutic measures, when, for instance, a suspected lesion yields rapidly to the treatment mentioned below.

By serological methods it is now possible to detect in the blood serum a specific result of syphilitic infection. Bordet and Gengou showed that if any *antigen* (*i.e.* a substance that has the power of calling into existence *anti-bodies* on being injected into an animal) is mixed and incubated at 37° C. with serum containing such anti-bodies, the two combine together and at the same time fix *complement*, a ferment normally present in the blood. This phenomenon has been applied to the diagnosis of syphilis, and is known as the *Wassermann reaction*. The antigen originally consisted of an extract of syphilitic liver, and this was added to serum taken from the patient to be investigated. If complement is fixed, the patient is to be regarded as syphilitic. This fixation is determined by means of a special hæmolysis test. If red cells from some animal, like a sheep, are injected into another animal, such as a rabbit, they behave as antigen, so that the corresponding anti-body, or hæmolysin, is formed. If the red cells and serum containing the hæmolysin are mixed, hæmolysis occurs so long as complement is present, but does not occur in the absence of complement. For the test the suspected serum believed to contain syphilitic anti-body is heated to destroy complement and then mixed with the extract of syphilitic liver, and with normal guinea-pig's serum containing complement. These are kept for an hour at a temperature of 37° C., and the mixture of rabbit's serum containing hæmolysin (but deprived of complement by previous heating to 56° C.) and the washed ox corpuscles is then added,

and the whole mixture further submitted to 37° C. for two hours. If the serum in question contains anti-body of syphilis, the complement will be bound, or fixed, by it in the first incubation, and the hæmolysin in the rabbit's serum not meeting with free complement in the second incubation, will not destroy the corpuscles — *i.e.* there will be no hæmolysis. This event is called, in regard to complement fixation and to the presence of syphilis, a *positive reaction*. If, on the other hand, the anti-body of syphilis is not present, the complement will not be bound in the first incubation; it will co-operate with the hæmolysin in the second incubation, and hæmolysis will take place. This is a *negative reaction*. By carrying out the whole process on quantitative lines it is possible to judge from the amount of hæmolysis that occurs the amount of syphilitic anti-body present in the patient's serum.

As a matter of fact, the reaction as carried out at the present day has been modified in one particular way. For some unexplained reason it has been found unnecessary to use a syphilitic liver to prepare the antigen. Certain other substances will do, and an extract of sheep's heart mixed with cholesterin answers the purpose, so that this is always used now as antigen, because it is easier to obtain. These facts indicate that the original explanation of the Wassermann reaction no longer holds good.

The Wassermann test gives a positive reaction in primary cases in from five to eight weeks after infection, in 95 per cent. of secondary cases, in 75 per cent. of tertiary cases, and in 50 per cent. of cases where syphilis is latent (D'Arcy Power). It is very frequently found (70 per cent. or more) in the case of apparently healthy mothers of children congenitally syphilitic. The child itself may give a negative reaction in spite of the mother's positive result, or a positive reaction may appear with the outbreak of definite symptoms.

The Sachs-Georgi reaction is another serum reaction that has been used in diagnosing syphilis. It depends on the fact that serum from a syphilitic patient can flocculate a saline suspension of a mixture of alcoholic heart extract and cholesterin. Recently Dreyer and Ward have prepared and standardised a preparation of these substances that will keep indefinitely, and various observers say that a more accurate quantitative measure of syphilitic anti-body can be obtained by this method ("Sigma Reaction") than by the Wassermann reaction, while at the same time the test is a much simpler one to carry out.

In patients under treatment the reaction may become negative, and yet in many cases, if a dose of salvarsan (*see* below) is injected, the reaction again becomes positive, showing that the syphilitic infection is still present; and freedom cannot be pronounced until the negative reaction persists in spite of this so-called "provocative injection" of salvarsan.

Although a *negative* Wassermann reaction is strong evidence that a patient is not suffering from syphilis, a *positive* reaction has been obtained in other conditions, *i.e.* in pregnancy, in malnutrition such as may occur in diabetes, in diseases of the liver, in non-syphilitic skin diseases after treatment with arsenic intravenously, and in leprosy and yaws and high fevers. These facts must be taken into account before diagnosing syphilis.

**Prevention.**—As syphilis is rarely conveyed otherwise than by direct contact, it should be easy to prevent its spread, if those who are known to be infected could be made to abstain from contact, sexual or otherwise, with healthy persons; but those who are infected do not always know that they are so.

Legislation has many difficulties to contend with. Following the report of the Royal Commission in 1916, the State has made grants of money for the purpose of providing means of bacterial diagnosis, of promoting the establishment of treatment centres all over the country free to all who apply, of educating practitioners in the use of newer methods of treatment, and of supplying salvarsan or its substitutes free to medical practitioners. In the vast majority of cases which have been exposed to infection, immediate disinfection—thoroughly washing the

parts and rubbing in the 30 per cent. calomel ointment mentioned on p. 120 will prevent infection taking place. A solution of mercury cyanide (1 in 1,000) is an even better antiseptic.

Pregnant women known to be syphilitic, or demonstrated to be so by the Wassermann test, should be promptly treated, to secure the health of the offspring.

The following facts may guide the medical man in advising patients. Both primary and secondary lesions are contagious, and the blood during these periods contains the virus. On the other hand, it seems clear that the normal secretions —saliva, milk, sweat, and semen—do not contain the virus and so do not cause the disease when they are inoculated into abrasions. It will be seen that syphilis of the fœtus is probably always due to maternal infection (*see* p. 121).

As a rule, syphilis confers upon the sufferer immunity from fresh infection, though by some it is stated that the immune person is only an *uncured* syphilitic. Instances are recorded in which, after a long interval, a fresh primary sore and fresh secondaries have appeared, and here we must suppose that the protective influence, if it ever existed apart from the spirochæte and its toxins, has died out, as it does rarely in the exanthems.

**Treatment.**—The extraordinary tenacity of the spirochæte of syphilis and its possible influence for years after infection upon the bodily structures makes it essential that treatment should be prompt and persistent over a long period of time. Three groups of drugs have a pronounced effect upon the disease: (1) mercury and its compounds; (2) potassium iodide; and (3) certain arsenical compounds.

Salvarsan or dioxy-diamino-arsenobenzol dihydrochloride was introduced by Ehrlich (1909), and was known for a time as "606." It is best employed by intravenous injection, which should be carried out on an empty stomach, with the bowel empty. It is often advisable to give glucose just beforehand (*see* p. 124). Great care is required in the preparation of the fluid to be injected and in the operation, to avoid any of the fluid getting into the subcutaneous tissue. The dose of salvarsan is dissolved in 30 to 40 c.c. of sterile distilled water; some 10 to 20 drops, in proportion to the dose of salvarsan, of a 15 per cent. solution of sodium hydrate are added, and a precipitate forms, which again dissolves on shaking, or on the addition of a few drops more of the alkali. To the clear solution are now added 250 c.c. of a 0·5 per cent. saline solution, made with sterile freshly distilled water. In the injection the actual solution of salvarsan should be preceded by ½ ounce of the saline, to make sure that the needle is properly in the vein, and should be followed by a similar amount of saline to wash out the needle thoroughly. Other names of preparations of "606," besides salvarsan, are arsenobenzol (the official B.P. name), arsenobillon, kharsivan, diarsenol, arsphenamine.

Neo-salvarsan, or "914," is an allied drug, dioxydiamino-arsenobenzol-monomethylene-sulphoxalate of soda, and is also known as novarsenobenzol (B.P.), novarsenobillon, neokharsivan, neodiarsenol, neoarsphenamine. Its advantage over salvarsan is that it is soluble in water and so can be injected directly into a vein by means of a needle and syringe. The solution must not be kept, but must be injected immediately it is made. Neo-salvarsan is more easily given than salvarsan, it produces less disturbance, but when given intravenously it has not as much therapeutic effect.

Sulpharsenol, closely allied to "606," causes very little pain when given subcutaneously or intramuscularly, and is of value when the Wassermann reaction continues to be positive. It has been found that a subcutaneous injection of this compound gives a better therapeutic effect than an intravenous injection of the same dose. It can be given without reference to meals. Silver salvarsan is a combination of sodium salvarsan with silver and has been found very useful in neuro-syphilis.

In the days when mercury and iodides were alone available for treatment, it was generally agreed that a two years' course was desirable, and even so in nearly half the cases the Wassermann reaction still remained positive. The results were disappointing. Frequently the patients relapsed while under treatment or showed intolerance for mercury in the form of stomatitis or albuminuria. The introduction of "606" caused a revolution. Spironemes disappeared from the lesions within twenty-four hours, and some of the most resistant lesions themselves within a week. Many believed that a dose or two was sufficient to produce a cure of syphilis, and the error was only realised when recurrences of symptoms were observed, which frequently took the form of syphilitic meningitis and cranial nerve paralyses. It is only now being generally recognised that a prolonged course of treatment is desirable, and owing to the incomplete treatment of patients in the early years of the new remedies, we are not yet in a position to state what the effect of the full treatment really is. The course of treatment depends on the stage of the disease and the Wassermann reaction. Harrison (**14**) gives it in tabular form as follows :—

## ROUTINE TREATMENT OF SYPHILIS CASES.

### A.—Primary Cases with Negative Wassermann.

*Alternative Arsenobenzol Treatment.*

| | Day of Treatment. | 606 Iv | 914 Iv | S Im | SS Iv. | Hg Im. | KI |
|---|---|---|---|---|---|---|---|
| | | gram. | gram | gram | gram. | grain. | grains. |
| (1) | 1 | 0·3 | 0·45 | 0·48 | 0·20 | — | — |
| | 8 | 0·3 | 0·45 | 0·48 | 0·20 | i | |
| | 15 | 0·3 | 0·45 | 0·48 | 0·20 | i | — |
| | 29 | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| | 36 | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| | 50 | 0·5 | 0·75 | 0·60 | 0·25 | i | — |
| | 57 | 0·5 | 0·75 | 0·60 | 0·30 | i | — |
| | 58–64 | — | — | — | — | — | v t.d.s. |
| | 65–71 | — | — | — | — | — | vii t.d.s. |
| | 72–77 | — | — | — | — | — | x t d.s. |
| | 78 | 0·5 | 0·75 | 0·60 | 0·30 | i | — |
| | 85 | 0·5 | 0·75 | 0·60 | 0·30 | i | — |
| | 92* | 0·5 | 0·75 | 0·60 | 0·30 | i | — |
| (2) | 93–126. Rest. | | | | | | |
| (3) | 127–133 | — | — | — | — | — | v t.d.s. |
| | 134–140 | — | — | — | — | — | vii t.d.s. |
| | 141–147 | — | — | — | — | — | x t.d.s. |

(4) Repeat treatment as from first to ninety-second day.
(5) Suspend all treatment but test the blood every three months.
(6) At end of one year test blood and cerebro-spinal fluid after provocative injection.
(7) During second year test blood every six months.
(8) At end of second year test blood and cerebro-spinal fluid after provocative injection.

* Blood test on 92nd day.

In the headings of the tables—

606 = Salvarsan, kharsivan, arsenobillon, or diarsenol.
914 = Neo-salvarsan, neo-kharsivan, novarsenobillon, or neodiarsenol.
S = Sulpharsenol.
SS = Silver salvarsan.
Hg = Mercury.
KI = Potassium iodide.
Iv. = Intravenous.
Im. = Intramuscular.

B.—Primary Cases with Positive Wassermann.

| Day of Treatment. | | 606 Iv | 914 Iv. | S Im. | SS Iv. | Hg Im. | KI |
|---|---|---|---|---|---|---|---|
| | | gram | gram. | gram | gram | grain. | grains |
| (1) 1 | 239* as in "A" | | | | | | |
| (2) 240 | 307. Rest. | | | | | | |
| (3) 308 | 314 | | | | | | v t.d.s. |
| 315 | 321 | | | | | | vii t.d.s. |
| 322 | 329 | | | | | | x t.d.s. |
| (4) 330 | | 0·4 | 0·60 | 0·60 | 0·25 | i | |
| 337 | | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| 344 | | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| 358 | | 0·5 | 0·75 | 0·60 | 0·25 | i | |
| 365 | | 0·5 | 0·75 | 0·60 | 0·25 | i | |

(5) Suspend treatment and continue blood tests at intervals as in "A" 5–8.

* Blood test on 92nd day.

*N.B.*—This programme is based on the assumption that at the end of the first ten injections the Wassermann reaction was negative. If the Wassermann reaction was positive after the tenth injection proceed as laid down for cases with secondary signs.

C.—Cases with Secondary Clinical Signs.

| Day of Treatment. | | 606 Iv. | 914 Iv. | S Im. | SS Iv. | Hg Im. | KI |
|---|---|---|---|---|---|---|---|
| | | gram. | gram. | gram. | gram. | grain. | grams. |
| (1) 1 | 365* as in "B" 1–4 | | | | | | |
| (2) 366 | 433 Rest. | | | | | | |
| (3) 434 | 441 | | | | | | v t.d.s. |
| 442 | 448 | | | | | | vii t.d.s. |
| 449 | 455 | | | | | | x t.d.s. |
| (4) 462 | | 0·4 | 0·60 | 0·60 | 0·25 | i | |
| 469 | | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| 476 | | 0·4 | 0·60 | 0·60 | 0·25 | i | — |
| 490 | | 0·5 | 0·75 | 0·60 | 0·30 | i | — |
| 497 | | 0·5 | 0·75 | 0·60 | 0·30 | i | — |

(5) Suspend treatment and continue observations as in "A" 5–8.

* Blood test on 92nd and 365th day.

D.—Tertiary and Latent Cases.

| Day of Treatment. | |
|---|---|
| 1–57 | First course of seven injections as in "A" 1. |
| 58–77 | Potassium iodide. |
| 78–147 | Rest. |
| 148–182 | Course of five as in "B" 1. |
| 183–203 | Potassium iodide. |
| 204–336 | Repeat 78–203. |
| 337–469 | Ditto. |
| 470–567 | Rest. |
| 568–602 | Course of five as in "B" 1. |
| 603–623 | Potassium iodide. |
| 624–749 | Rest. |
| 750–781 | Course of five as in "B" 1. |

It will be observed that much use is made of the Wassermann reaction to indicate the progress of treatment. Further, in early cases the treatment is continued for long after the Wassermann reaction is negative, because even so the disease may still be active. In fact a few patients have a negative reaction even though they are quite obviously suffering from secondary syphilis. In a case with a negative reaction a small "provocative" dose of arsenobenzol may be given. If the reaction now becomes positive it is definite proof of the activity of the disease.

The treatment of syphilitic myocarditis and aneurysm (*see* p. 356), of hepatic syphilis (*see* p. 490), and of neuro-syphilis (*see* p. 792), are dealt with in the appropriate places.

The only contra-indications to treatment along these lines are intolerance on the part of the patient. Intolerance to arsenobenzol is fully discussed on p. 123, and it is essential to take the precautions mentioned there, since many deaths from poisoning have been reported.

In the above scheme *intramuscular injections* of mercury are used. This is an effective and fairly simple method. One-third of a grain of the perchloride is dissolved in 20 minims of water and injected into the gluteus maximus once a week (Bloxam), or 1 grain of red iodide of mercury in 64 minims of distilled water, with sufficient sodium iodide to dissolve it (dose 2 to 6 minims). Calomel can also be employed in the same way, 1 grain being suspended in sterilised olive oil; and the metal mercury itself, worked up with lanolin or other fat in the preparation known as *grey oil*, is injected in a dose equivalent to 1 or 1½ grain.

The method of *inunction* is undoubtedly a very thorough way of introducing mercury into the system, but it requires to be done by expert attendants, and on a uniform method. Mercurial ointment to the extent of 30 or 40 grains, mixed with half as much lanolin, is rubbed into the skin by an assistant for from fifteen to thirty minutes. Different parts of the body are selected on successive days, for instance, the arm one day, the forearm the next, the back, chest, thighs, and legs, on successive days, and then the arm again. A complete course includes a daily inunction for six weeks, a rest of three months, another course of six weeks' inunction, a rest of three months, and then a month's inunction, six months' rest, another month's inunction, six months' rest, and finally twenty days' inunction, bringing thus the whole period up to two years.

*Oral* administration of mercury is on the whole the least effective method, but the simplest, and so it is still commonly used. The more usual preparations are the perchloride in doses of $\frac{1}{16}$ to $\frac{1}{12}$ grain (60 to 80 minims of the liquor) three or four times a day, and hydrargyrum cum creta (grey powder) in doses of 1 or 2 grains with the same frequency. Hutchinson advised 1 grain of grey powder

with 1 grain of Dover's powder, if necessary, to be given every six, four, three, or two hours.

During the use of mercury the patient should abstain from smoking and from stimulants, frequently clean the teeth and mouth, and live in every way as healthy a life as possible. Excessive treatment causes salivation (*see also* Chronic Mercurial Poisoning, p. 683).

*Iodide of potassium* is especially useful in the treatment of later manifestations, but in other stages it may be used in combination with the perchloride of mercury. Under its use the most serious and alarming nervous symptoms, due to syphilitic gummatous lesions, rapidly subside, ulcerating skin lesions quickly heal, pains in the bones subside, and periosteal nodes disappear. Five or 7 grains three times a day are often sufficient, but in serious cases it should be pushed to ½-drachm or drachm doses three times daily; or as much as 20 grains may be given in a little milk every two hours through the whole day and night. The advantage of this, no doubt, lies in the thorough saturation of the system; otherwise, as the salt passes away rapidly by the kidneys, the amount in the body may fall very low in a long night interval. If iodide, in any dose, causes coryza, it should be taken much diluted—*e.g.* in half a tumblerful of water; arsenic may be added if it causes eruptions (*see* Medicinal Eruptions). General tonics, good food and sea air are desirable if it causes much depression, or the iodide of sodium may be given instead in corresponding doses, or a mixture of the iodides of potassium, sodium and ammonium in equal parts. If these fail, recourse may be had to mercury, either alone or with a tolerable dose of potassium iodide.

The destruction of the primary sore will probably not prevent generalised infection, but, in view of the presence of organisms therein, it is advocated by some as an additional measure. Excision, cauterisation, and the application of an ointment—calomel 33 parts, lanoline 67, and vaseline 10—have been tried. In the latter case the patient is advised to try to "rub the sore away with the ointment."

## CONGENITAL SYPHILIS

Children born of parents suffering from syphilis in the first or second stages may themselves be infected with the disease. As a rule, this transmission does not take place in the tertiary stage. Congenital syphilis is due to infection of the fœtus by the mother, who may herself have been the subject of syphilis before conception or may contract the disease during pregnancy. Fœtal infection may occur through the placenta at any time during the pregnancy, and the later it occurs the better chance there is of the fœtus surviving. Infection may occur during parturition. In this case a primary chancre develops at the site of an abrasion (*e.g.* from forceps), as the result of infection from primary or secondary lesions in the maternal passages.

It was formerly stated that a child might be infected with congenital syphilis from the father, the mother being healthy. Further, under these circumstances, the syphilitic infant might infect a wet-nurse- *e.g.* causing a chancre on the nipple—but would not infect its own mother, thereby showing that the mother was in some way protected against infection, though she might manifest no lesions whatever of a primary or secondary kind. This is called *Colles' law.* The now generally accepted explanation is that the father has really infected the mother, who however shows no signs of the disease, and she passes on the infection to the fœtus, and this is proved by the fact that the Wassermann reaction has been found to be positive in 95 per cent. of mothers of syphilitic children when they have been examined soon after the birth of the child.

Infection *in utero* does not always occur when there is maternal syphilis, and sometimes its transmission is irregular, as is shown by the fact that the same parents may have children showing a positive Wassermann reaction alternately with children showing a negative reaction, and one child may be badly affected, another not at all.

**Death of the Fœtus.**—One effect of syphilis in the parent is the early death of the fœtus, with resulting miscarriage or premature birth. In fact, 15-20 per cent. of all miscarriages and still-births are due to syphilis. The fact of miscarriages having occurred in the history of a married woman may be important evidence as to syphilis in herself or her husband. It is not so easy to say the exact cause of the fœtal death, whether from the immediate effects of the syphilitic virus or from some disease of the placenta. Hard yellow masses have been found in this structure, but usually there are no naked-eye characteristics. Microscopically the villi show increase in size and vascularity. Endarteritis is absent. On the other hand, the fœtus not infrequently presents lesions of the bones, viscera, and skin which show that it may be profoundly diseased. In the bones there is irregularity and deepening of the line of calcification at the junction of the epiphysial cartilages and the shaft. This is described as osteochondritis or *epiphysitis*, and resembles the changes in rickets, except that in syphilis the calcification is more evident. The cartilage may be separated from the bone by soft granulation tissue or pus. The changes in the liver are described on p. 490. The lungs show increase in the interalveolar tissue, with compression of the alveoli and capillaries and sometimes gumma. Increase of connective tissue is present in the thyroid, thymus and pancreas, and ulcers in the intestines. Spirochætes may be found where the lesions are marked.

**Early Symptoms.**—Epiphysitis, which occurs in about 16 per cent. of cases, is sometimes present in children born alive, the principal epiphyses being separated from their bones, and the limbs consequently lying useless, so as to give the appearance of paralysis. Occasionally also the child is born with a bullous eruption on the skin, or the rash comes out very soon after birth. The palms and the soles are always affected. This is a syphilitic rash, and must be distinguished from *Pemphigus neonatorum*, which is a bullous impetigo due to sepsis usually at the umbilicus. But in a large majority of cases the child is born not only alive, but healthy, fat, and plump, and remains so for three or four weeks after birth. Then it acquires a nasal catarrh, *rhinitis*, causing the symptom commonly known as *snuffles*, with a discharge, at first thin and serous, afterwards thicker, purulent, and drying up into crusts, which obstruct the nostrils, so that sucking is difficult. At the same time a *rash* appears, most commonly on the buttocks and adjacent parts of the thigh, back, and abdomen. It consists most often of circular patches, brownish red like the lean of ham, dry, shiny, and inelastic; the patches run together, and form larger areas of irregular shape, but mostly with a well-defined edge. This is to be distinguished from intertrigo, which occurs in the flexures, and from the "napkin rash," a bright shiny red inflammation affecting the convex surfaces over the area which comes in contact with a dirty napkin (*see* pp. 929, 930); and the two conditions probably sometimes co-exist. Less frequently the rash is papular, pustular, and bullous. Other lesions occurring in early infancy are stomatitis, ulcerations about the lips and angles of the mouth, rapidly forming cutaneous abscesses, and periostitis; and the spleen is enlarged in very many cases. With all this the nutrition of the child may be little affected, but sometimes wasting results, and the face acquires a withered and shrunken appearance like that of an old man. In this stage death may occur; but under treatment, or otherwise, all the symptoms may subside, and the child may show no indications of the taint for many years, when, often about the time of puberty, symptoms appear which are more or less comparable with those of the third stage of the acquired disease. The Wassermann reaction is positive in 90 per cent. of the cases, and the mortality may be as much as 40 per cent.

**Later Symptoms.**—These are—periostitis with the formation of nodes; synovitis, especially a chronic synovial effusion into both knees; scaly or lupoid skin eruptions, which are not very common; bilateral deafness coming on with noises in the ears, but without pain or discharge; disseminated choroiditis;

dark brown punctate pigmentation of the *fundus oculi*; iritis; and keratitis. The last is common in inherited syphilis, not so in the acquired disease; it causes opacity of the cornea, which gradually increases till the cornea looks like ground glass. It is associated with ciliary congestion, and in late stages vessels may encroach upon the cornea, producing a "salmon patch." Its tendency is to recover. But, in addition to these fresh lesions, inherited syphilis may be recognised by some permanent deformities, the result mostly of those changes which took place in infancy. Such persons present a broad forehead, with unusual prominence of the two halves of the frontal bone; the bridge of the nose is broad and sunken; around the mouth are numerous linear cicatrices radiating from the orifice as a centre; and the permanent teeth, as was first pointed out by Hutchinson, show features from which alone an absolute diagnosis of the condition may be made. It is only the upper central incisors that can be relied upon for this purpose, though other teeth may be similarly affected: they are short, narrower at the edge than near the gum, and the edge presents a single central cleft or notch. This notch is at first, soon after the eruption of the tooth, filled by a notched edge of exposed dentine, which soon breaks away. This change in the teeth must be distinguished from the simple transverse marking, which may result from the excessive use of mercury in infancy, causing stomatitis, and interfering with the proper development of the tooth sacs. Periosteal changes in the tibia may result in a convexity of the anterior border, the so-called *sabre-shaped tibia*. Visceral changes are also not uncommon, such as enlargement of the spleen, cerebral inflammation or degeneration (*see* Cerebral Diplegia), occasionally orchitis, interstitial hepatitis (*see* Cirrhosis of the Liver), anæmia with or without splenic enlargement, and gummas revealing themselves in adult life.

**Prevention.** This may be considered under two heads:

(1) *Prevention of Infection of the Mother.* Men with syphilis should not marry until there is no danger of transmitting the disease. They should have had adequate antisyphilitic treatment and be free from all clinical evidence of the disease, and the blood should have given a negative Wassermann for at least two years. Sometimes the blood remains persistently positive. In such cases there can be no certainty, and the risk though slight must be explained to the patient. Not uncommonly a husband becomes infected from irregular sexual intercourse during the later months of pregnancy or the puerperium. Adequate and prompt treatment must be undertaken and he must abstain from sexual relations until free from infection.

(2) *Treatment of the Syphilitic Mother before and during Pregnancy.*—The Wassermann reaction is of great value in diagnosing maternal syphilis, because many women suffer from syphilis without knowing it. The primary lesion may be hidden away and cause very little discomfort. It is certain that in some normal women the Wassermann reaction is positive during pregnancy to become negative again later. Hence, when the Wassermann is positive during pregnancy, antisyphilitic treatment should only be carried out if there is additional evidence of syphilis, *i.e.* a history of previous miscarriages, etc., or a positive Wassermann reaction obtained from the husband's blood, which should always be tested. Antisyphilitic treatment (*see* p. 117) may be given all the way through pregnancy, and should be continued afterwards. Women usually prefer Hyd. c. Cret. to mercurial injections.

**Treatment.**—Mercury acts with remarkable rapidity in infantile syphilis. A grain of grey powder three times a day, or liq. hyd. perchlor. 10 or 20 minims, will quickly cure the rash, snuffles, or other symptoms, and improve the nutrition of the child if it is defective. The best method of treatment is by mercurial inunction, ¼ drachm of unguentum hydrargyri being rubbed into the abdomen once daily after bathing. A flannel binder is worn over the place. The mother should be treated if possible. Iodide of potassium is of less value, but may

be given in doses of 2 or 3 grains three times daily. To mucous patches, or ulcerations of the skin, calomel powder or mercurial ointments may be directly applied.

Neo-salvarsan also causes rapid disappearance of the rash and of the rhinitis; it has been used by injection into the jugular vein or scalp veins in doses of from 0·075 to 0·15 gramme. Injection into the superior longitudinal sinus has also been carried out. However, mercury treatment will also be required. When the infants are breast-fed, the mother should be treated if possible.

## POISONING BY ARSENO-BENZOL

Many deaths have been reported from poisoning by organic arsenic compounds since their widespread introduction in the treatment of syphilis. At least three conditions must be differentiated.

(1) The sudden liberation of endotoxins from the spirochætes by the destructive action of the drug (Jarisch-Herxheimer reaction). There is pyrexia beginning six to ten hours after the injection, with rigors, nausea, vomiting, abdominal pain and diarrhœa. The temperature becomes normal in a few hours. To prevent this syphilitic treatment should be begun with small doses of arseno-benzol.

(2) "Water-fever," which is ascribed to the protein derivatives of the dead bodies of the bacteria in the sterile water. It starts within half an hour of injection, and there may be rigors and cyanosis. To prevent "water-fever," the distilled water should be freshly prepared and sterilised before use.

(3) Poisoning by the organic arsenic compounds. The poisoning may sometimes be due to impurities in the drug. Oxidation readily occurs during its manufacture, and the resulting substances are poisonous. (*a*) If maximal doses (0·6 gramme) of "606" are given at too frequent intervals *epileptiform convulsions* may supervene about five days after the injections, leading to death. Post-mortem examination shows a hæmorrhagic encephalitis, with widespread thrombosis of capillaries. There may also be a hæmorrhagic nephritis. By avoiding the use of too large doses and by leaving intervals of time between the various courses of "606," as already described, there should be plenty of warning of approaching danger and the "606" stopped. Injections of adrenalin (1 in 1,000) in 10-minim doses may be given for the convulsions. (*b*) Poisoning may cause *dermatitis*, which takes the form of an erythematous or papulo-erythematous eruption, leading on in some cases to exfoliative dermatitis and death. The onset may be delayed, appearing as late as three months after a course of injections. The treatment must be stopped at the earliest sign of an erythema, and this may be visible first of all at the bend of the elbow where the iodine has been applied at the previous injection. This is a valuable indication. A loss of ankle jerks also indicates the onset of poisoning with these drugs.

(*c*) The *liver* may be attacked. In some cases it may have been previously damaged, and the drug depresses its functions still further, so that symptoms of poisoning arise. It is probably the arsenic rather than the organic radicals in the compound that is chiefly responsible, but the latter may play some part. In fatal cases the liver is found to be small, there is wrinkling of the capsule and on section an appearance resembling acute yellow atrophy may be seen. The liver also shows fatty degeneration. The kidneys are degenerated.

The symptoms sometimes come on acutely after two or three injections have been given, but usually they appear any time from about ten days to six or seven weeks, or even twelve months, after a course of treatment. Jaundice is first noticed (*see* p. 474), with pale stools; later nausea, vomiting, epigastric pains, hæmatemesis, and, in fatal cases, delirium and coma. The jaundice may take several weeks to disappear. X-ray examination is a valuable method of diagnosing atrophy of the liver (Strathy and Gilchrist). In the vertical position there is

a diminution in the height of the shadow. The upper surface is more dome-shaped owing to relaxation of the capsule and traction of the lung, and the lower border is rather more vertical than normal.

At present all commercial samples of arseno-benzol and its derivatives are tested by the Medical Research Council so as to exclude impurities. Patients must be carefully examined before treatment for signs of liver or kidney disease, and must be watched during treatment for signs of jaundice. In doubtful cases the hepatic efficiency may be determined (*see* p. 473), the levulose and van den Bergh's tests being specially valuable. It is a good plan to give 2 ounces of dextrose just before injection, especially if the patient is a woman. The treatment consists in giving light diet with plenty of carbohydrates and little fat in order to diminish the work of the liver. Sodium bicarbonate is also used.

## FRAMBŒSIA

(*Yaws*)

This is a specific contagious disease which is prevalent in tropical countries. It is known as *yaws* in British colonies, as *pian* in French colonies, as *bubas* in South American countries, and as *puru* in Borneo and the Malay States. It also occurs under different names in Ceylon, West Africa, and the South Sea Islands. The disease affects the sexes equally, young people more than others, and dark races more than the white. It has considerable resemblance to syphilis, and is contracted by infection of some superficial lesion, such as a scratch, wound, or insect bite on the body, mammæ, hands, arms or legs, but rarely on the genital organs. After an incubation of from two to four weeks, during which there is some malaise, the *primary* lesion occurs at the site of inoculation, and consists of a small papule, which after a week becomes moist with a yellowish secretion, and this dries into a crust. Under this an ulcer forms; it may heal and leave a scar, or develop into a granulomatous mass. The lymphatic glands in the neighbourhood may enlarge and harden, but rarely suppurate. The *secondary* eruption appears from one to three months after the first appearance of the primary lesion, and is also preceded by some malaise, fever, and pains in muscles, bones, or joints. The lesions begin in the same way. One or more papules appear, which are at first about the size of a pin's head, and increase until they measure from ¼ to 2 inches in diameter. The epidermis gets thin, gives way, and leaves a raw surface, from which a sero-purulent fluid oozes, and this dries into a crust. In shape the tubercles are round, oval, or irregular from coalescence, soft, not sensitive to touch, but itching. They affect the face, lips, nostrils, neck, arms, axillæ, legs, thighs, palms and soles, buttocks, and vulva. If they improve, they contract, dry up, and leave a discoloration of the skin; but they may form large irregular sores, and lead to deep ulceration or gangrene.

Arthritis with fever, periostitis, muscular contractures, and neuritis are conditions which may accompany the eruption. As a rule, the disease ends with the shrinking of the papules, but there may be a *tertiary* stage of gumma-like nodules in various tissues, deep ulcers of the skin, or nodes on the bones.

Histologically the lesions of this disease are granulomata; but the essential cause appears to be a spirillum, or spirochæte, the *Spironema pertenue*, which has been found by Castellani in the primary lesions, in the unbroken papules of the general eruption, in the spleen, lymph glands, and bone marrow, but not in the blood.

The disease has been conveyed to monkeys by inoculation of scrapings from the papules, and of the blood of a patient.

The disease untreated may last from six to twelve months, or even for years.

It is rarely fatal; and when it is so, it is generally from secondary infections, leading to phagedæna, septicæmia or pyæmia.

**Treatment.**—Salvarsan given in the same dose as in syphilis (0·6 gramme) cures yaws promptly. Of 400 cases in Antigua all but two were cured by one intramuscular injection, and the remaining two were cured by a second injection (McDonald); of 500 cases in Trinidad 498 were cured, and of these 409 by a single injection. Two other arsenical preparations, soamine and orsudan, have a similar, but much less marked, effect (Alston). Formerly potassium iodide, mercury, and liquor arsenici et hydrargyri iodidi were used. Locally the eruption may be washed daily with solution of perchloride of mercury (1 in 1,000), and ulcers may be treated with astringent and antiseptic preparations, such as iodoform and boric acid.

## TUBERCULOSIS

Tuberculosis is an infection by a specific micro-organism—the *Bacillus tuberculosis* of Koch, which is characterised by the formation in one or more organs of certain bodies called *tubercles*.

**Ætiology.**—Tubercle bacilli are minute rods, straight, or very slightly curved, measuring 3$\mu$ in length and 0·5$\mu$ in breadth. They have rounded extremities, and present two or more bright spots, often one at each end, which were first regarded as spores. Like other micro-organisms, they can be stained by special reagents, and a method of detecting them by this means in the sputa is described hereafter (*see* Diagnosis of Phthisis). Some recent observations point to the possibility that tubercle bacilli are not, as formerly thought, fission fungi, but that they belong to the class of *Streptothrix*, one of the Hyphomycetæ. The bacilli are widely spread in nature. Human, bovine, avian, reptilian and piscine types exist having different biological characters. It is claimed that by special methods of cultivation these differences can be abolished. Both human and bovine types are pathogenic to man.

Tuberculosis is practically an endemic disease: the human type of bacillus is probably widespread in all large communities; and there is good reason to believe that it infects and causes the growth of tubercle in a great number of persons, without its presence ever being detected. Post-mortem observations show that old healed foci of tuberculosis are present in most people. Living bacilli have also been found latent in the glands of about 10 per cent. of people where the autopsy has revealed no macroscopic or microscopic evidence of tuberculosis. The conditions determining its development are (1) the virulence of the organism, (2) the susceptibility or resistance of the recipient individual, and (3) the mode of introduction.

1. Under the first head there is little that can be said but that the *virulence* undoubtedly varies.

2. In regard to the *condition of the recipient* importance attaches to *hereditary* influence. The conclusion has been drawn from statistics that the children of tuberculous individuals have a special hereditary predisposition to the disease (Pearson). At the same time it must be remembered that such children are brought up in surroundings where they may receive massive doses of bacilli, and Ward's suggestive observations point to this as being the most important factor (*see* later). On the other hand, the long narrow chest, showing deficient apical movement, may predispose to phthisis. This is often a congenital characteristic. Experiments with animals have shown that the semen from tuberculous vesiculæ seminales or testes may in some cases produce a tuberculous fœtus. Placental tuberculosis may also do the same, but these factors are of no importance as a cause of tuberculosis in later life. There is a great variation in the relative resistance of the different races of mankind to infection by tubercle. Primitive races like the Red Indians and Australasian natives, who have

hitherto been free from tuberculosis, show a very high mortality when exposed to infection by contact with civilised communities. On the other hand, the Jews, who, owing to city life, have been exposed to infection for many generations, show a very high resistance.

A disposition to tubercle may be *acquired* by any circumstances, or combination of circumstances, which expose the body to massive infections or seriously lower its vitality, whether these be a deficient supply of food and fresh air, or prolonged debilitating illnesses, or special toxic influences. The most frequent of these are (*a*) overcrowding and deficient ventilation, working in close rooms in the fumes of gas, etc.; (*b*) deficient supply of food, which frequently co-operates with the first cause, as well as the next; (*c*) exhausting work in association with the preceding; (*d*) exposure to wet and damp Buchanan showed that amongst communities living on damp and imperfectly drained soils there was an undue proportion of deaths from phthisis and lung diseases; strong rain-bearing winds (**17**), (*e*) excessive indulgence in alcoholic drink; (*f*) diabetes mellitus.

In the case of the lungs, (*a*) inflammatory lesions may prepare the soil for the growth of tubercle. This is particularly shown by the liability of tuberculosis of the lungs to follow pneumonia and the broncho-pneumonia of measles and whooping cough. (*b*) Chronic irritation from the inhalation of dust particles (pneumoconioses). For instance, silica predisposes to tubercle in the following way. After inhalation it becomes converted by hydration into colloidal silicic acid. Mineral silicates also become decomposed by the $CO_2$ of the tissues into silicic acid. The latter causes a granulomatous mass to be formed, consisting of a central area of necrosis, surrounded by inflammatory layers. Tubercle bacilli, present in such small numbers that the tissues would ordinarily deal with them, multiply rapidly in the central area, become too many for the local defences and so spread to other parts (**15**) (*c*) Lack of blood supply, as in congenital heart disease, are also factors predisposing to phthisis. On the other hand, it is noteworthy that in mitral stenosis, where the lung is chronically congested, phthisis is very rare.

The *age* of the individual is an important factor. Tuberculous invasion is especially common in young persons: in infants and young children the meninges, peritoneum, lymph glands, bones, and joints are attacked; in young adults pulmonary tubercle is common. Persons over forty are more rarely affected for the first time with tubercle, though tuberculous lesions may persist in them up to the ages of fifty, sixty, or seventy.

3. Tubercle bacilli may enter the system by *breach of surface of the skin*, by the *genito-urinary tract*, by the *respiratory* passages, and by the *alimentary canal*.

The first two modes are rare and of no practical importance; but in the past persons performing post-mortems have sometimes had their hands infected, contracting a post-mortem wart (*verruca necrogenica*), in which tubercle bacilli are found. Butchers occasionally contract similar lesions. They are very slowly progressive, and the tubercle very rarely spreads to other organs.

There has for a long time been a controversy as to the relative frequency of infection by the *respiratory* and *alimentary* tracts; the former is now held chiefly responsible. To answer this question we must first of all consider whether bovine or human tubercle is the commonest. (1) *Bovine* tubercle may be contained in milk from tuberculous cows and in tuberculous meat. The latter is not an important source of infection, because, apart from stringent regulations about its sale, meat is never eaten uncooked in this country. Infection by bovine tubercle will necessarily take place by the alimentary canal. (2) The researches of Cornet discovered the chief agent in the diffusion of *human* tubercle. It is not the air expired by the phthisical patient, but the sputum, which may be loaded with the specific micro-organisms. If this is repeatedly ejected on to the floor of a room and allowed to dry, or if quantities of it dry upon handkerchiefs,

the air of the room may at length be sufficiently impregnated to become dangerous to healthy people breathing it. From the floor and walls of rooms formerly tenanted by phthisical people Cornet obtained bacilli by the inoculation of which he produced tuberculous disease in healthy animals. This helps to explain the deadly influence of deficient ventilation in workshops, factories, barracks, and similar institutions. Human tubercle bacilli may thus be readily inhaled; but it does not necessarily follow that infection will take place by the lungs, because it has been argued that the bacilli and other dust particles are caught up in the mucus of the bronchi, are carried upwards by ciliary action or by coughing, and reaching the mouth, are eventually swallowed, and so produce infection by the alimentary tract. However, experiment has shown that dust particles and bacteria can readily penetrate by inhalation to the inmost recesses of the lungs, and further that guinea-pigs and cattle can be more readily infected in inhalation experiments than when equal doses of bacilli are ingested. Again, in the industrial disease known as silicosis, which strongly predisposes to tubercle, the particles are obviously inhaled, because the lung and bronchial glands may be quite solid, while the mesenteric glands are hardly affected. It is not denied that when dust is swallowed particles may reach the lungs and bronchial glands, but if swallowed, it could hardly collect in the lungs in such quantity as occurs in this disease without invading the mesenteric glands and lymphatics to a considerable extent.

Several careful observations have been made on the frequency of bovine and human tubercle in different types of the disease and at different ages, the bovine variety being distinguished by its greater virulence on injection into rabbits. Bovine tubercle accounts for rather less than 1·5 per cent. of phthisis cases. On the other hand, in tuberculosis affecting the cervical and axillary glands 73 per cent. are due to bovine tubercle in children under ten, and about 30 per cent. in children over ten. Again, in tubercle of bones and joints, the percentage of bovine cases was found to be 28 per cent. under five years, 25 per cent. between five and ten, and only 9 per cent. between ten and sixteen (Griffith). Bovine infection is responsible for an average of 18 per cent. of genito-urinary tuberculosis in England (**16**). Fraser found a considerably higher percentage of bovine tubercle in surgical tuberculosis at Edinburgh, viz. 61·2 per cent. It is probable that variations occur in different localities. Tuberculous meningitis in children is most commonly secondary to caseous bronchial glands, and it has been found that over 90 per cent. of such cases are due to a primary tuberculous focus somewhere in the lungs (Ghon, Canti). This argues in favour of tuberculous meningitis being in most cases due to human tubercle, and this has been found to be so (Park and Krumwiede). In this connection mention must be made of some further work by Eastwood, F. Griffith and A. S. Griffith on thoracic tuberculosis in children. These authors found that out of sixty-six cases when the anatomical evidence was strongly in favour of infection by inhalation sixty-five were due to human and only one to bovine tubercle. Out of twenty-three cases where the anatomical evidence was in favour of infection having taken place through the alimentary canal, eighteen were due to bovine and five to human tubercle. The conclusion from these figures is clear; it is that tuberculosis in adults is almost exclusively of the human variety, but in young children a fairly large proportion may be due to bovine tubercle, and this will usually be the case if anatomical considerations point to infection by the alimentary tract. Further, from the relatively favourable course of cervical gland and abdominal tuberculosis in children we may draw the conclusion that bovine tubercle in man produces a milder disease than human tubercle.

The importance of infection from a human source has been shown in another way by Ward, working in rural and semi-rural districts in South Devon. In two series of all types of tuberculous cases at all ages 60 per cent. had been in contact with other tuberculous cases, whereas in a series of control non-tuberculous

people only 12 per cent. had been in contact with tuberculous cases. The occurrence of conjugal tuberculosis pointed in the same direction: in 60 per cent. of cases where the husband or wife was tuberculous the other partner also had tubercle.

Mention must be made of the *auto-infection* theory of tuberculosis. It is that, since the majority of children have had the disease, any future manifestation is due to the old disease breaking out again. While not denying that this may occur, especially when the infection in childhood has been severe, the probability is in favour of the disease in adults being a *re-infection* in most cases.

**Morbid Anatomy.**—An elementary inflammatory focus caused by the *Bacillus tuberculosis*, which is too small to be seen by the naked eye, has the following structure: externally *lymphoid* cells, within these *epithelioid* cells, and in the centre a *giant cell*, with several nuclei. These cells are connected together by a delicate protoplasmic network, and the characteristic *bacilli* are present in the body of the giant cell or lying between the epithelioid cells. Sometimes the giant cells are absent, sometimes also the epithelioid cells, so that the tubercle may consist only of lymphoid cells. As the tuberculous focus enlarges the cells undergo a process of coagulative necrosis from deficient vascular supply —for no vessel penetrates within the focus and as a result of some chemical substance secreted by the bacilli.

A tubercle in its earliest stage, known as a *miliary* tubercle, is a translucent pearly grey nodule about the size of a millet seed, consisting of a central core of necrotic material surrounded by elementary foci. As it enlarges it becomes opaque yellow and cheesy in the centre, while at its periphery the tubercle may be invading more and more of the organ in which it is situated, the new tubercle becoming cheesy in its turn. In this condition it is known as a *caseous* tubercle. Under the microscope the caseous material shows shrunken cells, fat granules and *débris*. In the solid organs, large, spherical caseous masses are formed, as may be seen in the brain and spleen. If the disease invades a surface, vascular tuberculous granulation tissue is formed, similar to ordinary granulation tissue, but containing large mononuclear, epithelioid and giant cells, and sometimes well-formed tuberculous foci. Ulceration readily occurs. This may be seen typically in the intestine.

Some caseous masses ultimately undergo *calcification* (largely by formation of calcium phosphate), the tubercle bacilli are destroyed, and the lesion ceases to be infective. Another way in which tubercle terminates is by a *fibroid change*; chronic inflammation and induration of the surrounding tissue take place, and the tubercle itself shrinks into a fibrous nodule. This is more common on the surface of the pleura and peritoneum, but also takes place in the lungs.

**Pathology.**—In the great majority of cases the tuberculous invasion is *localised* to one part of the body, and only slowly spreads either by extension in the tissue it invades or by means of the lymphatics to other parts in the neighbourhood, especially the lymphatic glands. This localisation is a satisfactory indication of the resistance of most people to the infection. The results of tuberculous disease in each particular organ of the body will be described separately (*see* Phthisis, Tuberculous Peritonitis, Tubercle of the Kidney, etc., etc.).

A *general infection*, in which tubercle appears at the same time in several organs of the body, is nearly always preceded and caused by some local lesion in the lungs, bones, lymph glands, genito-urinary organs, or other part. It is known as *general* or *miliary tuberculosis*. It is due to general dissemination of the infection by the blood stream, with the formation of miliary tubercles or minute tuberculous foci in any organ of the body. Such a spread may be due in a few cases to tuberculosis of the thoracic duct; but in the great majority a vein has been found eroded by a tuberculous mass, so that caseous material has been discharged into it, and quite suddenly the body has been flooded with numerous tubercle bacilli. These have been found in the blood after death and during life.

This invasion is characterised by severe constitutional symptoms, presumably due to intoxication, particularly high temperature and rapid pulse. Sometimes several of these invasions may occur in a case, and this will be shown after death by the different sizes of the miliary tubercles in the various organs. Miliary tubercles are not large enough to interfere obviously with the functions of most organs. There are, however, two notable exceptions. Miliary tuberculosis of the meninges leads to interference with the functions of the brain, and to a less extent the same condition of the lungs interferes with its functions. The consequence is that, unless these organs are affected, miliary tubercle cannot be diagnosed with any certainty. It may be found accidentally at post-mortem in a case where the symptoms have been ascribed solely to the primary lesion, whether in the lungs, as in phthisis, or in the kidneys, etc. For this reason it will be best to describe miliary tuberculosis and tuberculous meningitis together, the distinction between the two being noted in the appropriate places.

## GENERAL OR MILIARY TUBERCULOSIS AND TUBERCULOUS MENINGITIS

**Ætiology.**—This disease occurs at all ages, but is generally regarded as more frequent in children than in adults; and it certainly affects males more than females. So far as its causation is concerned, it is constantly associated with and secondary to more advanced tubercle elsewhere in the body. It may arise in the course of phthisis, hip joint disease, caries of the spine, or other tuberculous complaints. Many cases, especially in infants and children, seem to arise in persons previously quite healthy, or at most after a few weeks' malaise; but, particularly in these instances, after death some older lesion is found, caseating bronchial glands, less commonly caseous mesenteric glands, or other hidden focus.

**Morbid Anatomy.**—In miliary tuberculosis one finds the most typical examples of tubercle. Through the *lungs* the tubercles are, as a rule, uniformly scattered more or less thickly. Every form of tubercle may be seen, from small grey dots to larger caseating tubercles; and sometimes these last may be breaking down in the centre, forming minute cavities. Definite patches of pneumonic consolidation occur, but are not common. Some inflammation of the bronchi, especially the smallest, is always present. Tubercles are sometimes found on the pleuræ, and pleurisy is often the result. In cases grafted on a former phthisis consolidation and cavities will also be present. In the other organs mentioned tubercles are also found, of different ages in different cases.

The characteristic appearances seen in the *meninges* consist of the presence of tubercles and of the effusion of lymph between the pia mater and the arachnoid. The lymph, which is gelatinous and translucent, or opaque and grey, or greyish yellow, but never purulent, consists of exuded fluid, fibrin, and lymphocytes in varying proportion. It is seen especially at the base of the brain, over the optic chiasma, the diamond-shaped space behind it, and the adjacent crura and pons. From this central point it commonly extends into the Sylvian fissure on each side, along the course of the middle cerebral artery, where it may be very abundant. The surface of the hemispheres is commonly free from lymph, or is at most a little dull or sticky, so that tuberculous meningitis is often called a *basal* meningitis; but it is common to find a small patch of lymph at the top of the cerebellum, at the anterior part. With the lymph are commonly mixed tubercles, varying from mere points up to the size of millet seeds, and occasionally beginning to caseate. The tubercles are especially abundant on the branches of the Sylvian arteries and on the pia mater. By separating the arteries and pia mater from the brain, and floating them in water, the tubercles may be seen as minute white dots. Under the microscope the smaller tubercles present aggregations of lymphoid corpuscles in the perivascular sheath; the larger tubercles may present all the characteristic features; but usually giant cells are absent.

The relation of the inflammatory lymph to the tubercles is very variable. There may be abundant lymph in the characteristic situations, with few, if any, tubercles discoverable; there may be a good number of tubercles with very little lymph. The ventricles of the brain are commonly distended with fluid (whence the old name *acute hydrocephalus*); the convolutions are flattened against the skull; the brain tissue is soft and often obviously congested. The cranial dura mater is not usually affected, but the spinal dura mater sometimes shows minute tubercles, and the lymph in the pia mater may extend to the cervical region of the spinal cord.

Miliary tubercles are also usually seen in and on the surface of the liver, kidneys, and spleen; while less often the choroid of the eye, the heart, thyroid, bone marrow, and peritoneum are involved. Any organ of the body may be affected, and no organ is invariably so. Sometimes the meninges may be apparently to the naked eye alone affected, but it is probable that microscopic examination would in such cases disclose evidence of infection in other organs as well.

**Symptoms.**—These will first be described as they occur commonly in children with meningitis. There is often a prodromal stage, during which the child is out of health, restless, loses appetite, gets thin, may be occasionally sick, and has constipation. The illness begins more definitely by headache or vomiting, or perhaps a convulsion. The headache is severe and continuous, with exacerbations from time to time; the child puts its hand to its head, and may be often crying, "Oh, my head!" or simply whining, or moaning, or occasionally uttering a sudden short shriek. With this there is a moderate degree of fever, quick pulse, excessive sensibility to light and sound, so that the child shuts the eyes, and desires to be left alone in bed; it resents being disturbed, and often curls itself up in bed away from intruding friends. The vomiting does not generally last long. If the illness begins with a fit, this is not often repeated. Occasionally there is squint, and there may be diplopia quite early.

After a few days, still with severe headache, there may be slight delirium, and the patient becomes drowsy. The head is sometimes retracted, and the neck is stiff; the abdomen becomes hollowed or retracted, the outlines of the muscles are obvious through the skin, and the margins of the ribs and the iliac crests are prominent. For this the terms *carinated* and *boat-shaped* are sometimes used. Kernig's sign is sometimes present (*see* p. 732). The pulse may be slow, and is often irregular; the respirations are slow, sighing, and irregular; the temperature is still generally high, or oscillates between 101° and 103°, but it may be extremely irregular, and sometimes the *typus inversus* is present, the morning temperature being high and the evening low. The ready occurrence of vasomotor paralysis is seen in the flushing of the face and the production of patches of redness wherever pressure is for a time applied. When the finger is drawn sharply across the skin of the forehead or abdomen, a broad red line quickly appears, and may persist five minutes or more. This condition, which is not peculiar to, but only more marked in, meningitis, is called *tache cérébrale*. Even as early as this, changes may often be observed in the optic disc, which at first becomes highly vascular, and then shows definite optic neuritis. Tubercles are seen in the choroid in a small proportion of cases. Usually some pulmonary symptoms are present; there is cough, and râles may be heard in the chest. Yet it is remarkable how full of minute tubercles the lungs may be without producing any physical signs.

From this point the case may steadily go on to a fatal termination without any fresh symptom. Food is taken badly, and the bowels are constipated. The drowsiness increases to coma, optic neuritis is more pronounced, the abdomen becomes more and more hollowed, the pulse more irregular, feebler, and generally quicker, and Biot's respiration may be seen (*see* p. 189), and the temperature may fall more or less rapidly, or just before death go up quickly to 106° or 107°. Mucus accumulates in the bronchial tubes, and with failing pulse death takes place.

But often the last two or three days are marked by local symptoms. An arm or leg, or an arm with the leg of the same side, becomes either rigid or paralysed, or there is slight facial paralysis, or squinting, or ptosis. The pupils are frequently unequal, and one or both may be insensitive to light. Frequently this stage is marked by convulsions, and these may recur several times before death. With the development of these symptoms coma becomes more profound, and death takes place, as above shown, or the patient is asphyxiated in a convulsion. Sugar is sometimes found in the urine in the last few days.

The illness lasts from ten days to three weeks, counting from the beginning of pronounced symptoms; but occasionally it may be four, five, or six weeks. The above course of the disease has been divided into three stages: a stage of *irritation*, one of *compression*, and the last a *paralytic stage*. But it is not always easy to distinguish between them, and in some cases the more typical symptoms may be very little marked, coma alone being prominent.

In the tuberculous meningitis of adults the symptoms are often much more rapidly developed. The onset may, of course, be masked by those of the disease already existing, *e.g.* phthisis. The patient may, with very little warning, become delirious, and have paralysis of a limb or of the face, or have a fit, quickly becoming comatose, and dying within a few days. Retention of urine has sometimes been observed as the first sign of meningitis in such cases.

The cerebro-spinal fluid in the early stages is quite clear, though it may be

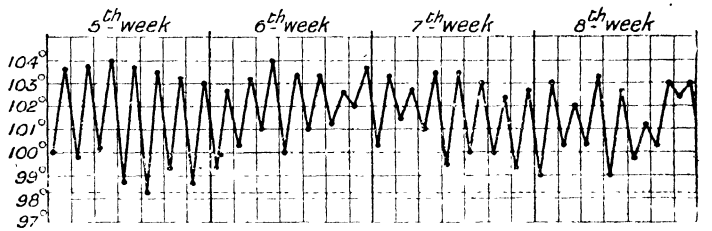



FIG. 11.—Chart of a Case of General Tuberculosis, fatal at the beginning of the Tenth Week.

under considerable pressure. Later it is faintly turbid, but never purulent. It contains an excess of lymphocytes, and sometimes tubercle bacilli may be detected. The blood is described as showing a leucopenia with a relative increase in polymorphonuclear leucocytes (Matthes).

In cases of general tuberculosis without involvement of the meninges, or when this occurs only in the last stages, the symptoms may be very obscure most of the time. The patient complains of weakness, inability to do his work, loss of flesh, anorexia, nausea or sickness, and headache. The bowels may be constipated or occasionally loose for a few days. Irregular pyrexia is present (*see* Fig. 11). The pulse is rapid and feeble and the tongue dry. In proportion as the course is prolonged the tubercles in the lungs grow, break down and produce prominent symptoms. There are cough, dyspnœa, scanty mucous expectoration, tinged, it may be, with blood, and sometimes pain in the side. The physical signs are at first suggestive of bronchitis. Resonance is but little affected; there may be a slight impairment of one apex, or, on the other hand, some increase of resonance over the whole chest. With the stethoscope one hears sibilant and sonorous rhonchi, fine and small râles, of which many are consonating. Only occasionally one gets scattered patches of high-pitched breathing or obscure dulness. If the condition is secondary to an old phthisis, the signs of this will, of course, be observed at the same time. When these conditions are well marked the patient presents a high degree of cyanosis, the face, lips, nose, ears, and cheeks being livid, and the fingers shrunken and blue. Death at length takes place, after from

three to eight or ten weeks, with increasing dyspnœa, lividity, prostration, and drowsiness. Meningitic symptoms may supervene at any time.

**Diagnosis.**—1. *Pre-meningitic Stage.*—In the earlier days, the absence of any other symptom than fever gives little grounds for a certain diagnosis, and almost any one of the disorders causing prolonged pyrexia (*see* p. 23) may have to be considered. The insidious beginning and the comparatively rapid prostration with a febrile illness easily lead one to confound this disease with *enteric fever*, and the symptoms of bronchitis, as long as they are moderate, rather increase the difficulty. The points in favour of enteric fever are rose spots, typical diarrhœa, distended abdomen, and a successful Widal reaction (*see* p. 16); those in favour of miliary tuberculosis are rapid emaciation from the first, irritability, the mixed pallor and cyanosis of the face as contrasted with the pink flushed cheek and white face of enteric fever, and the rapidity of breathing out of proportion to the other signs of illness. The occurrence of any cerebral symptom should at once give the clue to the nature of the disease. The fundus of the eye should be examined for tubercles, though they are present in only a minority of cases; optic neuritis is less likely to give assistance, since it is not generally seen before the onset of the cerebral symptoms themselves, and, moreover, occasionally occurs in enteric fever. The pronounced pulmonary cases are more likely to be confounded with *bronchitis* or *broncho-pneumonia*. High fever, rapid emaciation, and marked cyanosis distinguish acute tuberculosis from simple bronchitis. Cases of broncho-pneumonia present greater difficulties; the physical signs, the remittent pyrexia, and some cyanosis are common to both. Generally the shorter duration, or the presence of rather decided areas of consolidation, will point to broncho-pneumonia; but broncho-pneumonia may last long enough to be mistaken for tuberculosis. When a child has pulmonary complications after whooping cough it is often very difficult to distinguish between broncho-pneumonia and tuberculosis; in this case it is more common to diagnose the former disease or to overlook the fact that the latter may be there. It may be suspected if the symptoms are prolonged for several weeks with increasing cyanosis and wasting.

2. *Meningitic Stage.*—The characteristic features in tuberculous meningitis are, the insidious onset, pulmonary symptoms when present, headache, irritability leading on to drowsiness and coma, and the early involvement of the cranial nerves; choroidal tubercles when present are pathognomonic. The disease must be distinguished from other forms of meningitis and so-called meningism and other forms of intra-cranial disease, such as tumour, abscess and thrombosis of the cerebral sinuses. There is no real difficulty, because the cerebro-spinal fluid in tuberculous meningitis, with its increase of lymphocytes, is quite characteristic. In many other types of meningitis the onset is abrupt, as in cerebro-spinal fever, or there is an obvious primary focus, as in suppurative meningitis following otitis media or other condition. The cerebro-spinal fluid is definitely turbid and contains polymorphonuclear leucocytes. Pronounced opisthotonus and a marked Kernig's sign are more in favour of cerebro-spinal than of tuberculous meningitis. Acute poliomyelitis or encephalitis lethargica might cause a difficulty; in the former disease the onset is abrupt. In cerebral tumour and abscess the temperature is not usually much raised, and the pulse may be slow.

Another condition that may simulate meningitis to a certain extent is the *exhaustion* following upon malnutrition, bad feeding, or severe diarrhœa in quite young infants. The child is drowsy or comatose, with pale face, sunken eyes, dilated, irregular pupils, and irregular, sighing respiration. It is distinguished from meningitis by the history, the absence of fever and local paralysis, the depressed fontanelle, and the speedy improvement under restorative and supporting treatment.

**Prognosis.**—Tuberculous meningitis is a very fatal disease, but un-

doubtedly a few cases in which tubercle bacilli have been found in the cerebro-spinal fluid have recovered. But these few cases do not alter the absolutely unfavourable prognosis that should be given when once the nature of the disease is known. On occasions there may be for a time a remission of symptoms. Much the same thing applies to miliary tuberculosis without meningitis.

**Treatment.**—This should be directed to the relief of symptoms. Lumbar puncture is the most effective measure; it relieves the headache, and temporarily alleviates the symptoms, and may be performed daily. Of internal remedies iodide of potassium is often given in doses of 3 or 5 grains to children, and the bromide in similar or larger doses, or chloral may help to allay the pain in the head.

## LEPROSY

(*Lepra, Elephantiasis Græcorum*)

A chronic infectious disease characterised by nodular lesions of the skin, mucous membranes, and nerve trunks. Bacilli (*B. leprœ*), first described by Hansen, are constantly present in the affected tissues. They closely resemble tubercle bacilli and are acid-fast, but differ in some colour reactions, and in always being straight. The bacilli may be latent in the tissues for many years before the disease breaks out.

**Ætiology.**—The disease is not common in Europe, except in Norway, and is, as a rule, only seen in England in the case of patients who have lived in the East or West Indies. But it is found in some parts of South Europe, and, amongst other places, in India, Burmah, Siam, China, Japan, the north-east of Africa, the Cape of Good Hope, the West Indies, Mexico, Central America and parts of South America, and many islands of the Pacific. It is, however, not peculiar to one kind of climate or soil. Males are more often affected than females, and the disease is commonly contracted in early life, before the age of thirty, and rarely in infancy. Thus in the Philippines 44 per cent. of children living for seven to ten years with leper parents became infected (Denny). The firm belief that it is contagious has for centuries influenced the customs of the countries in which it is prevalent; and though nurses have lived in leper institutions for years with impunity, because elderly people are not very susceptible to the disease, direct transmission has sometimes been proved, the disease has been successfully inoculated in man, and its spread in new countries after the arrival and settlement of a leprous person has been observed even in modern times, as in New Caledonia and Mauritius. It is not congenital, and heredity seems to have but little share in its occurrence.

**Pathology.**—Infection takes place from a case of leprosy directly through the skin, at the site of some previous abrasion. The patient may have injured his skin by scratching or may have been bitten by insects. In many cases infection begins in or near the nose. It is doubtful whether the bacilli have ever been conveyed directly by insects. The primary lesion occurs in the lymph spaces of the papillæ of the corium beneath the epidermis. There follow two possibilities: (1) While there are still too few bacilli to be seen, they spread up and multiply in the superficial nerves of the skin; (2) The bacilli multiply in the papillary lymph spaces and spread downwards between the hair follicles and below them and also laterally. As the bacilli multiply they secrete a glutinous substance which keeps them fastened together in one mass and the skin loses its pigment. This is known as the *quiescent* phase. After a variable time we may get the *reactionary* phase, *i.e.* inflammation sets in, the skin vessels become dilated, the glutinous matrix gets broken up and bacilli with their toxins may get carried throughout the body by the blood stream. This may produce a general infection of nerve trunks in the skin, or scattered lesions in the corium may form similar to that just described. This inflammation may be caused by the injection of certain drugs,

by counter-irritation, and by various febrile diseases. During this phase the bacilli may be seen to be altered; some have a dotted or granular appearance; others form spores, because the conditions for their growth are not as suitable as before. The erythema and cellular infiltration cause a swelling of the skin, and the intrapapillary processes of the epidermis become flattened out and the hair follicles compressed. The cells vary in size from that of a leucocyte to three or four times this size and may contain several nuclei (*lepra cells*). Finally, we get the stage of *resolution*, when the erythema disappears, and the skin looks like crushed tissue-paper, because it is thin and wrinkled after the previous swelling and inflammation. The cellular infiltration is now replaced by fibrous tissue. Leprous lesions are constantly passing through these three phases, and they may all be present in a single spreading lesion. In this case we see an outer zone free from pigment; next an inflammatory erythematous zone, somewhat pigmented; and in the middle a scarred area (**18**).

Leprosy tends to confine itself to the skin, mucous membranes and peripheral nerves, though in fairly advanced cases the bacilli are found in the internal organs, which may be affected by lardaceous degeneration. When the nerves are attacked, they are often thickened to two or three times their normal size from proliferation in the sheath, by which finally the axis cylinders may be atrophied or destroyed. Nodular infiltrations also form, which can be felt under the skin in the case of superficially placed nerve trunks.

**Symptoms and Course.**—The lesion of the skin first shows itself as red or brownish-red spots on the limbs or trunk (*Lepra maculosa*). They may be from ½ inch to 3 or 4 inches in diameter, are round or irregular in shape, and slightly swollen. Sometimes they form rings by clearing up of some spots at their centres, as described under the heading Pathology. Finally, they may fade, but are apt to leave pigmented stains, or sometimes white spots, behind them; and from time to time fresh outbreaks of spots occur.

The characteristic feature of *Lepra tuberculosa* or *L. nodosa* is the occurrence simultaneously with the spots, or after an interval, of a nodule, tubercle or *leproma*, *i.e.* a hard elevation of the skin from the size of a pea to that of a hazel nut or larger. Such nodules may persist for a long time, and may ultimately disappear, leaving pigment spots behind, or they may soften, break through the skin, and leave indolent ulcers, having weak granulations, and discharging a scanty thin pus. The nodules appear mostly on the face, on the dorsal surfaces of the hands and feet and on other parts of the limbs. On the face they are apt to produce great deformity by the enormous thickening of the eyebrows, the nose, cheeks, and lobes of the ears; and the very characteristic appearance thus produced is described as *leontiasis*, from its resemblance to the face of a lion. The eyelids are often involved, and by ulceration of the tubercles there is extension to the coats of the eyeball; but the optic nerve, retina, lens, and vitreous usually escape. The nodules also develop in the mucous membranes of the mouth, the gums, the palate, the larynx, or the nose; and the voice may be rough and hoarse, or high-pitched and feeble, in consequence. The ulcers may eat deeply into the parts beneath, so as to erode tendons and bones and open the joints. In advanced cases, the blood shows deficiency of red cells and of hæmoglobin, with a low colour index, poikilocytosis, polychromatophilia, and sometimes an excess of eosinophils.

In *Lepra anæsthetica*, which is due to involvement of nerves, nervous symptoms predominate. In the early stages pain and tingling and burning sensations are felt in various isolated patches on the limbs and trunk, especially in the course of the ulnar and peroneal nerves. Later numbness and actual anæsthesia to touch and pain are observed in irregular areas; and over these patches the skin is either paler than normal or more pigmented, the hairs are small and wanting in pigment, and the skin generally is smooth and glistening. After a time the exposed nerve trunks, such as the ulnar and peroneal, are felt to be

thickened. The muscles in the same areas, especially the interossei of the hands and feet and the muscles of the forearms, and even the bones, become atrophied, and clawhand, dropped wrist and dropped foot may result. In this anæsthetic form also, deep ulcerations take place over the joints of the fingers and toes, and the phalanges, or carpal or tarsal bones may ultimately be shed, the terminal phalanges, it is said, often being spared, and the wounds sometimes healing up with remarkable completeness (*Lepra mutilans*).

Mixed cases are not uncommon, in which nerve lesions and skin lesions co-exist; but there is a tendency for nerve lesions to show themselves when the skin lesions are healing, and *vice versâ* for the symptoms of the nerve lesions to disappear when the skin lesions are spreading. The symptoms of the patient depend on the stage of the disease. In the quiescent phase he may feel quite well and be able to carry on his work, in spite of his harbouring millions of bacilli. In the reactionary phase there will be malaise and fever. When the phase of resolution appears, it means that the patient has gained some immunity and that the disease is becoming healed. Contrary to prevailing opinion in the past, leprosy is really a self-healing disease, though it may run a course of many years with exacerbations and remissions. The majority of lepers segregated in institutions have reached the final stage of their illness. They are healed, though they suffer from deformities. They harbour no living bacilli, and they are no longer infectious. Leprosy itself does not kill; but death may occur from tuberculosis of the lungs, nephritis, dysentery, secondary infections causing gangrene or pyæmia, or other intercurrent affection, and occasionally from obstruction of the larynx, the more direct result of the leprosy.

**Diagnosis.**—Leprosy must be differentiated from tuberculosis and syphilis, and these diseases may co-exist with it. The Wassermann reaction does not help, as it may be positive in leprosy. The presence of anæsthesia associated with a skin lesion, or in some area of skin, is an important indication. It is rarely completely absent in leprosy. Early deposits are often felt on the forehead or naso-labial folds. The bacilli can be sought in the serum of a nodule, in the pus from an ulcer, or in the nasal mucus. In the male the nipples are usually enlarged.

**Prognosis.**—Nodular leprosy is apt to be a more acute and serious disease than nerve leprosy. It has been suggested that the prognosis has been greatly improved of late, with new methods of treatment, which can be used in all stages of the disease. A patient who has passed through the third stage of the disease may be considered cured if over a period of two years there has been no increase in number or in size of the anæsthetic lesions and repeated bacteriological examination has given negative results. If skin lesions in the first and second stages of the disease have disappeared without treatment, or if anæsthetic patches have largely disappeared and the bacteriological examinations have been negative for two years, the case may be regarded as "arrested" but not absolutely cured.

**Prevention.**—Nodular leprosy, especially if there are discharges from the nodules or the nose, is the most infectious stage of leprosy, and it is essential to isolate such cases from children and persons under thirty years old, who are very susceptible. Most lepers in asylums have passed through the third stage and are not infectious. Compulsory powers of isolation are necessary, and at the same time the knowledge of new methods of treatment should be generally diffused, so that patients may be induced to declare themselves.

**Treatment.**—Patients must be placed in healthy surroundings, given good food, and the bowels kept freely open. Scrupulous cleanliness is essential, and the patient must avoid overstrain and exposure to bad weather. It has been suggested that the lepra bacilli may be destroyed by injecting the esters of the fatty acids of hydrocarpus and chaulmoogra oils into the subcutaneous tissue under the skin lesions and anæsthetic patches, or by painting trichloracetic acid

in distilled water (1 in 3) over the lesions every six or ten days. The principle of the treatment is to make the leprous lesions pass through all three stages as quickly as possible. The fatty acid esters act by stimulating the tissues to react by their irritant action, so that the bacilli are destroyed. They may be freely used in the first and third stages of the disease, but great circumspection must be used in the second stage, when the tissues are already reacting. Trichloracetic acid is especially valuable here. In the third stage reactions are more difficult to obtain and large injections may be made. If potassium iodide is given by mouth in doses increasing from ½ grain, many patients will react more easily. It must be given with great circumspection. The ethyl esters of chaulmoogra oil in the form of a preparation called *moogrol*, in doses of 1 to 5 or 6 c.c., are also injected intramuscularly into the buttocks, and favourable results have been reported.

## GLANDERS

(*Equinia, Malleus, Farcy*)

Glanders is a disease which affects chiefly horses, mules, and asses, though sometimes other domestic animals, and is occasionally transmitted accidentally to man. Grooms, stablemen, and others in charge of horses are most liable to contract the disease, which in its acute forms is a febrile disorder, characterised by special lesions of the nasal and respiratory mucous membranes, by the formation of subcutaneous nodes and the implication of the lymphatic vessels and glands, and by a cutaneous eruption. It also occurs in a chronic form. The term *farcy* was given to cases in which the subcutaneous nodules (farcy buds) with the lymphatic lesion were prominent features; but it is not desirable to have two names for one disease, and glanders is now the appellation generally adopted.

The disease is mostly transmitted to man by accidental inoculation of wounds, cuts, or abrasions, either in grooming a glandered animal or in skinning one dead of the disease, or a horse may bite its groom and convey the disease by means of its saliva, or may sneeze and discharge some nasal mucus into the eye, nose, or mouth of any one standing near. It is stated that it may be conveyed by eating the raw flesh of a glandered animal, and that it has been caught in this way in menageries. It may also be communicated from man to man. The bacillus of glanders (*B. mallei*) is found in the nodules; it is about the size of the tubercle bacillus, but is thicker and is decolorised in Gram's method.

**Pathology.**—On post-mortem examination in acute glanders the changes characteristic of pyæmia are often found: increased fluidity of the blood and abscesses of the lungs, the pyæmia being secondary to the local lesions.

The characteristic lesions of glanders are found in the mucous membranes, the skin, and the lungs. In the nasal mucous membrane, subepithelial nodules occur, from the size of a millet seed to that of a pea, consisting of lymphoid corpuscles or pus corpuscles. In a later stage these nodules have suppurated, and left ulcers with yellowish bases. Around these fresh nodules of infiltration have formed, which go through the same process. The septum may be perforated. If recovery takes place, irregular puckered scars are left. In the lungs, similar nodes form, the centres of which break down into a caseous detritus. These are accompanied by patches of broncho-pneumonia, which may form abscesses. Similar nodes form in the conjunctivæ, frontal sinuses, pharynx, larynx, intestines, skin, subcutaneous tissue, and muscles.

**Symptoms.**—*Acute Glanders.*—The disease begins with malaise, headache, lassitude, loss of appetite, and pains in the joints and limbs. For a time there is often a resemblance to rheumatic fever or enteric fever, or there may be pain in the side or dyspnœa. If a wound or scratch has been infected directly, it becomes inflamed, tense, and painful; and the skin around has the appearance of ery-

sipelas. The sore ulcerates, and discharges a sanious fluid, and the lymphatics in the neighbourhood may become enlarged. The more characteristic features of the disease may not appear for a week or more after its commencement, though sometimes earlier. The *eruption* consists of small red papules, upon which vesicles appear; these soon form bullæ, or pustules, of different sizes, up to ½ or ¾ inch in diameter, hemispherical, flat or depressed in the centre, with serous, purulent, or blood-stained contents. The base of the pustule is inflamed, and infiltrated for some distance round. After a time the discharge escapes, and an ulcer covered with scab or slough remains. The *nodes* which form under the skin are at first hard and painful, and generally suppurate. The lymphatic glands are not always inflamed. The implication of the mucous membranes is shown by a discharge from the nose, which is at first a thin mucus, but afterwards becomes thick, viscid, purulent, fœtid, and often blood-stained. It is connected with the formation of the tubercle-like nodules already described.

Usually the disease progresses steadily, with symptoms of a pyæmic character. The temperature is high, but may oscillate; the pulse is quick, and the tongue dry and brown. Albumin appears in the urine, low delirium with tremor is succeeded by coma, the breathing becomes more rapid, and death finally ensues, generally in two or three weeks from the commencement.

*Chronic Glanders.*—Here the local lesions predominate. They consist of ulcers with thick and hard edges, or abscesses about the joints, or inflammatory swelling beneath the skin or in the muscles. A pustular eruption may also occur, but it develops more slowly than in the acute form. The nasal mucous membrane may also be involved, and in some cases emaciation occurs, with hoarseness and pulmonary symptoms, such as cough and hæmoptysis. The average duration of the chronic cases is stated to be four months.

**Diagnosis.**—In early stages the disease may be mistaken for rheumatism or typhoid fever, and later for pyæmia. In chronic cases, syphilis, scrofula, and phthisis may be simulated. In veterinary surgery, the diagnosis is made by the injection into suspected animals of *mallein*, which consists of the chemical substances present in the artificial cultures of the glanders bacilli. If the animal is diseased, a definite "reaction" with rise of temperature occurs, similar to that produced in man by Koch's *tuberculin* (*see* Diagnosis of Phthisis).

**Prognosis** is very unfavourable. Only a few recoveries from acute glanders are recorded, and only about half of the chronic cases get well.

The **Treatment** must be supporting and stimulating. Quinine should be given internally; the nasal lesion should be treated with antiseptic injections, such as creosote, carbolic acid, iodine or potassium permanganate lotion. Abscesses of the skin should be opened when ready. For chronic cases carbolic acid, potassium iodide, arsenic, strychnine, and sodium benzoate have been recommended.

### MELIOIDOSIS

This is a rare disease, resembling glanders, and found in Burmah and the Malay States. It is due to the *Bacterium whitmori*, which closely resembles the *B. mallei*, and has been found in the blood, urine, sputum and cutaneous vesicles of dying patients. Numerous small pulmonary abscesses are produced. The disease begins with acute diarrhœa and collapse, resembling cholera. Then follow septicæmia and the pulmonary lesions, and the patient dies in ten days. In a more chronic form of the disease there are cutaneous abscesses and collection of pus in the lungs, liver and spleen.

## ANTHRAX

This term, formerly the Latin equivalent of *carbuncle*, is now generally used to designate a disease which affects various animals, and is communicated from

them to man. In animals it is known as *splenic fever ;* in man it includes *charbon* of the French, and *malignant pustule* of English writers. Its distinguishing feature is the presence of a bacillus (*B. anthracis*), which can be found in the local lesions, the blood, viscera, and secretions. This is a non-motile, gram-positive bacillus, varying from 5$\mu$ to 20$\mu$ in length—that is, considerably longer than the diameter of a blood corpuscle. The bacilli multiply by elongating and dividing. Outside the body they may produce spores within themselves, which subsequently become free. The spores have great vitality, and resist considerable changes of temperature ; they reproduce bacilli in a favourable environment.

Among animals this disease can be conveyed by direct inoculation, probably by the bites or stings of insects, or the bites of dogs that have eaten the flesh of animals dying of the disease. It is also transmitted indirectly by animals feeding in damp meadows or on moist soils, where the specific micro-organism contained in the dejecta of previously diseased animals may have been preserved in an active condition. Pasteur thought that the spores of bacilli multiplying around buried carcases might be carried to the surface by earthworms, but this was not confirmed by experiments instituted by Koch.

Infection in man occurs from the living animal, as in drovers, shepherds, and farmers, or from the carcase, and this is much more common. Thus slaughterers, butchers, and those who have to do with the hides may be infected through a scratch or wound, and rarely it may be contracted in eating the flesh of diseased animals. Most frequently, however, in England it occurs amongst tanners and those who have to handle the skins and hides that come from abroad, and among those who deal with wool and hair from the same animals. Thus wool-sorters, furriers, tanners, and others in like occupations may contract the disease either by direct inoculation through the broken skin, or by inhalation of dust or wool particles proceeding from the goods. Some cases have arisen from the use of shaving brushes, especially those of Japanese manufacture, made of animal hair infected with anthrax. Rarely it is transmitted from man to man by direct contact. Rag-sorters engaged in paper manufactories are subject to pulmonary anthrax, and the bacillus of anthrax has been found in the viscera.

**Anatomical Changes.**—In all fatal cases there may be found the changes indicative of acute septic disease : ecchymoses in the submucous and subserous tissues, in the substance of the heart, or in other muscles ; hæmorrhage or œdema of the lungs ; congestion and softening of the liver and kidneys. The spleen is not always enlarged. When the special *pulmonary* symptoms have been present, there are congestion of the mucous membrane of the trachea and bronchi, hæmorrhages into the lungs or under the pleura, swelling of the cervical and bronchial glands with hæmorrhage into or around them, fluid in the pleural cavities, and ecchymosis and gelatinous exudation in the neck and mediastinum surrounding the trachea and mediastinal glands.

In the *intestinal* form the peritoneum contains serum, which is often blood-stained ; there is semi-gelatinous infiltration of the mesentery and retroperitoneal connective tissue ; congestion and swelling of the mucous membrane and submucous tissues of the stomach and intestines in patches of $\frac{1}{4}$ inch to 1 or 2 inches in diameter, which are pink and fleshy on section, but on the surface discoloured, or excoriated, or covered with an adherent layer of blood. There are also submucous and subserous hæmorrhages, and the spleen and the mesenteric and lumbar glands are often enlarged.

Some cases of hæmorrhagic *meningitis* have also occurred.

**Symptoms.**—The different forms of the disease are local or external anthrax —*malignant pustule* proper—and *anthrax septicæmia*, which includes a pulmonary and a gastro-intestinal form. Either of the last two may be combined with the local variety.

*Malignant Pustule.*—Infection generally occurs through a scratch or abrasion on the face, neck, hands, or arms. After an *incubation* of a few days, or it may be

only some hours, the spot itches or burns, and a small patch of erythema appears, a little larger than an ordinary flea-bite with a minute vesicle on top. This gradually enlarges and bursts, discharging a thin fluid. The base of the vesicle then forms a brown or black eschar, and the skin around becomes red, swollen, and indurated, forming a prominence from 1½ to 2 inches or more in diameter. Around the central eschar there is often a ring of small vesicles containing serum, and the skin for some distance round may be œdematous, and the nearest lymphatic glands enlarged and tender. However, in most cases the pustule has not this characteristic appearance, but resembles vaccinia. For three, four, or five days the patient may feel in his usual health and continue at work; he then becomes feverish, with prostration, delirium, sweating, or diarrhœa; and finally, in many cases, death occurs, preceded by collapse. In *malignant anthrax œdema* no definite pustule forms, but an œdematous swelling, usually affecting the eyelids. It is otherwise like malignant pustule, and is mostly soon fatal.

*Anthrax septicæmia* varies in different cases. The early symptoms are generally restlessness, a sense of depression and exhaustion, and vague sensations in the limbs; then acute fever suddenly sets in with the usual symptoms, and, in addition, great prostration, embarrassed respiration, and rapid collapse. In the cerebro-spinal fever epidemic of 1915–16, the symptoms of this disease were simulated by five cases of anthrax septicæmia, the bacilli being found in the cerebro-spinal fluid (Reece).

In the *pulmonary* form difficult and laboured breathing with a sense of constriction, cyanosis, and great prostration, are the main features, without much cough or physical signs other than a few rhonchi and râles. The expectoration, if there is any, may be bloody. Delirium and coma may precede death, or the mind may be clear to the last. This is the *wool-sorters' disease* observed at Bradford and elsewhere.

In the *gastro-intestinal* form there are vomiting, abdominal pain, and diarrhœa, often with blood in the fæces, sometimes dysphagia and bleeding from the pharynx and mouth. Fever is slight, but dyspnœa and lividity, restlessness and convulsions of epileptic or tetanic character, precede the invariably fatal end.

**Diagnosis.**—The diagnosis of cutaneous anthrax is easy in quite an early stage, if only the practitioner thinks of the possibility of anthrax. All that is necessary is to examine a stained smear from the vesicle under the microscope. But it may be missed, because the lesion may be thought to be just a pimple. In a later stage it resembles vaccinia. In pulmonary and gastro-intestinal anthrax the diagnosis is much more difficult. But an unusual combination of symptoms, such as vomiting and joint pains, may arouse suspicion. Bacilli may be detected in the blood, vomit, expectoration, urine or cerebro-spinal fluid. But they are not generally to be found in the blood for some days. The diagnosis may be confirmed by inoculation of a rabbit, guinea-pig, or mouse with the secretions or with blood. The animal dies within two or three days with dyspnœa, dilated pupils, and, perhaps, convulsions; and the blood contains the characteristic bacilli.

**Prognosis.**—This is very unfavourable in cases left without treatment, the mortality being about 50 per cent.

**Treatment.**—In malignant pustule the correct treatment is complete surgical excision in the early stages; but this should not be carried out if the lesion has progressed much beyond the "flea-bite" stage. The part must be rested by means of splints or sandbags and antiseptic dressings applied. It is also important to administer anti-anthrax serum, such as Sclavo's, at the earliest possible moment. 60 to 80 c.c. are injected intravenously, and 60 c.c. on the day following, if there has been no reaction (shown by a rise of temperature) and the general condition has not improved. In default of serum, arseno-benzol should be used. In Bradford, where special precautions have been taken to make an early diagnosis, the mortality has been lowered from 16 to 5 per cent.

## FOOT-AND-MOUTH DISEASE

(*Aphtha epizootica*)

This disease of cattle and sheep is occasionally transmitted to man. The typical feature of the disease in cattle is the formation of vesicles and bullæ on the mucous membrane of the mouth, lips, and tongue. The affected parts become swollen, and the saliva dribbles away. The vesicles break, leaving a grey layer covering the base. Vesicles also appear on the feet round the borders of the hoofs, and they become pustular and produce crusts. In cows vesicles form also on the udders and teats. There is a moderate degree of pyrexia. The disease lasts about a fortnight, and generally ends in recovery, except in calves, of which from 50 to 75 per cent. die, some from enteritis.

The disease appears to be conveyed to man by direct inoculation, and by drinking milk from an infected cow; but it may also be carried from man to man in boots or clothing.

The organism is at present in doubt, and a recent claim of its discovery has been disproved. The virus will pass the finest filter. The *incubation* is from three to five days. Slight pyrexia and loss of appetite first occur; then vesicles are observed in the mouth, on the lips, tongue, fauces, and hard palate. They reach the size of peas, become opaque, break, and form shallow ulcers, with a dark red base. The lips become swollen, and saliva and mucus are more abundant than normal. Mastication, swallowing, and talking are somewhat painful. There may be some diarrhœa and abdominal pain.

Sometimes vesicles form on the fingers, especially about the nails; they become pustular, and run together; and similar vesicles occur on the toes, on the soles of the feet, and on the nipples of women. The duration is from ten days to a fortnight, and the disease is rarely fatal.

**Treatment.**—Washes of borax, of potassium chlorate, or of potassium permanganate should be used to the mouth, and painful ulcers should be touched with solid silver nitrate. Zinc or lead ointments or lotions to relieve itching should be applied to the eruptions on the fingers and toes.

## ACTINOMYCOSIS

Actinomycosis is the name given to a granulomatous lesion from which typical "sulphur granules," visible to the naked eye, are obtained. It is due to the ray fungus, which belongs to the streptothrix group (*see* p. 10), and is known as *Actinomyces bovis* (Wolff-Israel). In 1877 Israel, of Berlin, described the first cases in man, and in 1878 Ponfick showed the identity of the human cases with the cases occurring in cattle.

The actinomyces form masses which are visible to the naked eye as yellow, greenish-yellow, or grey, glistening, spherical, granular bodies, the "sulphur granules," mostly about ½ to 1 mm. in diameter, and consisting, under the microscope, of a central mass of closely woven mycelial threads and an outer layer of "club"-shaped bodies arranged radially and giving the appearance of "rays," from which the organism gets its name. The whole is encased with leucocytes. The organism grows anaerobically. Other organisms have been described producing granulomatous lesions in animals, and occasionally in man. But most human cases, at any rate, are due to the *Actinomyces bovis*. There are two theories as to its causation in man.

(1) Aerobic streptothrix organisms, growing at air temperature, have been isolated from grasses in marshy districts. It is supposed that man has been infected by eating straw, barley, etc., and in certain cases, particularly in stable men, actinomycotic infection has been found associated with a foreign body,

usually a barley corn. On the other hand, the streptothrix in man only grows at body temperature and is anaerobic, so that it must be different from the organisms found in straw.

(2) The *Actinomyces bovis* is a saprophyte in the mouth, growing, in particular, round septic teeth. It gains entrance owing to injury of the mucous membrane, and this may be caused by a foreign body, such as a barley corn or piece of straw. Further, there is clinical evidence that actinomycosis has followed the extraction of a septic tooth. Thoracic actinomycosis has followed the accidental disappearance of a tooth down the trachea, and in one case the knuckles have become infected in a patient who had knocked out an opponent's tooth with his fist.

This second theory of infection is the more probable.

Once introduced, the organism adheres to some point of the surface of the alimentary or respiratory passages, penetrates then to deeper parts, and forms local lesions in different parts of the body. These consist mainly of inflammatory changes, of more or less intensity, set up around the granules, so as to form slowly growing tumours, which ultimately suppurate, break down, and discharge. From the continued growth and multiplication of the parasite at one spot—as, for instance, in the liver—large tumours may be formed, three or more inches in diameter, consisting of a kind of cavernous tissue, the trabeculæ of which are

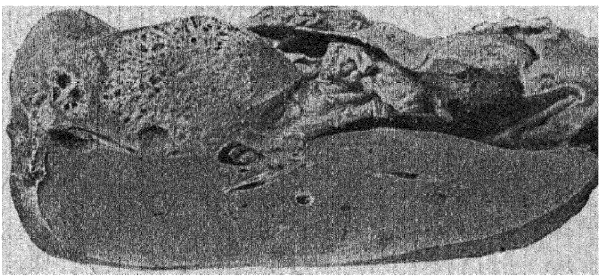


FIG. 12.—Sagittal section of a Liver showing the Lobulus Spigelii honeycombed by Actinomycosis.

fibroid, while the spaces contain pus, in which the yellow granular masses of fungus lie loose. A remarkable feature of the disease is the way in which lesions extend by contiguity from one tissue to the next over long periods of time; but occasionally the parasite is conveyed by the vessels to remote parts, and a more widespread deposit by *metastasis* occurs. It is characteristic that the lymphatic glands are not involved.

**Symptoms and Course.**—These depend on the seat of the primary invasion.

In many cases this is the *mouth*, when a tumour is generally first noticed under the skin over the lower jaw, or on the edge of the jaw. It is hard, does not affect the skin, is chronic in its course, varies in size from time to time, and tends to migrate gradually from the edge of the jaw down to the neck, leaving for a time a narrow band of firm tissue in its track. The tumour may shrink up in part, the inflammatory tissue cicatrising; but newer portions continue to form, and ultimately the skin becomes involved, obscure fluctuation is felt, and a thin, sero-purulent, odourless fluid, containing the sulphur granules, is discharged. A sinus is formed, which rarely closes, but continues patent with slight secretion. There is evidence to show that in these cases the parasite has entered by a carious tooth. The tumour may form in the substance of the lower jaw, and expand the bone. Invasion by the upper jaw results in tumours of the cheek or temple, and an extension to the base of the skull or the mediastinum by means of the

pharynx is a possibility which makes the implication of the upper jaw more serious than that of the lower. Penetration of the *œsophagus* has led to mediastinal abscesses and erosion of the vertebræ.

In *intestinal* actinomycosis, which involves most frequently the cæcum or the appendix, the mucous membrane presents on its surface patches of whitish material, covered with yellow and brown granules. The patches are about two-fifths of an inch in diameter and one-fifth of an inch thick, and adhere firmly to the membrane. The disease may also cause swellings in the substance of the intestinal wall, from which it may perforate into the peritoneal cavity, or, by means of adhesions, invade adjacent viscera or the abdominal wall at almost any point. The *liver* is often secondarily infected in intestinal cases, and then contains large prominent masses, having the structure above described. Clinically such tumours may present the characteristics of hepatic abscesses, with local pain, tenderness, remitting fever and rigors.

When actinomycosis affects the *lungs* the symptoms may be bronchitic or pneumonic. In the former case there may be a close resemblance to putrid bronchitis, the sputum separating into two layers, the upper clear and the lower turbid, the latter containing the ray fungus. When the substance of the lung is affected, pneumonia occurs in patches, the patients cough and lose flesh, and the expectoration is either thick and muco-purulent, containing the typical granules, or it may be viscid, translucent, and rusty, like pneumonic sputa. There is often a close resemblance to phthisis, but the posterior and lateral portions of the lungs are involved, not the apices; and the sputum is, of course, free from tubercle bacilli. If the inflammatory lesions reach the surface, they set up pleurisy or pericarditis. Effusion takes place, or the lung becomes adherent to the chest wall, which then becomes involved, and ultimately soft diffused inflammatory swellings appear on the chest, which may fluctuate, break, and discharge purulent fluid containing the fungus. From the lung also the inflammatory track of the organism may stretch through the diaphragm into the abdomen, or behind the diaphragm to the psoas and iliacus muscles, or between the ribs to the surface of the chest. In a case of this kind recorded by Pringle, in which the skin was secondarily involved, there were large, soft, fleshy, sarcoma-like growths on the back of the chest, of mottled, purplish-red and yellow colour, covered by very thin skin, and presenting small ulcerative openings, from which a sticky fluid oozed, and in which lay a purulent fluid containing actinomyces granules. These processes are commonly very slow, and are accompanied with varying amounts of fever in different cases. A primary infection of the *skin* is much rarer. The actinomyces is the cause of one of the white varieties of madura foot (*see* p. 143).

Infection by the *female genital tract* with extension to the ovaries and Fallopian tubes is also recorded.

**Diagnosis.**—The diagnosis may be suggested by the presence of indurated lesions which are nearly painless and slowly progressive, while surrounding tissue presents to palpation a special "wooden" resistance. The disease can only be positively identified by finding the sulphur granules in the pus, or on the granulating surface of the sinuses, or in the sputum and urine. Some care is required in looking for them. A few drops of pus may be shaken up vigorously in half a test-tubeful of water, fitted with a cork, when the granules will be seen on holding the tube up to the light. One of these may be then placed on a slide and lightly crushed with a cover glass, when a low power will show the characteristic radiating structure. On crushing further the mycelial filaments may be seen; they are stained rather irregularly by Gram's method. Finally, anaerobic cultures of the organism should be made. Actinomycosis of the brain has been detected by lumbar puncture, the fungus being recognised with the microscope in the sediment from the fluid.

**Prognosis.**—There were nine recoveries out of ten cases in which the face

or neck was affected, but the prognosis is bad in thoracic and abdominal cases (Colebrook).

**Treatment.**—Surgical measures should be adopted in the first place if possible, multiple incisions being made into the tumours and the granulating surfaces being thoroughly cleaned by rubbing with dry gauze swabs, in preference to a sharp curette, which injures the surrounding healthy tissue. Potassium iodide has a powerful influence in the treatment of actinomycosis, and very remarkable results have been obtained under its use. It should be given to the extent of 2, 3, or 4 drachms daily, and better at short intervals of two or three hours during the twenty-four hours than in two or three doses or at longer intervals (*see* p. 120). Vaccines and arseno-benzol are other remedies which have been employed. Deep X-ray therapy is of value.

## MADURA FOOT

This disease, the fungus disease of India, is met with in several parts of the world, but more especially in tropical parts.

It is due to the penetration into the foot of some form of fungus (Hyphomycetæ or Ascomycetæ), and its subsequent growth with resulting formation of granulation tissue, suppuration, and destruction of the soft parts and even of the bones. Early in the disease sinuses form and open on to the surface, discharging pus and granules (*sclerotia*), which are either black and dark brown, or white or yellowish white, according to the variety of the disease. The granules vary in size from that of a pin's head to that of a pea, and consist of masses of branching mycelial threads, which often have a radial arrangement. Several species of fungus have been described. The most familiar is the *Madurella mycetomi* or *Streptothrix maduræ*, of which the granules are black or dark brown, and from 1 to 2 mm. in diameter. One of the white varieties is really an actinomycosis, and is due to the same fungus. Experimentally the disease has been reproduced in monkeys' feet by inoculating the animal with the streptothrix from a typical case.

In man the foot becomes swollen; the plantar arch is filled up, and becomes convex, so that the toes are lifted from the ground; the skin darkens, and numerous nodules and openings appear upon the surface, from which escape pus and granules, and which lead into deeply penetrating sinuses.

The hand is sometimes similarly affected; but, except in the case of the actinomycotic form, the disease does not become generalised. The only remedies are excision of the tissue in the early stage or amputation of the foot when the disease is extensive.

## SPOROTRICHOSIS

In this form of disease the skin is infected, generally through a wound or scratch, by an organism of the order of fungi Hyphomycetæ, known as *Sporotrichum beurmanni*. The disease has not been often seen in England; it was first described in America in 1898, and many cases have occurred in France and other parts of Europe, in Brazil, Madagascar, and Ceylon.

A week or two after infection a number of pimples or small swellings appear on the arm, and spread to other parts of the body. They form hard pink nodules in the skin and subcutaneous tissue, from the size of a pea to that of an orange, and subsequently soften and ulcerate, or form abscesses. They may occur also in the mucous membranes, in the muscles, joints, epididymis and periosteum. Generally the lymph glands are not enlarged. The swellings present a close resemblance to syphilitic gumma or to tubercular nodules, and it may be impossible to distinguish them except by bacteriological methods. Microscopically a nodule consists of granulomatous tissue, containing epithelioid cells, giant cells, polymorphonuclear leucocytes, and groups of spores. By cultivation on suitable media a feltwork of mycelium with spores can be obtained.

The general health is little affected ; but the local lesions are of long duration, if untreated.

**Treatment.**—The disease yields promptly to treatment by potassium iodide, which should be given to the extent of not less than 1 drachm daily ; and the local lesions should be dressed with a solution of iodine 1 part, potassium iodide 10 parts, water 500 parts.

## ASPERGILLOSIS

Another fungus which may cause serious disease in man is the *Aspergillus fumigatus* of the order Ascomycetæ. It may grow in the lungs and produce caseating and suppurative lesions. This occurs as a rare condition amongst pigeon-feeders, in consequence of the fungus being contained in the seed, which the feeder puts into his mouth for transference to that of the pigeon. The lesions are like those of tubercle, with cavities, fibroid changes, or emphysema, according to the rate of progress and the resistance of the tissues ; and the symptoms are dyspnœa, cough, expectoration, and hæmoptysis. The fungus does not spread to other parts of the body, and recovery may take place spontaneously.

The aspergillus sometimes grows in the eye, ear, nose, on wounds and ulcers, on the skin (some forms of the disease called pinta) and in the tissues of the foot (one variety of madura foot).

## RAT-BITE FEVER

This disease, which occurs in China and Japan, where it is known by the names *sokŏdu* and *sokosho*, has been recently recognised in the United Kingdom, America, Italy, France, and Spain. It is conveyed by the bite of an infected rat, and has also followed the bite of a ferret or cat.

The wound heals normally after the bite, but after a period of incubation of from one to six or eight weeks pain occurs in the wound, the parts swell, vesicles form around, and even ulceration and sloughing may follow. At the same time the patient has headache, rigors, nausea and vomiting, and sometimes sore throat and hoarseness of voice. The temperature rises to 103° or 104°, continues high for a period which is generally from one to four days, but may be as long as twelve days, and then subsides to normal. There is often an erythematous eruption in patches (*erythema exudativum*) over the body, face and limbs, the lymph glands may swell, and leucocytosis up to 19,000 may occur, but the spleen is not enlarged. After an interval of from two to seven or eight days there is a second attack of pyrexia, like the first ; and these attacks of fever are repeated for several weeks, and it may be for months, or even years. The diagnosis may be confirmed by injecting sterile saline into one of the swollen lymph glands and then removing some of the gland juice. Spirilla may be seen by dark-ground illumination. The organism, formerly thought to be a spirochæte, is now known as *Spirillum minus* (*S. morsusmuris*). The mortality is said to be 10 per cent. Arseno-benzol or novarseno-benzol, given intravenously, has a specific effect on the disease. Frequently one injection of 0·6 gramme of novarseno-benzol is sufficient. It may be repeated if necessary after an interval of a few days.

## RELAPSING FEVER

(*Febris recurrens, Spirochætosis recurrens*)

Relapsing fever is a specific contagious disease, generally occurring in epidemics, not distinguished by any rash, but consisting of a short fever which terminates suddenly in six or seven days, and is followed by a relapse of the same nature after an interval of about a week.

**Ætiology.**—Relapsing fever attacks patients of all ages, and of both sexes, though male patients have been more numerous in the proportion of three to two. Epidemics have occurred in the British Isles years ago, but it is rarely seen here now, unless brought from the tropics by sailors. It occurs, however, in India, Russia, and America. There is a special form of the disease in Africa and Central America (*see* below).

The micro-organism of European relapsing fever is a spironeme, which was first discovered in the blood by Obermeier in 1873, and is called *Spironema obermeieri*, or *S. recurrentis*. Formerly the organisms, which give rise to different clinical forms of the disease in various parts of the world, were thought to be morphologically distinct, but this is not so. As usually seen *S. recurrentis* consists of an exceedingly fine thread, from 16$\mu$ to 40$\mu$ in length by 1$\mu$ in width; it is spirally coiled, and in constant movement of a rotatory or lashing character. Spironemes may adhere to one another so as to form masses, or they adhere, either singly or many, in a tufted manner to the blood corpuscles. Their quantity in the blood varies with the different stages of the illness. They are always present during the paroxysms, and frequently increase in numbers as the fever progresses, but about the time of the highest temperature, just preceding the crisis, begin to diminish; by the termination of the crisis they have completely disappeared from the blood, and they remain absent until near the advent of the relapse. Both men and monkeys have been infected by inoculation with the blood of patients suffering from the disease. Infection is effected by parasitic insects; and in India, Russia, North Africa, and America, but not Central and South America, the evidence is in favour of the transmission of the spironemes by lice, in whose bodies the organisms have been found.

The Central African tick fever is also a relapsing fever, but the pyrexial periods last only two or three days—that is, they are of shorter duration than those to be described; this disease is due to the *S. duttoni*, which is conveyed from person to person by the tick (*Ornithodorus moubata*). Infection takes place while the tick is gorging blood, because some of its infected body secretion is rubbed into the bite. The tick itself becomes infected by ingesting human blood containing the spironemes. The latter are changed into granules inside the tick's body, and these may be passed on into the next generation of ticks. Under certain conditions the granules may be changed back again into small spironemes about ten days after ingestion, and they tend to persist in this form throughout the life of the tick. There is reason to think that it is this form, or the granule stage immediately preceding it, which is most infective to the human subject. It is probable that relapse in man is due to the development of fresh crops of spironemes out of granules formed, possibly in the spleen, by those spironemes which caused the first attack (Leishman). The Persian form of tick fever is due to *Spironema berberum*. In Central America the spironeme (*S. venezuelense*) is conveyed by ticks (*Ornithodorus venezuelensis* and *O. talaje*). A somewhat similar life history has been made out in the case of *S. recurrentis* in the louse, but the infection in this case is not passed to the offspring in a hereditary manner.

**Morbid Anatomy.**—With the exception of the condition of the blood already described, there is no specific or constant lesion. The enlarged spleen, which is especially likely to be found when death takes place during a paroxysm, sometimes presents infarctions, and more rarely small abscesses. The liver also is usually found enlarged, firm, and loaded with blood; but the jaundice is not always explained by any alteration of its structure, or by obvious obstruction of its duct. The kidneys may be congested. Other pathological conditions have been alluded to in the description of the complications.

**Symptoms and Course.**—The period of *incubation* varies from two to sixteen days, but in a large proportion of the cases examined for this purpose it was less than nine days. The fever begins suddenly with a chill or rigor, soon followed by frontal headache, and pains in the back and limbs. After a short time

the chill gives place to a feeling of heat, the skin becomes dry and burning, and the headache and pains in the limbs are aggravated. On the first day the temperature may be 102·5° or 103°, or higher. The patient soon takes to his bed, and suffers severe thirst, with anorexia and perhaps nausea and vomiting, the face is flushed, and the tongue is covered with a thick white fur. The temperature continues high, reaching 104°, 105°, or 106° at night, though often falling a degree in the morning.

There may be with this an occasional rigor, and not infrequently free sweating. The pulse is rapid, 110 to 120, and the respirations are quickened to thirty or more. Lice are commoner in cold weather, and it is in the cooler parts of the year that epidemics occur. In India the hot weather season destroys the lice and so puts a stop to the epidemic, which recurs in the following spring. Infection occurs rather readily from relapsing fever patients directly, and nurses and bacteriologists have contracted the disease from blood, excreta, droplets of saliva in the breath, etc., after all lice have been destroyed. In some cases there is jaundice, which, however, is usually slight: in such cases the urine is stained with bile, but the fæces are normal in colour. There is frequently tenderness over the

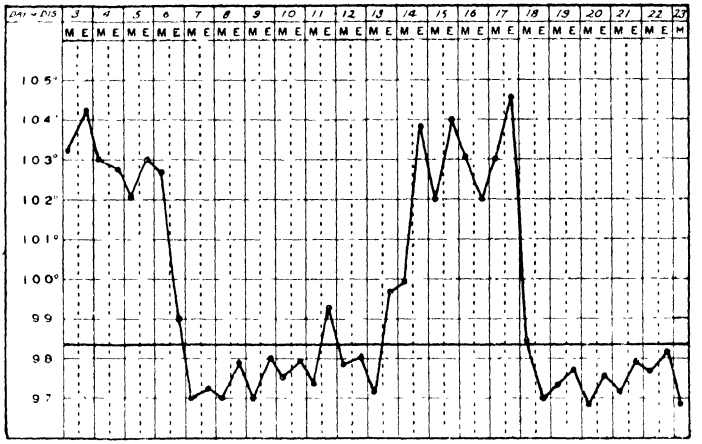



FIG. 13.—Temperature in Relapsing Fever.

hepatic and splenic regions, and both liver and spleen are enlarged. Occasionally there is well-marked herpes of the lips.

The urine is very variable, its quantity being affected by food, by vomiting, and by sweating. In the height of the fever the chlorides are diminished, and there is occasionally a small quantity of albumin. Epistaxis sometimes occurs. Though there is no typical rash, in some cases an eruption has been observed either of pink maculæ, circular and of the size of a pin's head, or of petechiæ, and there may be purpura hæmorrhagica and hæmaturia.

The condition remains much the same for about six days; the patient, as a rule, gets but little sleep, suffers severely from the muscular and arthritic pains, and is generally quite clear mentally till towards the end, when he becomes delirious. In some epidemics delirium has been observed as an early symptom and there have been symptoms of meningo-encephalitis, with temporary paralysis, especially in the Central Africa form (Tick fever); in this variety iritis is a frequent complication. All the febrile conditions then become greatly aggravated, the pulse quickens to 130, the respirations to forty, the temperature rises in a few hours to 106°, 107°, or even 108°, the face is flushed, the tongue dry, or brown, and the delirium is increased; and then suddenly the crisis takes place.

Perspiration breaks out, rapidly becomes profuse, the temperature, pulse, and respiration quickly fall. In a few hours the temperature is 98°, 97°, or 96° (*see* Fig. 13), the pulse seventy or less, the skin is moist, the tongue clean, and the patient free from delirium; and except that he feels weak, he may express himself as being comparatively well. However, where the fall of temperature is considerable—it may be 10° or 12° in twelve hours—and the sweating is profuse, there may be considerable collapse for some hours, especially in elderly patients. The crisis is sometimes marked by diarrhœa or by epistaxis. Recovery from this point is very rapid; the temperature, which has become subnormal, regains the normal level; the patient is soon ravenously hungry, and recovers strength so quickly as to be up in three or four days. He appears, indeed, completely convalescent, when suddenly about the fourteenth day, or a week from the termination of his first attack, he is seized again with chills and pyrexia, and the whole series of phenomena is repeated. He has the same high temperature, the pains in the head, back, and limbs, and the sleeplessness; and again, after a few days, occurs a second crisis, with the profuse sweating and the sudden cessation of fever. In some cases a second relapse occurs, and in some even a third, fourth, or fifth. But these last form a very small proportion of the cases. On the other hand, some patients escape without any relapse at all. Most commonly the relapse is of somewhat shorter duration than the first attack, lasting on an average four to five days, but it may be only two or three days. It is often milder than the first attack, and it may be indicated only by slight rise of pulse and temperature, and general malaise. But it may be more severe; and, indeed, a certain proportion of deaths takes place during the relapse. Convalescence from relapsing fever is often very slow. The disease in this country has been much less fatal than either typhus or enteric fever, showing a mortality of 4 per cent. (Murchison), but the mortality has been 14 and 18 per cent. in Russia, Egypt, and India. Death occurs most commonly at the height of the first attack, or immediately after the crisis, from exhaustion and collapse; and this is especially the case with old people. But in some epidemics suppression of urine and convulsions, pneumonia, dysentery, parotitis, have helped to increase the number of deaths.

**Complications and Sequelæ.**—An important complication is *pneumonia*, which has been frequent in some epidemics, and may be the cause of death; it may be associated with pleurisy, and rarely gangrene of the lung has resulted. The spleen may attain a great size, and it has been known to rupture, with a fatal result. *Diarrhœa* and *dysentery* sometimes occur in a severe form. *Jaundice* occurs in probably less than 20 per cent. of the cases, and may appear in the first attack alone, in the relapse alone, or in both paroxysms. Many of the cases in which it occurs are severe, or even fatal, but others are quite mild. In the severer forms it may be accompanied by epigastric and hypochondriac pain, vomiting of blood, albuminuria, hæmorrhages, delirium, coma, and subsultus.

Erysipelas and œdema of the legs occasionally occur, and sometimes the parotid or submaxillary gland inflames or suppurates. *Ophthalmia* occurred in some epidemics, commencing in the deeper structures of the eye, so that blindness was observed before the external signs of inflammation. Pregnant women almost invariably abort, and in such cases hæmorrhage from the uterus may become a danger.

**Diagnosis.**—During the first paroxysm of fever a definite diagnosis can only be made by finding the spironeme in the blood, and in every suspicious case one or more blood examinations should be made as early as possible. In the course of an epidemic, the sudden severe onset, absence of eruption, severe pains in the limbs, and jaundice when present, are distinctive; but in isolated cases the disease may be confounded with typhus, enteric fever, small-pox, rheumatism, or pneumonia, and in tropical countries with remittent malarial or with yellow fever. Typhus and small-pox are soon excluded by the absence of eruption; in other cases diagnosis may be difficult until the occurrence of the typical crisis.

Even then a latent pneumonia may be thought to explain the case, until the occurrence of a relapse a week later makes it clear. There is often a general resemblance to acute rheumatism in the flushed face, white furred tongue, sweating, and severe pains; but the existence of pain in the muscles and the absence of swelling in the joints should prevent a mistake.

**Prevention.**—Destruction of lice or ticks is the most important factor (*see* p. 78).

**Treatment.**—The administration of arseno-benzol or novarseno-benzol has a specific action on the spironeme, and produces an immediate improvement in the patient's condition. The organisms quickly disappear from the blood, and the pyrexia diminishes. The drug should be given intravenously as soon as relapsing fever is diagnosed, but is apparently inefficacious if given in the apyrexial period. A dose of 0·45 gramme of novarseno-benzol is usually sufficient. After treatment with arseno-benzol compounds relapse is exceedingly rare.

Apart from this, the disease must be treated like other fevers. Sponging with tepid water, or packing in wet sheets, will give temporary relief when the fever is very high; and headache may be treated with cold applications. On the other hand, if there is much tenderness of the liver or spleen, fomentations give relief. During the severe perspiration of the crisis the patient must be kept as far as possible dry; and the tendency to collapse must be met by additional bedclothes, hot bottles, and diffusible stimulants. Cardiac failure requires the administration of digitalis.

## SAND-FLY FEVER

(*Three Days' Fever, Phlebotomus Fever*)

This is a short fever which occurs in hot climates (India, Italy, Southern Austria, Egypt), and is believed to be conveyed by the bite of a small fly, the sand-fly, or *Phlebotomus pappatasii*. It is thought that the flies convey by their puncture an organism, but this has not yet been identified. The disease is most prevalent during the early summer and the autumn months, when the sand-flies are most numerous.

The period of incubation is from three to five or seven days; and the onset is sudden, with headache, intense pain behind the eyes, fever, anorexia, chilliness, but not usually rigors, and aching pains in the loins and limbs, especially in the muscles. The conjunctivæ are injected; the face is flushed; there may be pharyngitis or tonsillitis. Vomiting is rare, and when a marked feature is more suggestive of malarial infections than of sand-fly fever. Epistaxis is not uncommon, and there is generally constipation at first, and later sometimes diarrhœa. The temperature rises to between 101° and 104°, continues high for about two days, and has usually come down to normal by the end of the third day. The increase in the pulse rate is not in proportion to that of the temperature: with the highest pyrexia the pulse may be below eighty, and is rarely more than 100 per minute. The blood shows leucopenia, with slight relative increase of the mononuclear leucocytes. The marks left by the insect are minute red dots, or larger papular infiltrations. It is often found that persons who show marked skin reactions to the sand-fly bites are less liable to attacks of sand-fly fever. Typical attacks of the fever often occur in patients who show practically no signs of having been bitten.

The prognosis is always good, but convalescence may be delayed by anæmia, loss of flesh, or neurasthenia. The disease confers immunity, but relapses occur.

**Diagnosis.**—Sand-fly fever may be confounded with malaria and dengue. In dengue the pains are mainly in the joints. In malarial infections the fall of temperature even without quinine is much more rapid than that of sandfly-fever, which always takes from twenty-four to forty-eight hours to reach normal.

**Treatment.**—The patient should rest in bed and should take a saline purgative. According to most authorities, Tinct. Opii ℥x. is the only drug which effectually relieves headache. The bites may be painted with tincture of iodine, to prevent secondary infection.

## MALARIAL FEVERS

Especially in marshy districts, but also in other localities possessing special features, certain diseases occur endemically which are known from their clinical characters as *intermittent* or *remittent* fevers, and from their source as *malarial, paludal,* and *marsh* fevers. The word *malaria* (*mala aria,* bad air) was also used to indicate the virus or infective agent before it was known that this was an organism invading the blood corpuscles and conveyed from man to man by mosquitoes. It is obviously now a misnomer.

The milder forms of the disease which occur in most temperate countries are the *intermittent* fevers (known in England as *ague*) characterised by the periodical recurrence of febrile attacks, separated by intervals of comparative health. The attacks last a few hours, and recur every other day (*tertian* fevers, the second attack occurring on the *third* day of the illness), or every third day (*quartan* fevers), and the subsidence of the temperature to the normal after each attack constitutes the intermission from which their name is taken. Tertian fever is more common in temperate climates; quartan is the least frequent form, but it is common in certain parts of Italy, Africa, and India. In the hotter parts of the temperate zone, and in tropical countries, the attacks are often more irregular in their occurrence, and of longer duration, with shorter intervals between them; or the temperature fails to reach the normal between the attacks, so that the fever is only *remittent* instead of being intermittent (*see* p. 21); or the temperature is constantly so high that the fever is *continuous.* These forms are more severe than the simple intermittent, and form the *æstivo-autumnal* fevers of Italy, and the *subtertian, remittent, continuous* and *malignant* varieties of the tropics.

**Ætiology.**—These fevers are due to the action of one or more micro-organisms which are contained within the red corpuscles; and the organisms are conveyed from man to man by certain varieties of mosquitoes belonging to the genus *Anopheles,* in the bodies of which they pass through one stage of their development. The ætiology of malarial fevers is therefore largely explained by the life history of the mosquito. Where the mosquitoes can breed and flourish, and where surface water is provided for the growth of their larvæ, malaria may occur; where mosquitoes cannot breed, malaria will be absent. Even in mosquito-breeding countries or districts those who can protect themselves from the bite of the insect will escape the disease. Besides the presence of Anopheles, there is another condition which must be fulfilled before malaria can become endemic in any district. The mosquito becomes infected by sucking blood from human beings who are carriers of malarial parasites. Unless, therefore, human carriers are present, there can be no malaria.

Though not peculiar to tropical countries—indeed, it was once prevalent in England—the disease is more frequent where the atmospheric temperature is high; and it is practically confined within the latitudes of 63° N. and 57° S. In this country the disease still lingers in Thanet and the Isle of Grain.

The parts of England formerly subject to it were the borders of the Thames in Kent and Essex and the fen districts of Cambridgeshire and Lincolnshire. Though these are all low-lying districts, and salt-water marshes are quite as favourable to its occurrence as fresh-water areas, malaria also occurs at high levels in some parts of the world, as, for instance, in the Apennines at a height of 1,100 feet above sea level, in the Pyrenees at 5,000 feet, and in Peru at 11,000 feet.

No race is quite immune, though negroes appear to be less susceptible than white men. The disease may be contracted at all ages. People in ill-health are

more liable to it, as well as those exposed to damp, to cooling influences, or to excessive heat of the sun, and those who indulge in immoderate eating or drinking.

**The Malarial Parasites.**—The micro-organisms of malaria were first described by Laveran, and are now known as members of the family *Plasmodidæ*. Of these at least three occur in man, and others in similar diseases in birds. These three are the parasite of benign tertian fever (*Plasmodium vivax*), the parasite of quartan fever (*Plasmodium malariæ*), and the parasite of the subtertian or malignant tertian fever (*Plasmodium falciparum* or *Laverania malariæ*).

The first and second of these organisms are protoplasmic bodies (*trophozoites*), which exhibit amœboid movements, and are first seen in the early part of the intermission within the red corpuscles, looking like clear spaces. In the course of a few hours they enlarge and occupy more and more of the corpuscle; and granules of pigment (*hæmozoin*) accumulate in their interior. Then the pigment aggregates towards the centre of the organism, which now loses its amœboid character, contains a nucleus, and is called a *schizont*. A process of division (*segmentation* or *sporulation*), first of the nucleus, then of the cytoplasm, takes place, so that the schizont breaks up into from six to fifteen or twenty smaller clear bodies, *spores* or *merozoites*; and, at the same time, both these and the pigment granules become free in the blood plasma. This process of segmentation corresponds with the onset of the paroxysm of ague, and during its course, while some of the merozoites are no doubt included in and destroyed by leucocytes, others enter into fresh red corpuscles, and start a new cycle of events by enlarging to form trophozoites and schizonts, which again break up into merozoites. This method of multiplication is called *schizogony*.

The quartan parasite is slower in its development, taking three days to fill the corpuscle and undergo segmentation, whereas the tertian parasite takes only two days; and this difference accounts for the difference between the lengths of the intervals in the quartan and tertian fevers. Moreover, the quartan parasite has a more distinct outline, never so completely fills the corpuscle (which is otherwise but little changed), presents coarse granules of dark brown or black pigment, has slower amœboid movements, and breaks up into from six to twelve segments. On the other hand, the tertian parasite has a less defined outline, causes swelling and pallor of the corpuscle, has fine yellowish-brown pigment granules, more active amœboid movements, and divides into from fifteen to twenty spores.

There may, however, be two crops or sets of tertian parasites, which segment or sporulate on alternate days, when the fever will be recognised as one of *quotidian* or *double tertian* type; and there may be two sets of quartan parasites, maturing on different days, producing a *double quartan* fever, or three sets, producing a *triple quartan*, or, since every day is involved, another *quotidian* type. From the merozoites are also developed sexual forms—a female type, or *macrogametocyte*, with small rounded excentric nucleus and a cytoplasm full of granules and pigment; and a male type, or *microgametocyte*, with a large nucleus and clearer cytoplasm. The formation of the sexual cells in the human body is a process distinct from *schizogony* and is generally known as *gametogony*.

The subtertian parasite (*L. malariæ*) has a life cycle in the human host quite distinct from the other two species. The trophozoites are very small, and one or more may invade the same corpuscle. They are often seen as small ring-like bodies one-seventh of the diameter of the red corpuscles, with high refractive power and containing less pigment than the other forms. The period of development is irregular, varying from twenty-four to forty-eight hours, and in the later stages (sporulation) they are not found in the peripheral blood vessels, but exist only in the capillaries of the internal organs (*e.g.* spleen and bone marrow). The macrogametocytes and microgametocytes form *crescentic* bodies, each being a colourless, transparent, immobile mass, with pigment granules in the interior, longer than the normal diameter of the red corpuscle which appears to be stretched

over it. The macrogametocyte is longer and thinner than the microgametocyte, has a darker cytoplasm, and more compact chromatin.

**Relation of the Mosquito to Malarial Parasites.**—The mosquitoes concerned in malarial infection are the *Anopheles maculipennis*, and some other species of the genus *Anopheles*. They not only transfer the malarial organisms from one human individual to another, but they allow in the interior of their bodies a true sexual process (*sporogony*) to take place, quite different from that which occurs in the human blood.

When the blood of a patient containing male and female gametocytes is sucked into the stomach of the mosquito, the gametocytes escape from the corpuscles and undergo further changes. The microgametocyte, after some alterations in pigmentation, throws out three or four fine filaments, or *flagella* (*microgametes*, or *spermatozoa*), which perform lashing movements, and ultimately separate from the sphere and move freely in the fluid. The macrogametocytes become reduced in size, and remain as granular spheres, without flagella (*macrogametes*). The liberated flagella approach the granular spheres, enter their substance, and thus impregnate them. As a result the spheres become elongated bodies with a pointed extremity, and are in a position to penetrate other substances; they are called *zygotes*, or *ookinetes*. The zygotes then penetrate the wall of the stomach, where they have been observed, and where they enlarge to a diameter of 60$\mu$ or 80$\mu$, so as to project into the body cavity of the animal. In the interior of the zygotes, which, having reached the outside of the stomach, now become spherical *öocysts*, are formed a vast number of minute rods (*sporozoites*), which are discharged by the bursting of the zygote or öocyst into the blood of the mosquito. Thence they reach the cells of the salivary gland at the base of the proboscis, from which they are carried during the process of puncture into the blood of the next human being attacked by the insect. These sporozoites enter the human corpuscles, and so give rise to the amœboid bodies, or trophozoites, first mentioned. In the case of the subtertian parasite (*Laveranía*) the crescentic gametocytes become spherical before developing into microgametes and macrogametes.

**Morbid Anatomy.**—In fatal cases there are punctiform hæmorrhages of the meninges and white substance of the brain; the capillaries contain infected red cells, and their endothelium may be fattily degenerated. In the enlarged spleen the trabeculæ of the pulp are distended with infected red cells, but the Malpighian corpuscles are not pigmented. In the liver the endothelium of the capillaries is swollen and pigmented. The liver cells contain hæmosiderin, most abundantly round the central vein of a lobule. In the bone marrow there are parasites and *hæmozoin*, both free and within the large mononuclear leucocytes, and in macrophages. Crescents are often found here when scanty elsewhere. In long-standing cases the yellow marrow may become red.

In malaria with intestinal symptoms parasites are abundant in the capillaries of the villi.

In old cases, and in malarial cachexia, the spleen has become more fibrous, firm, tough, and pigmented, especially in the connective tissue surrounding the follicles, often with a thickened capsule, adherent to surrounding parts, presenting infarcts, and in long-standing cachexia perhaps lardaceous.

The blood may contain brown or black pigment granules, either free or within the white blood cells; and this pigment, found also in the spleen, liver, brain, kidneys, heart, and in the lymphatic glands and marrow of the bones, gives a slaty or dark grey colour to the various tissues. The condition is described as *melanæmia*. The liver is pigmented, mainly in the periphery of the lobules; the capillaries are dilated, and their epithelium is pigmented. The hepatic cells are atrophied. The marrow of the long bones is usually red, and normoblasts are common.

**Symptoms of Benign Tertian and Quartan Infection.**—The period of

*incubation* is from three to twelve days, shorter in the irregular, longer in the regular forms; but it may break out almost immediately on exposure. However, the first recognisable attack may take place years later. In some cases there are prodromal symptoms, consisting of malaise, headache, pains in the limbs, epigastric fulness, nausea, and slight chills or flushes.

A typical attack of malaria or ague consists of three stages: the *cold* stage, the *hot* stage, and the *sweating* stage.

*The Cold Stage.*—The patient feels tired and listless, has headache and pain in the back and loins, then feels chilly, and the *rigor* begins. He generally lies curled up in bed, shivering all over, and his teeth chattering. The surface is actually cold, but the thermometer, placed in the mouth, rectum, or axilla, will show that the temperature is already considerably above normal. Indeed, it commences to rise some minutes (or even one or two hours) before the rigor; but the sensation of cold and the actual cold of the surface are due to contraction of the superficial vessels. The urine is abundant, clear, and of low density. Nausea is usually present, and vomiting is extremely common. This stage lasts from half an hour to two hours, and the axillary temperature rises rapidly, attaining to a height of 105°, 106°, or even more, towards the end of the period.

*The Hot Stage* begins with a sense of warmth diffusing itself over the body, and the surface, hitherto cold, becomes intensely hot. The temperature in the axilla rises still a little higher than it was at the end of the cold stage; the arteries are relaxed; the pulse becomes quick, full, and hard; the carotids throb, the face is flushed, and the head aches. There is a tendency in some cases to stupor or delirium. The urine during this stage is scanty, dark, and of high density. There is often an eruption of herpes about the mouth. This stage lasts from three to four hours.

*The Sweating Stage.*—The skin, hitherto dry, now gradually becomes moist. Sweating is profuse for some hours; the pains and discomfort of the hot stage are relieved; the pulse becomes softer and slower, and the tongue moist. The temperature falls, at first slowly, then more rapidly, until the normal is reached; and, finally, with the subsidence of the temperature the sweating ceases, and there is a return to the preceding state of health. During this stage the urine is of high density, and deposits a sediment of urates.

When the attack is over the patient feels perfectly well, but after an interval determined by the nature and number of the parasites he is seized with another similar paroxysm.

Benign tertian infections, however, by no means always show the typical malarial attack as described above. Not infrequently the temperature may remain raised for a day or two and then suddenly fall to normal or subnormal with profuse sweating in a few hours. Vomiting is very frequent, especially at the onset of an attack.

The spleen is not, as a rule, palpable during a first attack of malaria, but in cases which have lasted some time it can usually be felt as a soft tender mass below the costal margin. In still more chronic cases it becomes definitely hard and often less tender.

The blood, in addition to the presence of malarial parasites, shows a diminution of hæmoglobin and of both red and white corpuscles. Of the latter the polymorphonuclear leucocytes and lymphocytes suffer most, while there is a relative increase of the large mononuclear cells. This condition persists for some weeks after the attack.

The attacks take place mostly in the morning hours or at noon, the tertian especially at noon. Succeeding attacks are, however, not always at the same hour, but in some cases may get earlier and earlier, in others later and later. The former are said to *anticipate*, the latter to *postpone*.

Ordinary attacks of tertian fever are rarely fatal, though death may happen

in very young or in old people, or in those debilitated by previous illnesses. The spleen, if enlarged, is liable to be ruptured by slight trauma.

The complications of simple tertian fever are not numerous. Bronchial catarrh, epistaxis, and albuminuria occur occasionally; anæmia is a constant feature in patients who have had the disease for some time; neuralgia, especially of the supraorbital nerve, is not infrequent in chronic cases.

**Malignant Tertian Infection** (*Subtertian, Æstivo-autumnal*).—The incubation period of malignant tertian infection may vary from ten days to many weeks. As a rule, the onset of the disease is insidious, with a progressive deterioration of the general health and occasional slight rises of temperature. Often when the patient first comes for medical advice he is already markedly anæmic, and may have a definitely enlarged spleen. Typical malarial rigors are rare in malignant tertian infection, and the fever tends to be lower and more irregular than in benign infections. Gastric symptoms with troublesome vomiting are often a marked feature, and may be those for which the patient first seeks medical aid.

Various types of malignant tertian infection have been described according to the predominant symptoms. The severe forms may be grouped under three main headings: the typhoid form, the algid form, and the cerebral form. Needless to say, a case may not conform strictly to any one of these types.

In the typhoid form, at one time known as typho-malarial fever, the temperature remains high for many days with very little remission. The general appearance of the patient is very suggestive of enteric fever. Asthenia is marked, and the patient may develop a low delirium. Vomiting is, however, commoner than in enteric fever. Jaundice may occasionally become a marked feature. If the disease is recognised and sufficiently vigorously treated with quinine, the temperature drops rapidly, and the symptoms improve. If, however, the patient is treated as enteric, the fever may continue for one or two weeks and a fatal issue ensue with coma, delirium, and collapse.

The algid form of malaria is much less common. The temperature falls below normal, and a profound state of collapse supervenes. The mouth temperature may be exceptionally as low as 88°. The skin is cold and covered with sweat. Vomiting and diarrhœa are continuous and intractable, and occasional blood may be vomited or passed *per rectum*. Parasites may sometimes be demonstrated in smears from a hæmatemesis. The prognosis in this type of case is bad even with quinine treatment.

The cerebral form is usually of very rapid onset. The patient is quite well until suddenly he is seized with severe headache, dizziness, and drowsiness. These may pass rapidly into complete unconsciousness, or he may develop hemiplegia, paraplegia, aphasia, or epileptiform convulsions. In other cases there may be wild delirium and mania. The temperature is not always markedly raised, but there may be hyperpyrexia. The latter is extremely common in hot climates. The rectal temperature may rise to 110° or more with cyanosis, stertorous breathing, coma, and convulsions, and the condition exactly simulates heat-stroke.

**Mixed Infections.**—It is not uncommon to find both the benign and malignant parasites present in the blood at the same time, and it is probable that this is even more often the case than would be supposed, as the observer is apt to give up the search as soon as a parasite of either type is discovered. Thus in many cases of severe malarial attacks in which laboratory reports indicate the presence of benign tertian only it is quite possible that a more prolonged search might demonstrate the presence also of malignant tertian.

Many patients also show benign tertian at one time, usually in the early summer months, and malignant tertian during malarial attacks in the autumn. It would appear that the two parasites are often present in the same patient, but that one or other predominates in the blood at different seasons of the year. In

cases of mixed infection the temperature charts are naturally less typical, and the symptoms tend to be more severe.

The characters of malarial epidemics vary widely in different countries, even though the parasites involved are apparently morphologically identical. Thus during the War malaria in Macedonia tended to be of the severer types, while in Mesopotamia, though there was a great deal of malignant tertian infection, severe cases were rare and mortality relatively low. The difference is probably accounted for not so much by increased virulence of the parasites in certain areas as by a variation in the dose of the parasites. Where troops were relatively less able to protect themselves from mosquitoes, and where the latter were present in tremendous numbers, the type of attack tended to be very severe.

**Malarial Cachexia.**—In those who have had repeated attacks of malaria, and in some who have resided in malarial districts without developing such attacks, as well as in the natives, children and adults of badly affected areas, the bodily health is seriously undermined. The most prominent feature is *anæmia*, causing a sallow, earthy look, with pallor of the lips; it is due to the destruction of the blood corpuscle by the parasite and the conversion of the hæmoglobin into black pigment. There are also numerous functional disturbances, dizziness, loss of appetite, digestive disorders, pains in the joints and muscles, lassitude, and indisposition for exertion. In severer forms there is ascites, œdema, or hæmorrhage, but no fever. The spleen in these cases is enlarged, reaching even below the level of the umbilicus, and forward to the middle line, constituting the *ague cake* of early English writers. It is hard and often tender. The liver may also be enlarged. Occasionally it is *cirrhosed*, with moderate reduction in size; but the relation of cirrhosis to malaria is by no means definitely settled.

**Relapse.**—It is relatively rare for a malarial infection to be completely cured after the first attack, even though treatment be vigorous and prolonged. This is true of benign, but malignant infections are sometimes cured; in fact, on the whole the former type is more liable to relapse than the latter. Relapses have much the same general character as first attacks, but as a rule are less severe, partly because the patient usually takes quinine early. Some cases of apparent relapse may be due to re-infection if the patient is still exposed to infection, but very commonly relapses occur long after he has left malarial countries. Parasites can usually be demonstrated in the blood during a relapse. A chill or over-exertion favours the development of an attack. The infection tends to die out after two or three years, the attacks becoming less severe and less frequent, provided re-infection does not take place.

The **Diagnosis** of intermittent fevers is generally easy; the attacks are often distinctive in themselves, and their nature is confirmed by the recurrence at regular intervals, provided that the intervals represent a tertian or a quartan form. Daily rigors are less to be trusted, as they occur in various septic conditions, such as *pyæmia, abscess of the liver,* and *malignant endocarditis*. The pyrexial attacks in other forms of suppuration and in phthisis may simulate ague; on the other hand, the rigors in these complaints are quite irregular. If treated with quinine, on the supposition that they are malarial, no result will be obtained, whereas malaria yields to an adequate dosage with this drug. In the severe forms of malarial fever, the nature may be overlooked from the prominence of some local disorder, and from the slight development of the febrile characters; thus the comatose form may be mistaken for apoplexy, and others for pneumonia or cholera. The resemblance of the continuous forms to typhoid has already been mentioned. On the other hand, there are *latent infections*, which do not develop sufficiently to produce the typical outbreak. In all these cases the diagnosis can be made by an examination of a blood smear stained by Leishman's method (*see* Examination of the Blood). Parasites are more readily demonstrable in films during the acute attacks, but immature forms may usually be found during the apyrexial period on careful examination. It must, however, be realised

that a single negative blood examination is of practically no value. This is especially true if the patient has been taking quinine either as a prophylactic or during the attack of fever. The administration of even 10 grains of quinine is often enough to clear the peripheral blood of parasites without reducing the fever. Assistance can be also derived from enumeration of the leucocytes: generally they are in excess of the normal during the rigor and fever, and then fall below the normal (leucopenia) until the next attack. A differential count shows a great increase of the large mononuclears, especially during the apyrexial period, and a rise of these corpuscles to 20 per cent. of the total leucocytes at this period is strong evidence of malaria in otherwise doubtful cases (Stephens). The leucocytes, especially the mononuclears, may be pigmented.

In any fever of obscure origin in a country in which malaria is endemic, a possible diagnosis of malaria must be borne in mind. Even in cases in which parasites cannot be demonstrated in the blood, it is wise, if there is any possibility of malaria, to try the effect of quinine. This is especially true if the patient is dangerously ill, as if quinine is withheld owing to failure to find parasites the chances of recovery may be seriously prejudiced. At the same time it must not be forgotten that when a malarial subject is attacked by any acute infection, such as pneumonia or influenza, he usually gets a malarial attack in addition. Hence the discovery of parasites does not necessarily mean that the patient is suffering from malaria alone.

**Prevention.**—This can be furthered by (1) the drainage of malarial areas: ague has thus almost entirely disappeared from the British Isles; (2) the extermination of the mosquito: this has been attempted by pouring kerosene upon pools in which mosquitoes breed, so as to destroy the eggs and larvæ: 1 ounce is sufficient for 15 square feet of surface; (3) the protection of the exposed parts of the body by fine gauze nets, especially at night, when alone some species of mosquito make their attacks; (4) the constant use of quinine internally (to the extent of 5 grains daily) by those who are resident in malarious districts. The value of this so-called prophylactic quinine cannot by any means be regarded as definitely proved. Its administration certainly renders it more difficult to detect parasites, and may prevent symptoms of malaria becoming manifest, but it is very doubtful whether it really prevents infection. As soon as the daily quinine is discontinued attacks of fever are apt to ensue. In many cases it may do definite harm, as the patient may drift into a state of malarial cachexia without an acute attack of malaria having called attention to his condition. Estimates have been made in India and elsewhere of the prevalence of the disease—first, by examining the blood of a large number of individuals and learning the percentage of individuals in whom the parasites can be found (Stephens and Christophers); secondly, by ascertaining what percentage of children have enlargement of the spleen.

**Treatment.**—Quinine is by far the most effective drug in the treatment of malaria. There are various methods of administering the drug, and that which is most suitable in any particular case must be selected.

The most widely used method is oral administration. A dose of 10 to 15 grains three times a day may be given in the form of a mixture. Quinine sulphate is most commonly employed, but is liable to cause a good deal of gastric irritation. Moreover, quinine sulphate in tabloid form is very apt to become hard and to be passed unchanged in the stools, especially if *sugar-coated*. The hydrochloride or the bihydrochloride is, as a rule, more efficacious because it is much more soluble. The oral method of administration is absolutely contra-indicated when there is any vomiting or when a thickly furred tongue indicates gastric disorder. In such cases quinine by mouth is worse than useless, as it aggravates the gastric symptoms and is not absorbed.

For cases of subtertian malaria intramuscular injection is by far the best

method. The bihydrochloride may be injected in doses of 10 to 20 grains, dissolved in about 5 to 10 c.c. of distilled water. The best site for injection is deeply into the muscles of the buttock anywhere above the point of exit of the sciatic nerve. Needless to say, careful aseptic precautions must be taken. It is necessary to use a long needle and to avoid any of the drug being injected subcutaneously, as, if this occurs, sloughing of the skin will take place. Usually after two to three injections the patient has sufficiently recovered to take quinine by mouth.

In the most severe forms of malaria, such as the cerebral type, intravenous quinine is indicated. A dose of 10 to 20 grains should be given. Quinine bihydrochloride is most suitable, and may be given most conveniently dissolved in about 20 c.c. of distilled water into a vein at the elbow. If the symptoms are not markedly improved within six hours, a second intravenous injection may be given, and also use may be made of the intramuscular method. Intravenous injection should be performed slowly, from two to three minutes being taken to inject the dose. During the injection the patient may complain of tinnitus, vertigo, and dyspnœa, but dangerous symptoms are practically unknown.

Quinine is also sometimes given subcutaneously, and absorption is said to be more rapid by this method than by the intramuscular route. If it is employed, the drug must be given well diluted, as strong solutions will cause sloughing of the skin.

When acute symptoms have passed off, quinine should be administered orally in doses amounting to 30 grains daily for three weeks. In this stage gradually increasing doses of liquor arsenicalis up to 8 minims three times a day should be added to the quinine mixture. After three weeks the dose of quinine should be reduced to 10 grains daily, continued for one to three months.

Apart from quinine, treatment of malarial attacks must consist of confinement to bed, aspirin or Dover's powder for the relief of headache, and attention to the bowels. For the latter, calomel 1 gr. at night is to be recommended, and this should be followed by a saline aperient in the morning. When vomiting is a marked feature, all food should be withheld, and only water given by mouth in small quantities. When the temperature remains high, tepid or cold sponging is beneficial, and when there is hyperpyrexia, the drastic methods of temperature reduction recommended for heat stroke must be employed.

It is very important in the treatment of malaria not to employ quinine half-heartedly. It should be administered in full doses and by the intramuscular or intravenous methods if the patient is at all seriously ill. If this is done, and the patient does not react within a day or two, his condition is probably not due to malaria, and attempts must be made to seek another diagnosis.

The administration of quinine, especially over a long period, causes certain unpleasant symptoms. The most common of these is tinnitus, which may be so marked as to cause almost complete deafness. Loss of appetite, tremor, and attacks of vertigo are other unpleasant symptoms which often accompany a long course of quinine. Some individuals appear to be unduly sensitive to quinine, and may develop a widespread urticarial rash even after a single dose. In such cases there may also be severe attacks of vomiting. Occasionally quinine is better tolerated if it is given in conjunction with small doses of bromide. The following is a convenient prescription: ℞ Quinin. hydrochlor. gr. x, Acid. hydrobrom. dil. m. ii., Syr. Amant. ʒi., Aq. chloroformi ad. ʒss. t.d.s. p.c. In cases of intolerance to quinine injections of neo-salvarsan 0·45 gramme should be given.

Treatment of relapses is the same in principle as that detailed above, but not infrequently a malarial subject knows when he is about to have a relapse, and is able to take sufficient doses of quinine to abort it.

## BLACKWATER FEVER

*(Malarial Hæmoglobinuria)*

In certain parts of the world (the west coast of Africa, Madagascar and the opposite east coast of Africa, Siam, New Guinea, the Southern United States, Central America, and Venezuela, and Guiana), which are mostly tropical, and all within malarial areas, occurs the disorder known by the above names; and for the most part it affects those who have been some time in the locality, and who have had true malarial attacks previously. After a few prodromal symptoms, malaise, pains in the limbs and head, there is a definite rigor, followed by vomiting of bile, and soon a quantity of pink, red, or black urine is passed, the colour of which is due to hæmoglobin, and not to sound blood corpuscles (*see* Hæmoglobinuria). It is very acid, albuminous (to the extent of ½ to 2 per mille by Esbach's test), and deposits a dirty brown sediment of epithelium, granular *débris*, and hyaline casts. With the rigor the temperature rises to 104° or more, but falls somewhat after a few hours, rising again with rigor, and this may be repeated. The patient is more or less deeply jaundiced, and the liver and spleen enlarge and are tender. In favourable cases the symptoms last about a week; in fatal cases vomiting continues; the urine becomes more albuminous and scanty; suppression may occur, and death follows from coma or collapse or uræmia. The mortality is from 16 to 50 per cent.

**Pathology.**—Recent researches have shown that blackwater fever is invariably associated with the subtertian malaria parasite and is probably in some way due to the toxins produced by that organism. It has been demonstrated that, if examined before the onset of blackwater, trophozoites of this parasite may be found in small numbers, but that during the process of hæmolysis the corpuscles containing the parasites are broken up and destroyed, and usually a complete cure of the malaria infection supervenes after recovery from blackwater fever. The exact mechanism underlying the hæmolysis has not been determined. Plehn believes it to be brought about by a process of *anaphylaxis*, that, owing to repeated destruction of parasites during the pyrexial attacks, the body becomes supersensitive to the protein of the malaria parasite, and when the supersensitiveness has attained a sufficiently high degree, the further solution of quite a small number of parasites may precipitate the crisis. In some way or other the administration of quinine in large doses may act as an exciting factor, and this has given rise to the popular theory that quinine is the *cause* of blackwater fever. That this conception is not entirely correct may be gathered from the fact that blackwater fever may arise in people who have never had quinine, and indeed the disease was known long before the introduction of this drug. It is probably correct to state that in some people with a special susceptibility quinine may act as one factor in the production of hæmolysis, in much the same manner as cold, alcohol, or other toxic substances. *Post mortem* the tissues are found to be jaundiced, the blood is very fluid, and the kidneys are enlarged and deeply congested, with degeneration of the tubal epithelium. The liver is enlarged, with areas of necrosis microscopically and much hæmosiderin in the cells, many of which show fatty degeneration.

**Treatment.**—Complete rest and good nursing are essential, and free action of the kidneys should be effected by abundance of diluent drinks. This effect may also be obtained by the intravenous injection of normal saline solutions. The specific treatment by quinine has often been thought to make matters worse and to increase the hæmoglobinuria, but its use has been sanctioned (Manson, Crosse) when it can be shown that malarial parasites are present. If used, the drug must be given as injections of the bihydrochloride and may be preceded by doses of calcium lactate. The sulphate should not be given.

# TRYPANOSOMIASIS

*(Sleeping Sickness)*

The trypanosomes are protozoal organisms which are the causes of disease in man, horses, and cattle. The species which give rise in man to the endemic sleeping sickness of Africa are the *Trypanosoma gambiense* and the *T. rhodesiense*. Another human trypanosome (*T. cruzi*) has been described in Brazil; it appears to cause fever, anæmia, and glandular enlargement.

The trypanosome of sleeping sickness (*see* Figs. 14 and 15) is an elongated tadpole-like parasite measuring in all from 18$\mu$ to 25$\mu$ in length, and from 2$\mu$ to 2·8$\mu$ in breadth; thus it is in length about three times the diameter of a red blood corpuscle. About the middle of its length is a large oval nucleus (*trophonucleus*). At the blunt extremity is a small particle of chromatin (*parabasal body*), and near this a smaller body (*blepharoplast*) from which proceeds a *flagellum*: and this, running along the free border of an *undulating* membrane to the other extremity of the organism, projects some distance beyond it. The parasites are found in the blood, lymph glands, and cerebro-spinal fluid, and their presence in the latter determines the symptoms characteristic of the disease. In the blood they move slowly in a spiral manner; they are never found in red corpuscles, but are devoured and destroyed by leucocytes. The disease has been produced in monkeys by inoculating them with blood or cerebro-spinal fluid containing the parasites.

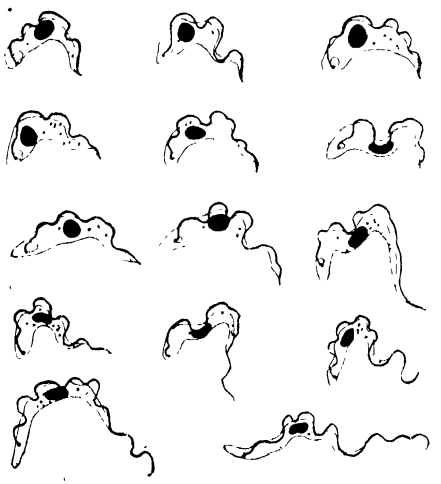


FIG. 14.—*Trypanosoma gambiense*, Dutton. (*After Bruce.*)

**Ætiology.**—The disease has been known for a long time to occur on the west coast of Africa, between the rivers Senegal and Loanda, and for some thousand miles inland. It has also been recently observed in Uganda, Rhodesia, Nyasaland and neighbouring parts of Africa. If it occurs in other parts of the world, the sufferer has always previously resided in Africa. So far it has rarely been observed in others than negroes. It attacks both sexes and all ages, except, perhaps, infants at the breast, or very old persons. The spread of the disease is exactly analogous to that of malaria, the parasite being inoculated into human beings by the bite of a particular variety of tsetse fly. Thus *T. gambiense*, which is the cause of the sleeping sickness in the Congo and Uganda, and is confined almost entirely to man, and does not affect cattle and wild game, is conveyed by the *Glossina palpalis*. And the *T. rhodesiense*, which causes the disease *nagana* in cattle and wild animals, and in much less proportion affects human beings, in Nyasaland and Rhodesia, is conveyed by another tsetse fly, the *Glossina morsitans*.

**Morbid Anatomy.**—The chief lesions are found in the nervous system, and consist of chronic meningo-encephalitis and meningo-myelitis. The pia arachnoid is opaque, slightly thickened, and perhaps adherent, and microscopically it is seen to be infiltrated with round cells, which are also abundant in the perivascular spaces. The subarachnoid fluid is increased in amount, of pale straw colour, and slightly turbid, but rarely purulent. The cortex of the brain is

deficient in pyramidal cells, and the cord may present a diffuse sclerosis, with degeneration of the axons, amongst which the trypanosomes may sometimes be demonstrated lying outside the blood vessels. The lungs are almost constantly congested and œdematous, and sometimes consolidated in one or more places. The heart is usually flabby and pale. The superficial and deep lymph glands are much enlarged, and their lymphocytes are much increased. In many organs there is a leucocyte infiltration round the vessels (Low and Mott).

**Symptoms.**—The course of the disease may be divided into three stages, the incubation, the febrile, and the cerebral stage.

*Incubation Period.*—The bite of the infected Glossina usually causes a local inflammatory reaction which lasts for some days and is markedly more severe than that which occurs after the bite of an uninfected fly. The period of *incubation* may be two to three weeks only, but is sometimes prolonged to some months or even years. As a rule symptoms are absent.

*Febrile Stage.*—The patient suffers from attacks of fever lasting about a week and recurring frequently. At the same time an erythematous rash, often circinate in form, appears chiefly on the trunk. The lymphatic glands in the posterior triangles of the neck also become enlarged. The pulse rate is rapid even during the apyrexial periods, and there may be hyperæsthesia on deep pressure. In some cases the disease would appear to die out at this stage.

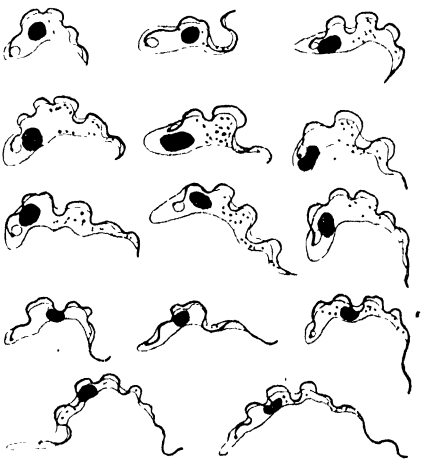


FIG. 15.—*T. rhodesiense*, showing the posterior nucleated forms, Nyasaland. (*After Bruce.*)

*Cerebral Stage.*—When the organism reaches the cerebro-spinal arachnoid cavity, the nervous symptoms develop. The characteristic of the disease is drowsiness, which gradually increases and passes into somnolence, and finally into profound coma. In the early stages, the patient may go about his work, but he is drowsy, listless, languid, or physically weak; he has a vacant expression, the upper eyelids droop, and the lower lip falls so that the lower teeth are exposed, and the lip is dry and cracked. Later the expression becomes vacant, the face is puffy, and the tongue and hands are tremulous. In a month or two more the prostration is increased; the sufferer walks with difficulty supported by a stick, the lower lip is more everted, and saliva dribbles over it. The temperature is raised a little in the evening, and the pulse is often very rapid. The lymphatic glands remain enlarged, but tend to become smaller and harder. Eventually the somnolence is such that the patient lies persistently in one position, either flat on the ground, face downwards, or curled up on one side, or fallen forwards in a kneeling position. During the later stages he eats only what is brought to him, and even stops eating the food still in his mouth. He would starve if not attended to by others. As he becomes more somnolent his secretions accumulate about him, and considerable bedsores form; he may suffer from choreiform spasms and twitchings, and rapid wasting and diarrhœa occur. Papulo-vesicular and pustular eruptions and a scurfy condition of the skin have also been noted. In a small number of cases mania develops at some period of the illness. The disease lasts from five to fifteen months.

**Diagnosis.**—The presence of trypanosomes is most easily demonstrated in fluid withdrawn by puncture of a lymph gland. They may also be present in the

blood. Cerebro-spinal fluid, if centrifugalised, may also show the parasites, with an increase of the cellular elements, and in fatal cases they have been demonstrated in the substance of the brain. In the latter fluid even before the appearance of trypanosomes there will be found numerous lymphocytes, large epithelial cells, and myelocytes, and an increase of serum globulin and serum albumin. The enlargement of lymph glands is a suspicious circumstance.

**Prognosis.**—Though sleeping sickness is a very serious disease a fatal result is by no means necessary; there are districts in Nigeria and Uganda where natives have now become tolerant of the parasite.

**Treatment.**—The patient should be removed from the endemic area. Successful results have recently been obtained by means of Bayer "205," an aniline compound, the composition of which has not yet been disclosed by its manufacturers, the Bayer Company (**19**). It may be injected subcutaneously, but is best given intravenously in doses of 1 gramme in 10 per cent. solution. These should be repeated at weekly intervals, until trypanosomes are not found and do not recur in the blood or cerebro-spinal fluid. In order to produce a big initial effect it might be better to give the three first injections on successive days. A course of treatment consists in giving 10 or 15 grammes. If the case is still refractory it would be best to try tryparsamide before repeating the Bayer "205." Since the injections usually cause albuminuria, with the appearance of casts in the urine, the kidney function should be tested from time to time.

Other drugs of use are compounds of arsenic or antimony. Among the former is atoxyl which is given by intramuscular injection. Manson recommends a dose of 3 grains every third day for two years. Occasionally this drug may cause optic atrophy, and in consequence a similar but less toxic arsenical compound, known by the trade name of soamin, is recommended by some. Tryparsamide, in doses of 2–3 grammes, is also extremely efficacious, especially in cases with cerebral involvement. The total course of treatment spread over two months should be 72 grammes. Antimony is most effective if given in the form of dilute injections of tartar emetic. The first dose given should be $\frac{1}{2}$ grain, which may be gradually increased to $1\frac{1}{2}$ grains. The injections are given bi-weekly for months on end, till a total of 100 grains has been administered.

The patient cannot be regarded as cured until all symptoms have been absent for many months, and until injection of his blood into susceptible animals fails to cause symptoms of trypanosomiasis.

## LEISHMANIASIS

This term includes diseases which are due to infection by a protozoan organism, the Leishmania.

These parasites were first described by Major-General Sir W. Leishman and Colonel Donovan in the organs of persons suffering from the tropical disease Kala-azar, and are known as Leishman-Donovan bodies. They are oat-shaped, oval or spherical, about one-third the diameter of a red blood corpuscle (1·5$\mu$ to 4$\mu$ by 1$\mu$ to 2·5$\mu$), and present two nuclear bodies, one large (*nucleus*, or *macronucleus*), oval and staining rather faintly, the other (composed of a *blepharoplast* and *rhizoplast*) small, rod-shaped, staining deeply, and generally with one end directed to the large nucleus, these two bodies lying against the periphery of the organism opposite to each other. They are found in the spleen, liver, bone marrow, mesenteric glands, kidney and intestinal ulcers; and they can be obtained from the enlarged spleen by puncture during life. They are well shown by the aid of Romanowsky's stain. They are the early stages of a species of flagellate protozoon belonging to the family Herpetomonidæ, and now called *Leishmania donovani*. Their further development can be observed in suitable media; thus the parasite multiplies by subdivision, a flagellum is evolved from

the neighbourhood of the blepharoplast, and the organism elongates into a fusiform body, which may reach 12–20$\mu$ in length, having the flagellum and blepharoplast at one end, while the macronucleus lies near the centre or near the other end.

Similar organisms (*L. infantum*) have been found in the Mediterranean area in cases of infantile splenic anæmia, which closely resembles Kala-azar and in a similar disease of dogs (*L. caninum*). The parasites discovered by Wright in Oriental sore also belong to the same genus (*L. tropica*), while a nasopharyngeal ulceration common in S. America is produced by a similar parasite (*L. braziliensis*).

## KALA-AZAR

### (*Black Fever*)

This disease is prevalent in India, Assam, and China, and has also been found in Tunis, Algiers, Malta, Arabia, Egypt, and the Sudan. The disorders known as tropical splenomegaly, Dum-Dum fever, and cachexial fever of Bengal are identical.

**Ætiology.**—Various species of herpetomonas or Leishmania have been found in the alimentary canals of flies, mosquitoes and bugs, and it is thought that some insect can convey *Leishmania donovani*, which causes Kala-azar, from man to man.

**Pathology.**—When the organisms first enter the body they enter the endothelial cells of capillary vessels; there they increase by fission to produce large numbers even in one cell. Escaping from these cells, they are taken up by the leucocytes, both polymorphonuclear and mononuclear, and reach the peripheral circulation. The further development of Leishmania appears to take place outside the human body.

**Morbid Anatomy.**—The conditions found after death, in addition to emaciation and muscular wasting, are enlargements of the spleen and liver, dropsical effusions, and ulceration of the large intestine. The spleen is large, firm, deep red in colour, with thickened capsule and trabeculæ; and he parasites are found in the cells of the pulp, and not generally in the Malpighian corpuscles. The liver is dark brown, or mottled brown and yellow, with a moderate degree of cirrhosis. The organisms are found in large endothelial cells, often in the dilated intralobular capillaries. They are never in the hepatic cells, which, however, are atrophied, and show nuclear and fatty degeneration. There are numerous parasites in the uninuclear cells of the bone marrow, which is converted into the red variety. Ulcers of various sizes are found in the colon and sigmoid, and cicatrices are common.

**Symptoms.**—The illness often begins with fever and presents an irregular, remittent or intermittent pyrexia. The fever usually lasts for three to six weeks, and then an intermission occurs for some weeks, and this is followed by more attacks of fever. Frequently the temperature chart may show two remissions in the course of twenty-four hours, and this feature has been regarded as almost diagnostic. Marked features are anæmia, earthy pallor of the skin, wasting and loss of strength, enlargement of the liver and spleen, occasional hæmorrhages from the nose, gums, or under the skin, pains in the ends of the long bones, and transitory œdema of the face and ankles, and even ascites in cases of much enlargement of the liver; diarrhœa, congestion of the lungs and pneumonia are also frequent, and the fatal result is often due to one of these, or to some other accidental complication. The condition of the blood is very constant. The red corpuscles are diminished to 60 or 50 per cent.; the hæmoglobin is diminished, and there is marked leucopenia, so that the leucocytes may be only 2,000 or 1,000 per cubic millimetre. The differential count shows great diminution of the polymorphonuclears and usually a large relative increase of the large mononuclears.

**Diagnosis.**—The Leishman-Donovan bodies should be sought for in the leucocytes of the peripheral circulation, but this may take a very long time, and search may be negative, or the exudate from an artificial pustulation of the skin may be examined (Cummins). Failing these, blood may be obtained from a puncture of the spleen, but this must be done with caution. Leukæmia and malaria must be excluded by examination of the blood, and typhoid by the absence of the agglutinative reaction.

**Prognosis.**—The disease lasts several months, and has been fatal in more than 90 per cent. of the cases.

**Treatment.**—The only specific treatment for Kala-azar now in general use is by antimony compounds. Tartar emetic is usually made up with sterile distilled water in a 2 per cent. solution and injected intravenously twice weekly for months on end. The initial dose is ½ grain of the salt for an adult, and the amount is gradually increased till the maximum tolerated dose, 2½ grains, is reached. Natives usually are less tolerant of tartar emetic than is the European; 100 or more grains may be necessary in order to effect a permanent cure, and, according to Napier, 60 grains per 100 lbs. weight of the patient should be the standard dosage. It is better to make up the solution fresh before each injection, and for doses above 1 grain, 10 c.c. of fluid should be taken. When the amount of diluent is restricted the resulting reactions, such as nausea, coughing and vomiting, are eliminated. Recent improvements in antimony compounds have given more rapid and satisfactory results, such as stibacetin and von Heyden 471—the individual dose of which is 3–5 grains. Great care must be taken during injection not to let any of the solution get into the tissues outside the vein, as thereby much local pain, swelling, and even sloughing may be caused. The effect of the treatment is soon seen in the increase in weight of the patient, the diminution in the size of the spleen, and the improvement in the blood condition. In children intravenous injection is often difficult, and Sir Leonard Rogers has substituted with some success the inunction of finely divided metallic antimony in lanoline.

Cutaneous Leishmaniasis has been treated successfully by similar methods, but in some cases X-rays, carbonic acid snow, and even injecting the sore with 1–2 grains of emetine, are equally efficacious.

## DYSENTERY

This is an inflammation of the large intestine, or colitis, and sometimes of the lower part of the ileum, resulting in extensive ulceration, and accompanied by fæcal discharges which consist almost entirely of mucus, muco-pus, or blood. The pathological changes and resulting symptoms are caused by the local invasion of living organisms, which are in some cases bacilli, in others amœbæ. Hence a division into bacillary and amœbic dysentery.

Generally speaking, in the tropics bacillary dysentery is the most widespread and important; it usually occurs in epidemics. Amœbic dysentery, on the other hand, is endemic and accounts for much ill-health and disability.

**Ætiology.**—Both the amœbic and bacillary types of dysentery depend on similar conditions for their prevalence. Infection in both forms occurs through the ingestion of the micro-organisms in food or drink. It has been found that dysentery is most prevalent at the seasons of the year when flies are most numerous. The stools of a dysenteric patient are highly infective, sometimes even though the patient may show no obvious symptoms. If flies have access to such stools they ingest amœbæ or bacilli, which remain in their alimentary tract. When the fly feeds it defæcates at the same time, and in consequence, when a fly carrying organisms settles on human food, it leaves upon it the dysentery organisms in the fæces. Dysentery therefore tends to become epidemic among populations where sanitation is defective, especially as far as disposal of human

fæces is concerned. Direct infection may take place from contamination of fingers by linen or vessels soiled by infected fæces. The disease may also be spread by an infected water supply.

The optimum conditions for the prevalence of dysentery are therefore found among armies in the field, unless strict sanitary precautions can be enforced. During the South African War of 1899 to 1902 the troops were decimated by the disease. In the recent European War comparatively few cases occurred in France, but in the Eastern theatres of war the disease was extremely prevalent during the spring and autumn months, when flies were numerous. The disease is endemic in most Eastern towns, where sanitation is defective and flies are innumerable. In England the disease is practically unknown, except in occasional outbreaks of a bacillary type in asylums.

## BACILLARY DYSENTERY

The bacilli which cause dysentery are known as *Bacillus dysenteriæ*, and several types have been described by Shiga, Kruse, Flexner, Schmitz, Sonne, and others. The main types are the Shiga-Kruse bacillus, which does not ferment mannite, and the Flexner bacillus, which ferments mannite. These bacilli are agglutinated by the serum of a patient suffering from bacillary dysentery, but not by the serum from an amœbic case. Five definite serological cases of the Flexner bacillus are now recognised and are known as V.W.X.Y.Z.

The bacilli grow and multiply in the intestine, and may indeed exist there for some time without giving rise to lesions. Usually, however, they form toxins which are absorbed into the blood; some of these are excreted by the bowel, and produce the dysenteric lesions: others may act upon the nervous system and cause various complications.

**Pathology.**—The lesions in bacterial dysentery are believed to be caused by the action of the toxins upon the tissues of the colon in the process of excretion. There is acute inflammation of the whole mucous membrane with areas of coagulation necrosis, which rapidly form shallow ulcers. The submucous, muscular and peritoneal coats of the intestine escape.

At an early stage the mucous membrane is intensely injected, reddened and swollen; the redness is often confined to the prominent edges of ridges of mucous membrane, or it may be irregularly distributed; and the surface is covered with mucus tinged with blood. The solitary follicles become swollen comparatively early, and after a time the surface of the follicle is abraded, and a little pit results. Later many parts of the surface, especially the summits of ridges or folds of mucous membrane, are covered with a whitish or green coagulated exudate or membrane, and the surrounding mucosa is hyperæmic and œdematous. Shallow ulcers of irregular shape and varying extent are then formed, which often at first do not involve the solitary follicles, but leave them surrounded by a little ring of mucous membrane. Ultimately these also may be separated and shed. The bowel then presents a red or yellowish-brown colour corresponding to the ulcerated parts, with the patches or islands of bluish-red or grey colour, representing the inflamed mucous membrane, still persisting.

In a later stage or severer form *gangrene* occurs, and patches of iron-grey, brownish-red, dark red, or black colour are met with, which evolve a gangrenous odour. Such patches may be quite small, or some inches in diameter. The bowel thus affected often contains a brown liquid of gangrenous odour, sometimes mixed with blood. When the sloughs separate they leave ulcers with undermined edges, which in favourable cases cicatrise. The mucous membrane between the sloughs or ulcers is hyperæmic and infiltrated with serum and blood.

The parts of the bowel liable to be diseased in dysentery are the rectum, colon, and cæcum; and the disease extends in some cases beyond the ileo-cæcal

valve to the lower part of the ileum. The disease is often advanced at one part while still only commencing in another. Early conditions are curable, and even from gangrenous stages recovery is possible if the lesions are not too extensive. Granulations spring up over the ulcers, and cicatrices result, which are at first below the level of the islets of retained mucous membrane; subsequently the surface becomes more uniform. Besides the changes in the lower bowel, there may be some catarrh of the stomach and small intestines. The mesenteric glands are swollen and reddened. The liver is swollen and hyperæmic; the spleen, as a rule, is small. The body generally is anæmic and wasted. Persistent suppuration of the dysenteric ulcers is the cause of *chronic dysentery*.

**Symptoms.**—In cases where it has been possible to fix the period of *incubation* this has varied from a few days up to eleven days.

In the *acute type* of the disease the patient is first taken with diarrhœa, and passes daily from two to five or six yellow or brownish-yellow loose fluid motions. There is some abdominal pain and a certain amount of malaise, with loss of appetite. After three or four days the stools become suddenly more frequent, so as to reach ten, twenty, forty, or sixty or more in the twenty-four hours, and they acquire the special characters of *dysenteric stools*. Each discharge is very scanty, and may not be more than a few drachms; but the total quantity in twenty-four hours may be from 30 to 50 ounces. The stools at the height of the disease contain no natural fæcal matter, but consist entirely of mucus, serum, blood, and pus, in varying proportions, with detritus and perhaps shreds of necrosed mucous membrane. In the early stages the discharges consist of yellowish transparent mucus, with small lumps or streaks of blood; in later stages blood is more abundant, in clots or lumps, floating in a red serous fluid. Sometimes pure blood is passed, either from early congestion of the mucous membrane or from ulceration of the walls of the vessels; pus may be present alone, or there may be a blackish or brownish-red offensive slimy fluid, containing portions of tissue which have sloughed away. Sometimes the stools contain curious tough masses of mucus, like frog's spawn or boiled sago. Two other frequent symptoms are griping abdominal pains and *tenesmus*, or the painful straining or desire to go to stool. The latter only occurs when the lower end of the rectum is affected, and it is accompanied by burning pains in the rectum and anus. It often results in no evacuation, or at most in a very small quantity. Frequent micturition is sometimes associated with it. These severe local conditions soon react upon the general health. The patient rapidly loses flesh and strength; the face is pale, sallow, or tinged with yellow; the tongue is covered with a thin fur; there is moderate fever, the temperature rising to 101° or 102°, with headache, dizziness, loss of appetite, and much thirst. An examination of the blood shows a diminution of the number of the red cells and a slight polymorphonuclear leucocytosis. In milder cases the symptoms abate after eight or ten days; the pain and tenesmus are less, and the stools gradually acquire more consistence, become more feculent, and are finally quite natural. In the severer cases the discharges are more and more mixed with blood and pus, or become greenish black and offensive (*gangrenous type*); while the patient's exhaustion increases, the motions pass uncontrolled, the anus and surrounding parts are excoriated, and death is preceded by collapse, with pinched features, livid extremities, hoarse voice, and scarcely perceptible pulse. Vomiting, which is an early symptom in many cases, becomes severe and continuous in the graver forms. The urine is scanty, wanting in chlorides, but not commonly albuminous.

Some cases pass into a condition of *chronic dysentery*: the stools are sometimes almost natural, at others consist of varying mixtures of mucus, pus, and blood, which have the same offensive odour as in the acute stages; and this may continue with more or less severity for months or years. The patient remains thin and weak, and may with judicious treatment recover, or may die eventually of

exhaustion, or as a result of such complications as peritonitis from perforation of the bowel or stricture from contraction of the cicatrix.

**Complications.**—The chief complications of bacterial dysentery are *iritis*, which is usually bilateral, and is the result of absorption of toxins, *arthritis* and *teno-synovitis*, *parotid buboes* and *abscesses* in different parts of the body, and *hæmorrhage* from the bowel in gangrenous cases. The arthritis usually involves one joint only and closely resembles a gonococcal arthritis; less often it is multiple. It occurs after acute dysenteric symptoms have ceased. A *peripheral neuritis* of dysenteric origin has been described.

**Diagnosis.**—The physician has first to be sure that the case is really one of dysentery, and then to determine whether it is bacterial or protozoal. Appeal is first made to the character of the stools, with the associated tenesmus and griping; but the two latter symptoms may be absent. Growths of the rectum and sigmoid can be excluded by rectal examination and sigmoidoscopy. The sigmoidoscope is of great value in the diagnosis of chronic bacillary dysentery, and as a guide to the success of treatment. The appearances are the same as those of ulcerative colitis (*q.v.*). The passage of blood in *intussusception* may also mislead. In the differentiation of bacillary and amœbic dysentery the complete absence of fæcal matter points strongly to bacillary infection, as does the presence of pyrexia. In the recent War dysentery, both bacillary and amœbic, was frequently associated with one of the paratyphoid fevers; and these fevers sometimes began with purely dysenteric symptoms.

In any suspected case of dysentery mucus from a very recently passed stool should be cultivated. Microscopic examination of the cellular exudate in the stools gives most valuable information; in bacillary dysentery polymorphonuclear pus cells predominate, while certain large refractive cells, known as *macrophages*, can be seen. Positive findings in bacillary dysentery are more frequent in the early stages of the disease, as when the latter has progressed for some days the dysentery bacilli tend to be overgrown by other organisms. A microscopic examination must also be made to exclude the presence of the dysentery entamœba. The agglutination reaction for Shiga's and Flexner's bacilli may also be tried, but is not so trustworthy, and cannot be obtained in the first seven days of the illness.

**Prognosis.**—Death in acute attacks of bacillary dysentery is comparatively rare, except in persons debilitated by hardship or other diseases. It is more likely to occur during exacerbations of infection in chronic cases. There is, however, considerable variation in the mortality in different epidemics. Infections due to Shiga's bacillus are generally more severe than the cases due to Flexner's bacillus.

**Prevention.**—This is the same as for amœbic dysentery.

**Treatment.**—The patient must be strictly confined to bed, be kept warm, and the use of a bedpan enforced. During the acute stages nothing should be given by mouth except small quantities of water or albumin water at frequent intervals. As the character of the stools improves a gradually increasing diet may be given. In cases of moderate severity the patient can usually take a light fish diet after ten days. Irritating substances, such as cabbage, should be avoided until convalescence is complete. Alcohol is contra-indicated in the acute stages, unless the patient is much disturbed by its absence.

Medicinally sodium sulphate should be given in drachm doses hourly. Under this treatment there is rapid relief of pain and a gradual improvement in the stools. After the first twenty-four hours the drug is usually given every two or three hours. Bismuth compounds should be avoided. The sodium sulphate should be continued in doses of 2 drachms twice a day until the stools are free from blood or mucus.

The treatment of bacillary dysentery has been revolutionised in recent years by the use of anti-dysenteric serum. This should be given as soon as the patient

comes under treatment if the case is severe and resembles bacillary dysentery clinically. Its administration must not be delayed pending culture of the stools. The dose should be large, not less than 60 to 100 c.c., and may be repeated in two or three days' time. The serum is polyvalent, and is usually followed by a most striking improvement within twenty-four hours. Small doses of 10 to 20 c.c. are useless. The serum is best given intravenously, especially in severe cases. It may, however, be injected subcutaneously into the flanks or intramuscularly.

If pain is very severe, it may be necessary to give morphia hypodermically. A hot-water bottle applied to the abdomen usually gives relief, and should always be tried before resort is had to morphia.

Cases which have become chronic respond to treatment much less satisfactorily than acute cases. A course of seven daily injections of serum should be given. Colon irrigations are beneficial in these cases. Perhaps the best solution to use is albargin (1 grain to the ounce). Solutions of quinine, tannic acid (4 grains to the ounce), and normal saline are also recommended. In severe cases appendicostomy and cæcostomy may be performed. If colon injections are employed, careful attention must be paid to the technique of their administration. No attempt should be made to pass the tube further than just into the rectum. The patient may lie on his left side, but the most efficient irrigation is obtained if the patient is strong enough to be in the knee-elbow position. The injection must be given slowly and at a very low pressure. It should be preceded by a saline colon wash-out.

## AMŒBIC DYSENTERY

Amœbic dysentery is caused by infection of the colon by a protozoon known as *Entamœba histolytica*. These organisms vary in diameter from 30$\mu$ to 10$\mu$, and are rounded bodies consisting of a refractile ectoplasm, which is protruded as pseudopodia, and endoplasm surrounding a small nucleus, usually excentric in position. It has been shown by Wenyon that the small forms sometimes known as *E. minuta* are merely a stage in the history of *E. histolytica*. As the acuter stages of the infection pass off the organisms form spherical cysts containing four nuclei.

**Pathology.**—Acute amœbic dysentery may affect the whole colon from rectum to ileo-cæcal valve, with large sloughing or gangrenous ulcers. These may be circular or may spread transversely to the length of the bowel; but they are always separated from one another by healthy mucous membrane. The appearance is different from acute bacillary dysentery, in which the ulceration, besides being more superficial, extends continuously over longer tracts of the colon. In chronic amœbic dysentery the ulceration is sometimes confined to the cæcum. The presence of the amœbæ in the submucous tissue causes infiltration and projection of the mucosa into the bowel here and there. Upon this projection a slough forms, and subsequently an ulcer. The ulcers extend deeply in various directions under the combined influence of bacteria and the amœbæ.

After death the large intestine shows œdematous thickening of all its coats, especially of the submucosa, gelatinous softening and suppuration of the latter, and formation and extension of ulcers by sloughing of the mucosa over it.

**Symptoms.**—The onset of amœbic dysentery is commonly extremely insidious, the first symptoms being attacks of diarrhœa alternating with constipation. Cases, however, of acute symptoms within a few days of infection are also met with. At first the stools are liquid and indistinguishable from ordinary diarrhœa. As the disease progresses they consist of fæces mixed with dark blood and some mucus. It is rare even in very severe infections for the stools to lose entirely their fæcal character. Constitutional disturbance is slight, and as a rule there is no pyrexia. Abdominal pain which is colicky in character is a marked feature, and as a rule tenesmus is present. The course of the colon is tender on palpation, and occasionally it may be felt to be thickened, especially in

the right iliac fossa. A leucocytosis up to 25,000 is usually present. Occasionally cases are of a severe gangrenous type, and black sloughs may be passed. The symptoms in most cases tend to improve after a time even if untreated, but only to recur with greater severity after a short interval. Relapse is extremely common even after efficient treatment, and those who have once had the disease are very prone to reinfection while in countries where the disease is endemic.

**Complications.**—The commonest complications are amœbic hepatitis and liver abscess (*see* p. 169). Amœbic abscesses of the lung, spleen, testes and brain have also been described. Perforation of the colon occurs rarely, and is invariably fatal, while intestinal hæmorrhage may take place from deep ulceration of the gut.

**Diagnosis.**—The dysenteric origin of the symptoms is determined by the considerations above mentioned, and by a microscopic examination of the fæces. Amœbæ are best found by examining on a warmed slide a piece of bloody mucus, or slough taken from fæces collected in a warmed bedpan, and free from urine; the amœbæ may be then living and active. A single negative examination for *E. histolytica* is of no value, especially if the patient has had any injections of emetine. The presence of many flagellate organisms, such as Lamblia or Trichomonas, should arouse suspicion of an amœbic infection.

It should be noted that the *E. histolytica* may be confounded with a non-pathogenic protozoon, the *E. coli*. The former has more rapid movements, no vacuoles, thicker pseudopodia, and often contains blood corpuscles in its interior. The cysts of *E. histolytica* may be distinguished from those of *E. coli* by the fact that they have only four nuclei instead of eight or sixteen. In the diagnosis of cases of chronic amœbic dysentery the sigmoidoscope is of great value. The mucous membrane looks normal, except for round red elevations on it; in the centre of each of these there is a small depressed yellow ulcer with a hæmorrhagic margin, at the point where the submucous abscess has broken through the mucous membrane. Flame-shaped hæmorrhages are also seen.

**Prognosis.**—Except in the gangrenous type of the disease and in chronic cases in debilitated subjects, the outlook as regards life is good.

**Prevention.**—The important part played by flies in the ætiology of dysentery has been already emphasised. It follows therefore that all possible measures should be taken to prevent flies breeding. As this object cannot as a rule be more than very incompletely attained, it remains to prevent the flies having access either to fæces or food and thus prevent them from carrying the disease. The former object is attained by care in the supervision of the disposal of fæces. It must be remembered that the stools not only of dysentery patients, but also of many apparently healthy persons, are infective. In camps, etc., latrines must be so constructed that the fæces are passed into vessels containing cresol or other disinfectant, and then destroyed either in an incinerator or by burial. Latrines should be well separated from cooking places, and situated if possible in such a position that the prevailing wind is away from dwellings. All food must be kept carefully covered either in wire gauze cupboards or under a fine mesh muslin, and should not be placed upon the table until the last possible moment. Kitchens should be provided with fly-papers or traps, and windows and doors rendered fly-proof. These comparatively simple precautions will do much to diminish the prevalence of dysentery.

All water in infected countries must be either boiled or chlorinated, but there is some doubt whether chlorination of water destroys the cysts. Fresh fruit or vegetables are to be avoided altogether or carefully washed in chlorinated water.

In practice it has been found impossible to deal adequately with the question of dysentery carriers, especially those who have cysts of *E. histolytica* in their stools. MacAdam has shown that among 2,000 men invalided from Mesopotamia for various causes over 30 per cent. were carriers of *E. histolytica* cysts. In view of such figures, it would seem to be more hopeful

to attempt the prophylaxis of amœbic dysentery by correct sanitation rather than by attempts to deal systematically with carriers, as only a comparatively small proportion of carriers ever have enough symptoms to bring them under observation.

**Treatment.**—As regards diet, nursing, and rest in bed, the same general principles apply in amœbic dysentery as in the bacillary infection.

As soon as a definite diagnosis of amœbic dysentery is made the alkaloid of ipecacuanha known as emetine should be administered. This may be given either intramuscularly or subcutaneously. For ordinary cases the latter is the method usually employed. It should be given in doses of 1 grain daily for twelve days. During this period the patient must be kept strictly in bed even though the symptoms may rapidly improve. Usually after three or four injections all blood and mucus has disappeared from the stools, and the bowels may tend to become constipated. This must be avoided by the administration of small doses of saline purgatives. After the course of 12 grains is finished, at least three specimens of the stools are examined for the presence of the cysts of *E. histolytica*. As these are derived from active amœbæ, their presence indicates that the disease is still uncured. Experience during the War has shown that emetine injections alone often fail to clear the stools of cysts. An attempt to do so should be made by the oral administration of emetine bismuth iodide. The drug is best given in cachets or gelatin capsules, 3 grains once a day for about twelve days. Unfortunately it is apt to cause nausea and vomiting. Sometimes the administration half an hour previously of 10 minims of tinct. opii may serve to avoid this. The patient must be kept on a very restricted diet, composed of milk, 3½ pints, toast, and, if necessary, one egg. If milk is not tolerated, meat extracts may be given. The drug should be given two and half hours after the last meal, and the patient must lie quiet for some hours afterwards and struggle against the desire to vomit. While emetine is being given the stools frequently remain liquid, as the drug acts as a mild intestinal irritant. If diarrhœa persists after emetine has been stopped, and if there are no amœbæ present in the stools, a bismuth mixture may be given and often quickly clears up the condition. Long after all active symptoms have disappeared the bowel remains unduly irritable, and any indiscretion in diet, more especially alcohol, is sufficient to produce an attack of diarrhœa. As a rule, those who have suffered from amœbic dysentery do well to avoid alcohol almost entirely for some years. Treatment of relapse is on the same general lines as that recommended above, though perhaps in mild cases it may be sufficient to give only six injections of emetine. Emetine has been shown to have a cumulative effect upon the heart and may cause extensive peripheral neuritis. No harm is done if the patient is confined to bed while the drug is being taken. Many cases of cardiac irregularity and even œdema of the legs have, however, occurred if the drug is given while the patient is up. In very severe cases of collapse with much loss of fluid due to the diarrhœa it may be necessary to give 3 or 4 pints of hypertonic saline intravenously, as recommended by Rogers for cholera (*see* p. 182). Unfortunately, a certain proportion of cases of amœbic dysentery are resistant to emetine and emetine bismuth-iodide.

It may sometimes happen that a definite diagnosis of either bacillary or amœbic dysentery cannot be made owing to lack of facilities for microscopical examination. In such cases, if severe and where there is any doubt, it is wise to give both anti-dysenteric serum and emetine. It must also never be forgotten that it is very common to find cases with a double infection. There is no evidence that in uncomplicated bacillary dysentery emetine is of any value.

Cases of amœbic dysentery who have had several attacks should cease to reside in countries where the disease is endemic. A mild colitis is apt to follow repeated attacks, and this renders the individual more liable to reinfection even if the original attacks have been apparently cured.

## AMŒBIC INFECTION OF THE LIVER

It has long been known that residents in tropical countries, in which dysentery is prevalent, are specially liable to suppurative lesions of the liver. Of recent years the close causal connection between tropical liver abscess and concomitant or antecedent amœbic dysentery has become abundantly clear. Not only do the statistics of liver abscess vary directly with the incidence of amœbic dysentery, but the *E. histolytica* can usually be demonstrated in the discharge from a liver abscess, though naturally an infection by amœbic dysentery cannot be proved in every case of liver abscess. This is not surprising in view of the fact that amœbic dysentery may be exceedingly insidious in onset, and frequently amounts to little more than occasional attacks of diarrhœa, symptoms, in fact, which few who have resided in the tropics for any length of time have failed to experience.

**Ætiology.**—Though amœbic dysentery is now always regarded as the principal factor in the production of liver abscess, especially the large solitary abscess sometimes known as tropical abscess, it is clear that from the sex and race incidence of the latter disease there must be some other factor. Statistics show that in India natives suffer more from dysentery and less from abscess than Europeans, and, further, that abscess is far more common in adult European males than in women and children. These variations may be well explained by the differences in the lives and habits of the two races and sexes, especially with regard to diet. The Indian and the European woman are much more moderate in diet, more especially in regard to alcohol, than the European male. The latter in tropical countries tends to take too much alcohol and too little exercise, both of which are factors which may well result in a congestion of the liver, thereby lowering its resistance to infection. This fact is borne out by experience, which tends to prove that amœbic infection of the liver is commonest among those who habitually indulge in considerable amounts of alcohol. Moreover, if the Indian takes to European habits in the matter of eating, and especially of drinking, he loses his relative immunity to liver infection.

**Morbid Anatomy.**—In the early stages of amœbic infection before abscesses are formed the liver may show only congestion and slight enlargement, with perhaps a few ill-defined greyish areas where the liver cells are undergoing necrosis. Microscopically, the earliest stage is seen to be clotting of the blood in the smallest branches of the portal vein, where the amœbæ have come to rest. This stage is called amœbic hepatitis. In a rather later stage the centre of one of these areas may be liquefied and a ragged abscess cavity formed. As the disease progresses the abscess cavity or cavities enlarge by necrosis of the surrounding liver tissue. Two types of abscess are found. There are the multiple small abscesses, and there is the large abscess, which is usually single, but sometimes two or three may be present. A single abscess is commonly situated in the upper part of the right lobe. Recent abscesses show no definite wall, but in rare cases they may become encysted, being surrounded by a smooth fibrous wall.

The content of an amœbic abscess is sometimes chocolate-coloured with streaks of blood. Sometimes it is green-tinged from admixture with bile. Unless secondarily infected, pyogenic organisms are absent, but amœbæ may almost always be demonstrated, especially in scrapings from the ragged abscess wall.

**Pathology.**—The amœbæ reach the liver through the portal vein. In the colon they are found in the submucous tissue, and hence may easily enter the radicles of the portal system. Liver abscesses can be produced experimentally in animals by rectal injections of pus containing amœbæ. *Post mortem* statistics show that the multiple small abscesses, varying from a walnut to a large pin's head in size, occur in cases where there is acute widespread ulceration throughout the colon, so that the liver is invaded by enormous numbers of amœbæ. On the

other hand, the large abscesses occur in more chronic states of ulceration often confined to the cæcum. Sometimes when a large abscess is diagnosed there is no evidence of dysentery at all; but *post mortem* examination has disclosed scars of old dysenteric ulceration, which was evidently the cause of the abscess. In a minority of cases liver abscesses are secondarily infected with other bacteria, such as staphylococcus, streptococcus, and *B. coli communis* (**20**).

**Symptoms.**—In few other diseases are the onset and course so variable as in amœbic infection of the liver. Many cases start with a gradual deterioration of health, which is so insidious that no definite date can be fixed for the commencement of symptoms. In others, however, definite symptoms of pain and fever may appear early and apparently suddenly. In cases where there is a definite history of dysentery the period which elapses between the disease and the development of liver symptoms varies widely. Exceptionally they may follow in a few weeks, more commonly after months or even years. From what has been said in the section on the morbid anatomy of the disease it will be understood that in all cases there is a presuppurative stage, known as amœbic hepatitis, which gives definite symptoms before, at any rate, an abscess of any considerable size has formed. The object of the practitioner should be to arrest the disease at this stage, and it is therefore well to summarise the symptoms which are most frequent

Of these pain is the most constant and usually the earliest to develop. It may begin as a feeling of weight in the epigastrium, but soon becomes a definite pain, localised usually to the right costal margin and over the liver area. It is aggravated by pressure on attempting to palpate or percuss the liver, and becomes exceedingly severe and lancinating in character if the right side of the thorax is suddenly compressed between the two hands. Fairly frequently pain may be referred to the right shoulder, but in these cases tenderness over the liver can always be elicited.

Amœbic hepatitis is usually, though not always, accompanied by pyrexia, which is higher by 1° or 2° in the evening than in the morning. Until an abscess has developed rigors and sweating are usually absent.

On examination the liver may be found to be somewhat enlarged both in an upward and downward direction, but, as a rule, rigidity of the upper part of the right rectus muscle prevents palpation of the liver below the costal margin. Some degree of leucocytosis is present, usually up to 20,000.

If these symptoms pass unrecognised or are treated as those of malaria, the condition of hepatitis will, as a rule, pass on to abscess formation. Rigors and drenching night sweats become prominent features, and the pyrexia tends to be intermittent in type. The complexion is muddy and cachectic, but actual jaundice is rare. Respiration is very shallow, and a cough is often developed. In a large proportion of cases signs of pleurisy and sometimes of consolidation are found at the right base. Lying on the left side usually aggravates the pain. In the cases where the right lobe is involved the liver dulness is found to be raised, the upper limit of dulness becoming dome-shaped in the right axilla. On X-ray examination the right side of the liver may be seen to be raised and the right diaphragm to be practically immobile. In late stages there may be œdema and local bulging in some of the intercostal spaces. In cases where the abscess is in the anterior portion of the liver it may be felt or even seen as a lump in the epigastrium, which is dull on percussion and tender on palpation.

**Terminations.**—If untreated the abscess may rupture spontaneously, most commonly into the lung, the pleura, or the peritoneum, rarely into the stomach or colon. In the case of rupture into the lung the pus is coughed up, and spontaneous recovery may occur. Apart from rupture or operative treatment, the patient usually dies of exhaustion after relapsing into a typhoid state.

**Diagnosis.**—Liver abscess can, as a rule, be definitely diagnosed by the

typical signs and symptoms as detailed above, but in every case the diagnosis can be confirmed by exploration of the liver with a wide, long needle attached to an aspirating syringe. If pus is not found at the first attempt, explorations in other spaces should be made. A local anæsthetic (2 per cent. novocaine with adrenalin) should be used for the skin. The liver puncture itself is not painful. If more than two or three punctures are required a general anæsthetic should be used.

*Differential Diagnosis.*—The commonest error in diagnosis in a case of liver abscess is to attribute the symptoms to malaria or pneumonia. The former can be excluded by the failure of the pyrexia to react to quinine properly administered, while signs suggestive of right basal pneumonia or pleurisy in a tropical country should be regarded as possibly due to liver abscess, especially if their onset has been insidious. A leucocyte count will aid in the diagnosis from malaria, as will also an examination with the X-rays. Whenever there is any reasonable doubt exploratory puncture should invariably be performed.

**Prognosis.**—In cases recognised early, where the condition is only one of hepatitis, the outlook with emetine treatment is invariably good. In undiagnosed cases or those in which there are numerous abscesses the outlook is bad, though vigorous treatment with emetine holds out some hope. The mortality of liver abscess treated by repeated aspiration is 14 per cent., or even less, but if treated by open operation the mortality is over 50 per cent., because secondary infection of the liver from the outside occurs.

**Treatment.**—If the patient is diagnosed before definite physical signs of abscess formation are found, emetine must be at once administered in daily doses of 1 grain subcutaneously. Usually within three days there is a marked improvement both in the pain and pyrexia. In such cases a course of 12 grains of emetine is given, and the condition is completely cured, or at any rate arrested.

In cases where an abscess has formed the pus should be all withdrawn through a wide aspirating needle, and the aspiration should be repeated or a further exploration should be carried out, if the temperature, etc., does not subside. At the same time a course of twelve subcutaneous injections of emetine should be given. There does not seem to be any advantage in injecting emetine into the abscess cavity. If the pus is too thick to be drawn through the needle, a trocar and cannula should be used attached to a Potain's aspirator. The pus obtained from exploration should be examined bacteriologically to see whether it is secondarily infected. After the pus has been removed the cavity becomes filled with serous exudation. It is not necessary to remove this unless it becomes purulent. Drainage of the abscess by open operation should only be carried out if the pus is found to be heavily infected with pyogenic organisms (**21**).

## MEDITERRANEAN FEVER

*(Malta Fever, Undulant Fever)*

This is a continued fever of long duration, bearing some resemblance to enteric fever, but distinguished from it by the absence of rose spots, the fact that Peyer's patches are not enlarged or ulcerated, and the low mortality.

The specific organism is a micrococcus (*M. melitensis*, Bruce), which can be obtained from the blood, from the urine, from the fæces, and by puncture from the spleen during life, and can be found after death in the spleen, liver, kidneys, bone marrow, and mesenteric glands.

**Ætiology.**—The disease is prevalent in Malta, and occurs in the other islands and along the coasts of the Mediterranean Sea ; and since its bacteriology has been studied it has been proved to exist in India, China, South Africa, the West Indies, and America. It affects the sexes about equally, but is more frequent in the young than in the old, especially between the ages of eleven and

thirty. It is more frequent in the warmer months of the year. It has been conclusively shown that the goats from which the milk supply is drawn in Malta, and other places concerned, are infected with the micrococcus, and that the use of goats' milk or goats' milk cheese as food is the most common cause of the spread of the disease. It may, however, be transmitted by contact of the milk with the abraded skin, and possibly by direct infection, or by means of blood-sucking insects.

**Morbid Anatomy.**—The spleen is large, often weighing as much as sixteen ounces, soft, and diffluent, with swollen and indistinct Malpighian bodies, and a great increase of lymphoid tissue; the liver and kidneys are congested, and the latter may be in a condition of glomerular nephritis. The mesenteric glands are slightly enlarged. Peyer's patches are to the naked eye normal; the most that can be seen with the microscope is a slight proliferation of the cellular elements.

**Symptoms.**—The *incubation* period is usually about fourteen days. The

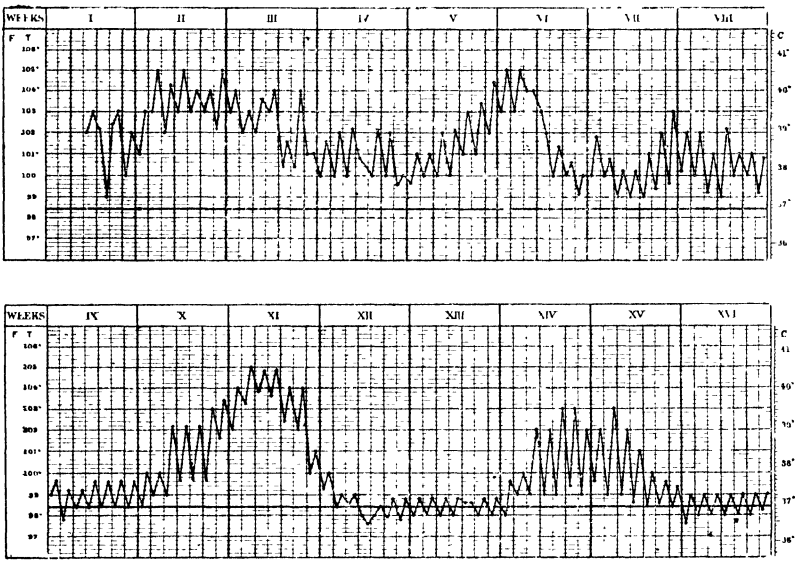



FIG. 16.—Temperature in a Case of Malta Fever of Undulant Type.

symptoms come on insidiously, and consist of pyrexia, headache, pains in the bones, sleeplessness, thirst, furred tongue, loss of appetite, nausea, and weight and tenderness in the epigastric region. The bowels are usually constipated; the spleen is always enlarged, and often tender or painful; sometimes the liver is enlarged; and profuse perspirations occur with crops of sudamina. The headache and severer symptoms may subside in two or three weeks, but the pyrexia continues much longer, even up to three, six, or nine months, and only slowly subsides. The temperature is not continuously high over all this time, but has exacerbations of two or three weeks, with intervals of a much lower or nearly normal temperature, so that a close resemblance to the chart of a relapsing enteric fever is presented.

Anæmia is generally marked, and the red corpuscles may fall below three millions, while there is a relative increase in the lymphocytes and mononuclears. In about half the cases the joints become red, swollen, and painful, and the micrococcus has been isolated from the effused fluid. Neuritis is also a common event, especially of the sciatic nerve, beginning acutely and persisting for a long

time in a less intense form. Children are liable to meningo-encephalitis. In some cases orchitis or epididymitis occurs. The urine is scanty, with uratic deposit and perhaps albumin in acute stages, but is generally normal at other times. Tachycardia or brachycardia are sometimes found. The disease commonly produces abortion.

The majority of the cases conform to the above or *undulant* type. Exceptionally there are acute or malignant cases, in which the temperature rises to 106°, 107°, or 108°, and death may take place from the twentieth to the thirtieth day. On the other hand, in some cases the patient is not ill enough to take to his bed, and in other cases the pyrexia may only last a fortnight. The mortality is 2–6 per cent., but is higher in young children and in people over forty owing to secondary infection with pyogenic organisms (**22**).

**Diagnosis.**—The diseases for which Mediterranean fever may be mistaken are the enteric fevers, malaria, rheumatism, tuberculosis, and septicæmia. The diagnosis can generally be made by agglutination tests: the serum of the patient agglutinates the micrococci sometimes as early as the fifth day in dilutions of 1 to 100 or 1 to 1,000, and this action persists far into convalescence, even for years. Another method is to cultivate the organism from the blood, from the spleen, or from the urine or fæces.

Recently a *paramelitensis* fever has been described, holding the same relation to Mediterranean fever as paratyphoid does to typhoid. In cattle another organism has been very commonly found, the *Bacillus abortus* (Bang), which is agglutinated by specific melitensis serum, but its infectivity to man appears to be slight, though cases of undulant fever caused by it are now being reported from Rhodesia.

**Prevention.**—This requires notification of cases of the disease to the proper authorities, frequent testing of the milk supplied by the goats, and segregation of the infected animals. The test employed is the "Zammit test," which depends upon the fact that the specific *agglutinins* are present in the milk, and will clump the micrococcus as in the Widal test for typhoid fever. Sedimentation (*see* p. 16) occurs on mixing equal quantities of a 1 in 10 dilution of the milk with an emulsion of *M. melitensis* in distilled water. Temporary protection appears to be provided to the individual who is exposed to infection by inoculation with a vaccine of the micrococcus. Milk should be boiled before being taken.

**Treatment.**—Neither quinine nor sodium salicylate has any influence on this disease. It may be treated in the same way as a mild enteric fever. Autogenous vaccines in carefully graduated doses have been used with success, but in the acute stages of the disease anti-toxic serum rather than vaccines should be administered. For the joint affection the use of iodine or friction with liniments is recommended (Bruce).

## SPIROCHÆTOSIS ICTERO-HÆMORRHAGICA

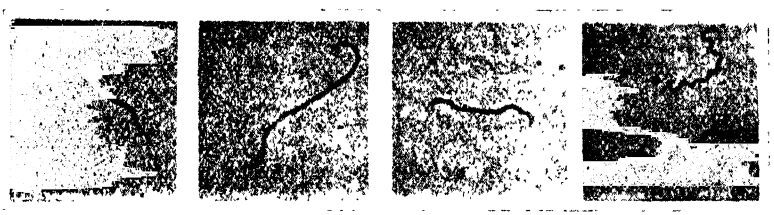
*(Infectious Jaundice, Weil's Disease)*

This is a febrile disease in which jaundice is the prominent feature and in which a spirochæte, the *Leptospira ictero-hæmorrhagiæ*, is found in the liver, kidneys, and urine (*see* Fig. 17, A, B, C.).

Jaundice, though sometimes due to coarse mechanical causes, such as blocking of a bile duct (*see* Diseases of the Liver), is also closely related to different kinds of infection. It is an essential part of yellow fever; it is an occasional event in relapsing fever, in typhoid and paratyphoid fevers, and in pneumonia, when the organisms or their toxins may be supposed to be specially directed to the liver or to the blood, so as to produce this complication. But there occur other cases of fever and jaundice, which cannot be included in the above, which are often

epidemic, which are rarely fatal, and which have received various names, such as febrile jaundice (Lancereaux), epidemic jaundice, infectious jaundice, infective jaundice, camp jaundice, and hepatic typhoid (Landouzy). It is probable that many cases of catarrhal jaundice are really due to infection, and not solely to obstruction of the common bile duct.

One form of what may be called infective jaundice, described by Weil, of Heidelberg, in 1886, has been recognised as occurring in different parts of the world, *e.g.* Germany, Egypt, India, and the United States, but rarely in England. Weil stated that it appeared in the hot seasons, that it was common in young adult males. The onset was sudden, often with a chill, and the symptoms were fever, headache, signs of gastric disturbance, jaundice, and muscular pains, especially in the calves. The fever lasted eight or ten days, and sometimes there was a relapse. The spleen and liver were commonly, but not always, enlarged, and the liver was often tender on pressure. Nephritis was often, and herpes and erythema occasionally, observed. Deaths were comparatively rare. The *Bacillus coli communis* was by some regarded as culpable, and in some fatal cases of Weil's disease the *B. proteus fluorescens* is said to have been obtained from various organs. In 1916 in cases apparently identical with Weil's disease Japanese observers



A B C D

FIG. 17.

A. *L. ictero-hæmorrhagiæ*, from blood of inoculated guinea-pig.
B, C. *L. ictero-hæmorrhagiæ*, from human blood on second day of the disease before jaundice had appeared.
D. *Spironema pallidum* (upper figure) and *Spironema refringens* (lower figure), from primary syphilitic sore. Magnitude 2,500. (*After A. C. Coles.*)

discovered a spirochæte, which is called the *Leptospira ictero-hæmorrhagiæ*. During the War a limited epidemic of jaundice occurred among troops in Flanders, which was shown to be due to infection by a similar organism (**23**). It is now generally admitted that spirochætosis ictero-hæmorrhagica and Weil's disease are the same.

Both in Japan and in the war area in France the spirochætes have been found in the blood, kidneys, and urine of rats, while recently it has been shown to be comparatively common in dogs, in whom it causes a rapidly fatal icteric disease. Further, it was observed that in France the disease was only contracted by men occupying certain wet sectors of trench. Moreover, rats caught in these sectors were found to be infected by spirochætes, which produced jaundice and hæmorrhages in guinea-pigs. From the evidence it would appear that infection spreads to man from the urine of the rat. It has been shown experimentally that infection can occur through an abrasion or even through undamaged skin. Possibly rats infected the water in the trenches and the spirochætes entered the body through the wet skin, or in the drinking-water. In this country the spirochætes have been found in rats, *Rattus norvegicus* (**24**), and one case has occurred after immersion in the Thames (**25**).

**Morbid Anatomy.**—The post-mortem appearances in different fatal cases have been as follows: Intense congestion and swelling of the mucous membrane of the duodenum, with swelling of the papilla at the entry of the bile duct, were

found in some fatal cases, but in others this feature was absent; the liver often with a greenish tinge, but otherwise normal in appearance; the spleen of normal size or smaller; the kidneys large and swollen and presenting many hæmorrhages occupying the cortical tubules: the lungs showing hæmorrhages, especially under the pleura, and sometimes pneumonic patches; petechial and larger hæmorrhages under the pleura, pericardium, and peritoneum; a soft flabby heart.

Under the microscope the liver shows polymorphonuclear cells in the interstitial tissue round the smaller bile ducts; and the kidneys present, besides the hæmorrhages, swelling and granular degeneration of the tubular epithelium.

**Symptoms.**—The onset is often sudden, and the temperature rises to 103°, 104°, or 105°, and the patient has pains in the head, eyes, limbs, and hypochondrium; he is soon very ill and weak, with anorexia, nausea or vomiting, sleeplessness, and even delirium. The temperature remains febrile for seven, eight, or nine days, gradually falling by lysis or more rapidly to the normal. On the fourth, fifth, or sixth day jaundice appears: sometimes it is slight, at others more intense; the liver may be enlarged and tender; and rarely the spleen is similarly affected. The pulse, at first quick, becomes relatively slow when jaundice appears. There is no alteration in the respiratory system Commonly there are albumin and casts in the urine. The organisms (*L. ictero hæmorrhagiæ*) may be found in the deposit from the tenth day onwards. The stools, often loose at first, later become infrequent and pale. The lymph glands are often enlarged, especially those of the pectoral region, of the neck, and the groin. The conjunctivæ are often deeply injected. The jaundice lasts from seven to nine days, and the other symptoms clear up as the temperature falls. In many cases there is a relapse of the pyrexia about the sixteenth or twentieth day; but this is often not accompanied by a second appearance of jaundice, and is milder than the primary fever. Numerous slight complications may be seen, such as erythema, urticaria, herpes; the most important are hæmorrhages in the form of epistaxis, petechiæ on the skin, conjunctiva, or mucous membranes, blood in the stools, hæmaturia, and hæmoptysis.

**Diagnosis.**—The disease may be confounded with the enteric (typhoid and paratyphoid) diseases when these are accompanied by jaundice; the agglutination tests of the enteric forms will serve to distinguish them. Moreover, the jaundice in the enteric forms occurs much later than it does in spirochætosis. In a few cases the spirochæte may be found by blood examination after prolonged search between the fifth and ninth days of the disease.

Guinea-pigs may be inoculated with blood from the patient; if infection takes place, the animal develops jaundice and numerous hæmorrhages beneath the skin and serous membranes, in the post-peritoneal fat, in the lungs, kidneys, adrenals, and occasionally in the muscles in about ten days' time. Spirochætes are seen in the liver stained by Levaditi's method. The guinea-pig must be inoculated before the fifth or sixth day.

The mortality is low; and the treatment at present is only expectant.

## YELLOW FEVER

Yellow fever is an acute specific disease, occurring within certain geographical limits, and characterised by fever of short duration, a yellow tint of skin, severe gastro-intestinal disturbance, and albuminuria, or suppression of urine.

**Ætiology.**—This disease was first met with in 1647 in the West Indies, and is peculiar to that group of islands, to North and South America, and to the west coast of Africa. It has, indeed, been occasionally carried to other parts of the world—for instance, in 1865 to Swansea, where a slight epidemic was the result; but it has never maintained itself away from the localities mentioned, which lie between the latitudes of 48° north and 35° south. It occurs almost

exclusively in crowded towns, and especially in those having a maritime commerce; and for the most part it is confined to low levels, and is rarely found higher than 2,000 feet above sea level.

It requires a high temperature, not less than 70° F. or 72° F., but it has been known to occur at lower temperatures, *e.g.* 65°. It is stated to be stopped absolutely by cold sufficient to freeze the earth. It is most fatal in the summer months—from May to August.

It affects all ages and both sexes, but it is more frequent and more severe in adults than in children. Negroes are certainly not completely immune, and they sometimes suffer severely; but more often they have it in a very mild form. This relative immunity was attributed by Sir R. Boyce not to a racial difference, but to the long contact of an indigenous people with an endemic disease. It does not usually occur a second time in the same person.

The infecting agent of yellow fever is transmitted from man to man by means of a species of mosquito, the tiger or brindled mosquito, *Aedes argenteus* (formerly known as *Stegomyia fasciata*), in the same way as the organisms of malaria and sleeping sickness. The mosquito dwells in towns, and not in marshes or swamps; and it breeds in clear water receptacles in the yards of houses, in cisterns, barrels, and tins used for the storage of water, in old bottles, meat and milk tins, flower-pots, etc., and in water collected in canoes.

In 1918 Noguchi (**26**) isolated from the blood of yellow fever patients a filter-passing organism, a spirochæte (*Leptospira icteroides*) which is indistinguishable morphologically from the *Leptospira* found in spirochætosis ictero-hæmorrhagica, a discovery which is not surprising, in view of the very similar symptoms of the two diseases. Injection of blood from a yellow fever patient into a guinea-pig causes death with hæmorrhagic symptoms, and the spirochæte can be recovered from the blood, liver and kidneys of the animal. Immunity reactions have shown that the *Leptospira icteroides* and the *Leptospira ictero-hæmorrhagiæ* are not identical organisms. Another morphologically indistinguishable organism is the *Leptospira hebdomadis*, which is the cause of a mild seven-day fever in Japan, with no tendency to jaundice.

**Anatomical Changes.**—The changes described are anæmia and acute fatty degeneration of the liver; acute catarrh of the stomach, with ecchymosis, or hæmorrhagic erosions; hæmorrhages in the tissue of the lungs and under the pleuræ; pale, yellow-brown colour of the muscular substance of the heart or acute fatty degeneration or ecchymoses; and acute glomerular and parenchymatous inflammation of the kidneys with fatty degeneration and sometimes miliary abscesses, causing a high degree of urea retention. The spleen differs strikingly from that of malarial disease in being usually quite unaffected. The hæmoglobin is diminished, and there are a few normoblasts. The leucocytes are often diminished, and if there is an excess it concerns the polymorphonuclear cells, and not the mononuclears. The fatty changes in liver and kidney are much more marked than in spirochætosis ictero-hæmorrhagica.

**Symptoms and Course.**—The period of *incubation* is about five days; that is to say, the symptoms appear approximately five days after the person has been bitten by an infected mosquito.

Sometimes suddenly, sometimes after a short period of languor, headache, or malaise, there are chills or rigors of more or less severity. These are followed by febrile reaction, the temperature rising in two or three days to 105°, or even higher. There are generally frontal headache and severe lumbar pains or pains in the joints. The pulse varies from 100 to 120, mostly not so quick in proportion to the temperature as in some other fevers. The tongue is generally covered with a thick creamy fur, leaving the edges and tip bright red. There are mostly tenderness and pain in the epigastrium, with nausea or vomiting. About the third day the conjunctivæ become yellow, and jaundice spreads to the whole body. The urine is scanty, and it constantly contains albumin, which may be

found as early as the second day of the illness. Bile pigment appears a few days later.

On the third or fourth day there is often a remission of temperature, and the general pains subside. This may be the commencement of convalescence, the yellow tint gradually clearing up, albumin disappearing from the urine, and the patient recovering in two or three weeks.

But in many cases the more serious symptoms continue. The temperature rises again to 103° or 104°; but the pulse remains slow, and even gets slower in proportion to the severity of other symptoms (Faget's sign). The jaundice deepens, and petechiæ appear under the skin; the urine is still less in amount, while the albumin increases, urea is diminished, and casts are present; and, finally, complete suppression for days may occur. The vomiting becomes frequent. At first only the gastric contents, mixed with more or less bile, are discharged, but afterwards the so-called *black vomit* occurs, due to the presence of blood, often in a form which is likened to coffee grounds. This is sometimes preceded by a limpid, ropy, opalescent fluid—*white vomit*. When blood is discharged by the stomach, it is generally passed by the motions as well. Hæmorrhage may take place also from the nose, mouth, or gums; and the tongue by this time has lost its fur, and becomes dark brown, raw, and covered with blood crusts or sordes. Delirium becomes pronounced, or the patient may sink into coma or fatal collapse.

The mortality varies from 5 to 75 per cent. in different epidemics. Death takes place sometimes within a few hours of the onset in the first paroxysm, more often after the remission of fever, either from collapse, from profuse hæmorrhage, or from coma, which is generally attributed to suppression of urine and uræmia. The temperature sometimes rises to 108° or 110° immediately after death.

**Diagnosis.**—There is generally little difficulty about the diagnosis in localities where the disease is prevalent. The early acute symptoms may present a certain resemblance to the onset of such an illness as small-pox or pneumonia. Later it has mainly to be distinguished from *malarial fevers*. These last are endemic, and not transmissible so as to affect healthy persons in new localities: they present intermissions of actual health, or, at least, remissions between the exacerbations, which recur with regularity; the spleen is enlarged, and the individual is not protected by his illness from future attacks.

Relapsing fever may be accompanied with jaundice, but the primary fever is longer, and the interval is one of very great improvement; the spleen is enlarged. Acute yellow atrophy of the liver begins more gradually, often with an apparently simple jaundice. Yellow fever is more rapid in the evolution of its symptoms, and has a higher mortality than spirochætosis ictero-hæmorrhagica, but a severe case of the latter, with "black vomit" jaundice, nephritis and bradycardia, is indistinguishable from yellow fever, and, similarly, mild yellow fever will resemble spirochætosis ictero-hæmorrhagica.

Guinea-pigs should be inoculated with blood from patients early in the disease.

**Prognosis.**—The unfavourable signs are a very high temperature, abundant albuminuria, suppression of urine, black vomit, or pronounced nervous symptoms.

**Prevention.**—This should be carried out by the same methods as are employed for malaria, namely, the protection of the individual from the bites of mosquitoes and the extermination of these insects. For the latter the first method used was to pour petroleum on pools to destroy the larvæ, but now certain small fish are used with equal success and less expense. Complete success has attended these measures in Havana, New Orleans, Panama, Rio and some other infected localities. Notification is, of course, necessary, and quarantine for five days after infection of those who are not immune is also

recommended, so that yellow fever is at present practically extinct in South America.

Preventive vaccination with killed cultures of the organism is highly successful.

A yellow fever patient can infect mosquitoes during the first three days of his illness; and a mosquito so infected can, after the lapse of about ten days, transmit the disease to healthy persons. It retains this power of transmitting the disease for many weeks.

**Treatment.**—Three important points in the treatment of the disease in the acute stage are not to move the patient, allow him plenty of water and no food. Anti-icteroides serum prepared by inoculation of the horse, when given before the fourth day, has reduced the mortality from 52 to 13 per cent. It produces rapid alleviation of subjective and objective clinical symptoms.

## DENGUE

Dengue, or dandy fever, is a disease occurring only in or near the tropics. It begins with fever of short duration, accompanied by pains in the joints and limbs; then, after a short interval of apyrexia, there is a relapse or second fever, and often a cutaneous rash.

Epidemics of this disease have been observed in India, Burmah, Persia, in Egypt and other parts of Africa, in North and South America, and the West India islands. The disease is usually transmitted by the bite of a mosquito *Aedes argenteus*, formerly known as *Stegomyia fasciata*, and the distribution of the disease is almost identical with that of the insect. The disease has also been produced experimentally by the intravenous inoculation of blood from an infected person. No organism has been detected, but there is experimental evidence that the disease is caused by a filter-passing virus.

**Symptoms.**—The period of *incubation* is from three to six days. The patient is suddenly seized with pain, usually in the legs or lumbar region, and with headache and fever. Occasionally the joints are affected, but more usually the seat of the pain is in the muscles. As a rule there is no redness or swelling of the joints, and the pains shift from one part to another, as in rheumatic fever. The headache is accompanied by pains in the eyeballs. The temperature rises to 102°, 103°, or even 105°, and the pulse is commonly a little over 100. In many cases also, this first fever is accompanied by a rash, either redness of the face or a general red colour; and the throat may be sore. But the rash disappears in twenty-four hours, and about the same time the pyrexia terminates—sometimes suddenly with critical symptoms, such as sweating. The pains abate, and the patient is in comparative comfort, but weak for two, three or four days; he then again becomes feverish, and a rash appears, which is either diffused like scarlatina, or maculated like measles. It begins on the palms and the backs of the hands, and spreads to the whole of the body. It lasts from a few hours to two or three days, and is followed by desquamation. In this second fever joint pains again occur, and may persist or relapse after the subsidence of the fever. The whole duration of the disease is about eight days. In some cases the joints remain painful and swollen for months after the fever has subsided. Leucopenia is a constant feature during the attack. It is practically never fatal, except perhaps in the case of infants and old people. Mild forms may present only malaise, sort throat, and the second or *terminal* eruption.

**Treatment.**—After attention to the bowels, salines and diaphoretics are recommended for the general condition. For the joint pains Dover's powder may be given, or small doses of antipyrin, aspirin, or phenacetin. Heat of skin may be relieved by cold sponging, and the irritation of the second rash by the use of camphorated oil. During convalescence iron should be given.

# CHOLERA

(*Asiatic Cholera*)

The name *cholera* is given to an acute specific disease, of which the principal features are the profuse discharge of watery evacuations from the bowels, vomiting, collapse, cramps in the calves and feet, and suppression of urine. It is constantly present in India, where even in the present day many thousands die of it annually. It has spread from time to time during the last hundred years to Europe, but has never obtained a permanent hold. England experienced severe epidemics in 1832, in 1849, and in 1854, and a milder and more restricted outbreak in 1866.

**Ætiology.**—Koch described in 1883 the *comma bacillus*, or *spirillum*, or *vibrio* of cholera. It is found in the rice-water evacuations, in the contents of the intestine after death, and in the mucous membrane of the intestine, just beneath the epithelium; but it has not been found in the blood. It is a little shorter than the tubercle bacillus, slightly curved, somewhat thicker in the middle than at the ends, and flagellated at one or both ends. The organisms grow in the interior of the intestine, as well as in the glands, epithelial cells and mucous membrane itself; they are believed to produce an endotoxin, which on being liberated causes the symptoms.

Although the cholera vibrio can be shown to be invariably present in every true case of cholera, it does not follow that every person infected by the vibrio must develop the disease. Numerous cases are on record of apparently healthy subjects who have never had any symptoms of cholera who pass large numbers of virulent vibrios in their stools. It would seem that the presence of some additional factor is necessary for the development of the disease, such as under-feeding, or more especially some derangement of the functions of the alimentary canal causing enteritis. Cholera is primarily a water-borne disease. The cholera vibrio has been proved not only to live, but to multiply in water, and in fæces it may persist for several months. The stools of cholera patients are highly infective, and may contaminate bedding, clothes, etc., or the hands of attendants. Defective sanitation may lead to pollution of the water supply. This was well illustrated in London in the severe epidemics of 1854 and 1866, and in India the spread of the disease is often determined more or less by the direction of the rivers. It has also been shown that *flies* can convey the vibrios to food substances, and the occurrence of outbreaks due to *cholera-carriers* is now clearly recognised.

Cholera is more prevalent in low-lying areas than in hilly countries, but epidemics may occur in any district where bad sanitation allows the contamination of drinking water. The disease is endemic in many parts of India, but the most favourable periods for epidemic spread appear to be the summer and autumn months.

**Morbid Anatomy.**—Decomposition proceeds slowly in those dead of cholera, and *rigor mortis* persists a long time. The right side of the heart is often distended with blood, the lungs are engorged, and the mucous membrane of the trachea and large bronchi is congested. There is often purulent mucus in the minute bronchi, and in cases dying in late stages there may be hæmorrhagic infarcts. The *intestine* contains in earlier stages rice-water fluid, in later stages liquid tinged with green. The mucous membrane is congested, and frequently sodden and pulpy, and the Peyer's patches and solitary follicles are swollen and prominent. The lower end of the ileum is the part most affected. The *spleen* is small. The *kidneys* are large, and show proliferation of the epithelium and cloudy swelling. Later on casts form in the tubules, the organs become paler, and fatty degeneration takes place.

**Symptoms.**—The *incubation* period is from a few hours to a week, most

usually about two or three days. The onset is usually sudden, but the patient often has been suffering from diarrhœa for a few days previously or has been in ill-health. With the onset of the disease diarrhœa becomes extreme, and usually within a few hours the stools lose their fæcal character and are of the typical rice-water appearance. A quart may be passed in a few minutes, and three or four times that amount in two or three hours. The fluid is neutral or slightly alkaline, of specific gravity 1,006 to 1,013, containing sodium chloride, albumin and mucin. On standing it deposits a finely granular whitish-grey sediment, consisting of epithelium, leucocytes, shreds of tissue, crystals of ammonium-magnesium phosphate, bacteria, vibrios, threads of algæ, and blood corpuscles. The purging is accompanied by borborygmi and gurgling, but by little pain or griping. After one or more hours of purging vomiting sets in; at first food is rejected; then large quantities of a watery or whey-like fluid, like the intestinal discharges, are vomited with comparative ease, as if regurgitated. The patient suffers from anorexia and thirst, the tongue is white and may become dry, and the epigastrium is sensitive to pressure. About the same time, in most cases, there are severe and extremely painful cramps in the calves of the legs, in the feet, and less often in the hands and trunk. Soon the patient sinks into *collapse*—the *algid* stage. The surface of the body becomes cold and livid, the hands, feet, face, and nose are pinched and blue, the eyes are sunken, and the breath is cold; the axillary temperature falls 4° or 5° F. below the normal, while in the mouth it may be even lower still. On the other hand, in the rectum and vagina it is raised, sometimes even reaching 105° or over. In severe and fatal cases the eyes become dry and the cornea cloudy. The pulse is small, thready, almost imperceptible, numbering from 90 to 100. Respirations are short and quick, from thirty-five to forty in the minute. There is great muscular prostration, but the patient is restless, throwing his limbs about; the voice is hoarse, or sinks to a whisper (*vox cholerica*), or only the lips are moved in the attempt to speak. Purging often ceases during collapse, but vomiting continues. The urine becomes scanty, and is often entirely suppressed—a condition which may begin quite early, and last thirty-six or forty-eight hours; it is probably only a result of the failing circulation. The blood pressure is found to fall to 50 or 60 mm. of mercury; the blood itself is much concentrated, the specific gravity is from 1,060 to 1,072, the red corpuscles reach as much as 8,000,000 per cubic millimetre, and the hæmoglobin and leucocytes, especially the large mononuclears, are correspondingly increased. The patient generally retains complete consciousness, though lying apathetic and indifferent, except when aroused by the pain of cramp. This stage begins six or seven hours after the first symptoms, and lasts twelve or twenty-four hours, when the patient may die without rallying.

In cases which survive the collapse there is a gradual rise of skin and mouth temperatures, sometimes to 101° or 102°; the skin begins to regain its natural colour, and loses its shrunken appearance; the cramps and restlessness cease; the pulse improves, and may become slower than in health; urine is again secreted, but frequently contains albumin and casts. The face becomes congested with patches of dusky redness, and the conjunctivæ are injected. This is described as the stage of *reaction*, and with subsidence of the temperature it often goes on to recovery.

**Varieties.**—The variations from this more usual course are many. Milder forms occur in which the disease does not pass beyond the first stage of diarrhœa. Sometimes diarrhœa does not even confine the patient to bed, but bacilli are nevertheless found in the motions; these may be called *ambulatory* cases, and contribute to the number of cholera-carriers. A more severe form, *choleraic diarrhœa*, begins suddenly, after exposure to cold or some error in diet, with profuse but painless diarrhœa, the motions being abundant, fluid, of yellow or yellowish-brown colour, containing epithelium, crystals of ammonium-magnesium phosphate and biliary constituents. There are from two to six or eight

motions in the day, and they are attended with borborygmi, and sometimes with cramps of the calves. The diarrhœa lasts from a few days to one or two weeks.

*Cholerine*, a still nearer approach to the severer attacks, also occurs suddenly and unexpectedly, vomiting accompanies the purging, the motions often become colourless, and there may be cramps, some cooling of the extremities, scanty urine, and albuminuria. Recovery is slow.

Sometimes the stage of *collapse* may set in at once, and the patient may sink before any purging has taken place, though there is abundance of fluid in the alimentary canal (*cholera sicca*). And in other cases beginning as usual with the diarrhœal stage the collapse may be very short, the patient dying suddenly from respiratory or cardiac failure; or in others, again, the algide condition is prolonged to thirty-six or forty-eight hours.

In some cases the patient may pass into the typhoid state with toxic rashes, which may be erythematous, papular, or hæmorrhagic. The urine is much diminished, or there may be complete anuria. Albumin and casts are present, and the patient dies with the symptoms of uræmia.

If the stage of reaction goes on to recovery, convalescence is usually slow and may be delayed by complications.

**Complications.**—During the stage of collapse there may occasionally be hyperpyrexia. Complications occur during the stage of reaction and are due to secondary infections. The most common are pneumonia and nephritis, less common parotitis, gangrene, and corneal ulceration.

**Diagnosis.**—Correct diagnosis is of very great importance, and, except when the disease is epidemic, may be a matter of extreme difficulty. From a clinical point of view the most important signs of cholera are diarrhœa with rice-water stools followed by vomiting, a big difference between skin and rectal temperature, and profound collapse. Cholera has to be distinguished from the following diseases:

1. *Diarrhœa.*—In this there are no symptoms of collapse, no cramps, and the stools remain fæcal, but it must be remembered that, apart from bacteriological findings, a mild attack of cholera may exactly simulate ordinary diarrhœa.

2. *Bacillary Dysentery.*—Here collapse in severe cases may simulate cholera very closely, but usually the presence of much blood, mucus, or pus in the stools leads to a correct diagnosis.

3. *Food Poisoning.*—In this disease vomiting as a rule occurs before the onset of diarrhœa. When this is so the diagnosis of cholera can be ruled out. The internal temperature does not usually rise in food poisoning, and a difference of 5° between skin and rectal temperature in a suspected case is almost pathognomonic of cholera. Collapse and muscular cramps may often be quite as severe as in cholera, and the macroscopic character of the stools can be exactly similar.

4. *Meningitis.*—Cases of cholera sicca are often indistinguishable from meningitis except after lumbar puncture. Rigidity, retraction of the abdomen, and a positive Kernig's sign may be present in both diseases.

5. *Malignant Malaria*, with profuse choleraic diarrhœa, frequently resembles true cholera. Suspicion should be aroused by its sporadic character as well as by the enlarged spleen. Parasites of malaria are usually easily detected in the peripheral blood.

Where facilities are available every suspected case should be subjected to bacteriological examination. Films made from a shred of mucus from the fæces stained with carbol fuchsin may show vibrios, but the diagnosis must always be confirmed by cultural tests or by agglutination tests with a known anti-cholera serum.

**Prognosis.**—When cases are treated in hospital with modern methods the

mortality is as low as 11 per cent. (Rogers). During epidemics very few cases can be thus treated, and the mortality varies from 40 to 60 per cent., and is always greater at the beginning of an epidemic than towards the end. It is more fatal to the very young and to the aged, to those who are in ill-health, are debilitated by insufficient nutriment or bad hygienic conditions, or are the subjects of chronic alcoholism. The unfavourable symptoms are profuse and violent discharges, rapid prostration, with much cyanosis, shrivelled and cold skin, profuse cold perspiration, and absence of pulse at the wrist.

**Prevention.**—Since cholera is spread in much the same way as enteric fever, the same methods must be adopted to prevent it (*see* p. 89).

In the Eastern theatres of war prophylactic injections of vaccine have been employed on a large scale, 1 c.c. of vaccine is injected subcutaneously. The local reaction is slight. Probably the immunity conferred lasts only a few months, and vaccination is therefore not performed until cases of cholera occur.

**Treatment.**—In any case of diarrhœa suspected of being cholera, or even if it occurs during a cholera epidemic, no purgative of any kind should be given, as it may do grave damage. Doses of tinct. opii or chlorodyne may be given, but if any signs of collapse appear these drugs must be withheld.

Of late years Sir Leonard Rogers has used intravenous injections of hypertonic saline and subcutaneous injections of atropine in the treatment of cholera. The patient on admission is at once given $\frac{1}{100}$ grain of atropine hypodermically, and this is repeated night and morning. The systolic blood pressure is taken, and also, if possible, the specific gravity of the blood. If the pressure is below 90 mm. of mercury or if the specific gravity is 1,063 or over, an intravenous injection should be given at once. A sterile solution of sodium chloride 120 grains, calcium chloride 4 grains, and distilled water 1 pint is used. Up to 4 pints may be injected. The temperature of the fluid varies with the rectal temperature. If this is below 99° F., the saline should never be injected at above 98° F., for fear of producing hyperpyrexia. If the rectal temperature is 100° or over, the fluid should be given between 80° and 90°. It is almost always necessary to cut down on a vein and insert a cannula. Some authorities recommend leaving the cannula in position for later injections, but this usually results in the blood clotting, and it is probably better to use another vein for subsequent injections. Not infrequently during the infusion a rigor occurs. This is not an indication for stopping the injection of saline. As a rule the infusion rapidly relieves the cramps, raises the blood pressure and diminishes the specific gravity of the blood. The indications for further injections are as stated above, but as a rule it has been found more efficacious to give a pint of the following alkaline solution: sodium chloride grains 60, sodium bicarbonate grains 120, followed by 2 pints of normal or hypertonic saline. The administration of the alkaline solution is specially indicated in cases with suppression of urine. Ordinary normal saline may also be given *per rectum*.

Tomb and others have reported favourable results with a mixture of the essential oils in the treatment of cholera. A mixture of the following formula is given:

| | |
|---|---|
| Sp. æther . . . . . | |
| Ol. cloves . . . . . | āā ℥ 30. |
| Ol. cajuput . . . . . | |
| Ol. juniper . . . . . | āā ℥ 5. |
| Ac. sulph. aromat. . . . . | ℥ 15. |

℥i. in ℥ss. of water, every half hour until vomiting and purging cease.

The patient must, of course, be kept strictly at rest, and the rectal temperature carefully watched. If it shows signs of rising above 100°, hot water bottles should be removed. During the collapse stage barley water may be given in small quantities at frequent intervals.

## PLAGUE

In the Middle Ages this term was used to designate any severe or fatal epidemic, but as now understood its meaning is restricted to one particular disease, the bubonic, Oriental, or Levantine plague. This is an acute febrile disease, usually attended by swelling of the lymphatic glands in the groin or other part of the body, but sometimes fatal without such lesions. Its history can be traced back to the second century of the Christian era, but the first great epidemic in Europe, the plague of Justinian, occurred in the sixth century. Epidemics were frequent in the Middle Ages, but since the year 1665, when London was devastated by the plague, these epidemics have gradually become less frequent in Europe, and in the first third of the nineteenth century were confined on this continent to the most easterly portions of the Turkish empire. Recently the plague has again become of importance to European peoples, from its occurrence in Hong Kong and South-Eastern China in 1894, whence it spread to Bombay and other parts of India in 1896. From this source the disease has occasionally made its appearance, and for longer or shorter time, in different ports of Europe, South Africa, and Central and South America. In India for the fifteen years 1896–1910 it caused more than seven million deaths, an average of 466,000; in 1913 the number was 217,000, and in 1914, 227,000. Some cases occurred in England in 1910.

**Ætiology and Epidemiology.**—The plague is a specific disease, due to a bacillus (*B. pestis*) discovered by Kitasato and by Yersin, which may be found during life in the blood, in the inflamed glands, in the fæces and urine, and in the sputum of certain cases, and after death in almost every organ of the body. The bacillus is a short rod, with rounded ends, measuring from 1$\mu$ to 1·5$\mu$ in length, flagellated, Gram negative, and staining more deeply at the ends than in the centre.

Plague occurs in epizoötics among rats and other rodents and is a very fatal disease (**27**). Large numbers of dead rats have been observed shortly before a human epidemic. Plague is spread among rats by means of rat fleas, which usually confine themselves to their hosts, the rats, but after the death of the latter may invade other species, and so may attack man if he is in the neighbourhood. Thus the spread of the disease depends on the close association of rats with human beings. This occurs in India where the black or house rat, *Rattus rattus*, a tame friendly animal, is very common. In more civilised countries this rat has almost disappeared, its place being taken by the brown or sewer rat, *R. norvegicus*, a much more shy and timid beast, which lives in burrows and avoids human society. Thus in the plague epizoötic among rats, rabbits, and hares, in East Anglia in 1910, there was no real danger of an epidemic and only a few cases of plague in man were notified. When the rat flea sucks the blood of an infected host, its proventriculus gets blocked with a living culture of plague bacilli, and the blood cannot get through into its stomach, but remains in the proventriculus distending it. On biting the next host the blood from the proventriculus escapes from the flea's jaws into the wound and so spreads the infection. The larger part of a community of rats may be killed off by plague, but the remainder are immune, and this immunity is passed on to succeeding generations, which accounts for the fact that a plague epidemic dies down after a time. Rats are usually responsible for a plague epidemic, but other rodents such as ground-squirrels, shrews and mice have been occasionally implicated. The tarabagan, *Arctomys bobac*, caused the epidemic of pneumonic plague in Manchuria in the winter of 1910–11. Pneumonic plague may spread directly by means of exhaled droplets of sputum from man to man, but bubonic plague is spread by rodents' fleas; hence infected rats, or other rodents, are necessary in the first place, although, when once the epidemic has begun, men may themselves transport infected fleas and so spread the disease from village to village. Climatic condi-

tions have a marked effect on the dissemination of plague, owing to their influence on fleas, which live longer and multiply more rapidly in a damp, cool atmosphere than in a hot, dry one. It has recently been shown that there are two fleas which commonly infest rats in tropical countries—one of which, *Xenopsylla astia*, is incapable of transmitting plague, and this is found to be the common rat flea in cities, such as Madras, where plague is very uncommon, or almost unknown. As a rule, plague epidemics follow the grain trade. The disease attacks people of all ages up to fifty, after which year it is much less common. One attack confers a relative immunity from others.

**Morbid Anatomy.**—The enlarged lymphatic glands are found to be inflamed, red or violet in colour, soft or pulpy in consistence, and surrounded by connective tissue infiltrated with serum or blood. The internal glands in the same neighbourhood are involved, thus the pelvic glands with inguinal buboes or the mediastinal glands with buboes of the neck. The liver is congested, the spleen is large and dark, the kidneys are swollen, and numerous hæmorrhages may be found in all these organs, as well as in the mucous membranes and skeletal muscles, and under the serous membranes. The lungs show patches of consolidation in the pneumonic form, but in the other varieties only œdema and minute hæmorrhages; there is often some pleural effusion.

**Symptoms.**—Several varieties of plague are now recognised. The more common form is the *bubonic*, which is characterised by glandular enlargements. The *incubation* is from two to five days, and the disease begins with lassitude, weakness, headache, vertigo, and shivering, soon followed by febrile reaction. Sometimes in this stage of invasion the patient is in a peculiar absent condition, with staggering gait and tremulous speech; or he is seized with indefinable fear and restlessness; or there may be nausea, vomiting, or diarrhœa. The fever is generally high, the temperature from 102° to 104° F., or in the worst cases over 107°, the pulse from 100 to 130. The tongue, at first moist and white, becomes dry and brown, and a typhoid condition may supervene with delirium or coma, sordes on the lips and teeth, failing pulse and cold extremities. The urine is scanty, acid, of high colour, and it usually contains albumin, and suppression occurs in some fatal cases. After one, two or three days' fever the local signs show themselves in the formation of glandular swellings in the groins, axillæ, or neck. Mostly only one group is swelled, and generally it is the inguinal glands that are affected. The factor which determines which group of glands is affected appears to be the skin area which is bitten by the infected flea. Thus, if the infecting bite is on the arm, the bubo forms in the axillary glands of that side. The swelling may be as large as a hen's egg, or larger, is attended with severe pains, and if the patient survives may suppurate about the seventh day. About this time also boils or carbuncles may appear, but they are not very frequent; they occur on the lower extremities, the buttocks, or the back of the neck. In the severest cases petechiæ, or larger subcutaneous hæmorrhages, appear shortly before death, either distributed generally over the body, or more marked in the neighbourhood of the enlarged glands, and there may be bleeding from the nose, lungs, stomach or bowels. Death takes place mostly before the sixth day. In cases that recover, convalescence begins from the sixth to the tenth day, but may be much protracted by suppuration of the glands.

In *septicæmic plague* the patient is struck down with great rapidity, and may be dead in twenty-four hours. The lymphatic glands may be somewhat swollen, but no large buboes form. The pulse rapidly fails, hyperpyrexia may occur, and delirium and coma end the scene. This no doubt includes cases formerly described as *fulminant* (*Pestis siderans*).

*Pneumonic plague.*—This is an important variety, which was first recognised in Bombay in 1896. It begins like the bubonic form, but within a day or two respiratory symptoms become urgent: there are very rapid, shallow breathing, cough, expectoration of much sputum tinged or streaked with blood, sleepless-

ness, restlessness, early delirium, and death within three days. The physical signs of consolidation are not prominent, but râles and rhonchi are present. After death patches of pneumonia are found scattered through the lung, and buboes are usually absent. The bacillus is found in the pneumonic patches and in the sputum.

*Pestis minor* and *Pestis ambulans* are varieties in which the fever is slight and the symptoms are mild, so that sometimes the patient may walk about during a great part of the illness.

**Diagnosis.**—This is especially difficult with the first cases imported into a new district, and such early cases have been mistaken for yellow fever, typhoid, typhus, or malaria; and confusion with diphtheria, parotitis, and soft sore and "climatic bubo" has also occurred. The distinctive early symptoms are the expression of the face, the hesitating speech, and staggering gait, and later on the numerous buboes. But the ultimate diagnosis must depend on the detection of the bacillus, which may be found in the blood, in the juice or pus from buboes, or in the sputum. The contents of buboes may be removed with a sterilised syringe, placed on a slide or cover glass, gently heated to dry and fix, coloured with carbol fuchsin, or methylene blue, or Leishman's stain, and examined with a $\frac{1}{12}$-inch oil immersion lens

**Prognosis.**—In the septicæmic and pneumonic forms death is invariable. In bubonic plague the mortality varies from 40 to 80 per cent.

**Prevention.**—This comprises notification of the disease; the isolation of any patient suffering from the disease; the disinfection of dwelling-houses, clothes and bedding, preferably by solutions of perchloride of mercury; the disinfection of persons taken from infected houses; the destruction as far as possible of rats. In a country like India anti-plague measures should be carried out in the quiescent season in villages where an epizoötic is persisting, as these provide the starting point of a fresh epidemic when the weather becomes cooler. Steps must also be taken to prevent the transport of rats in ships from one port to another. Anti-plague inoculation has now been tried on a large scale with marked success. An injection of 4 c.c. of vaccine is given subcutaneously. Both the local and general reaction is frequently severe, but this should not deter residents in infected areas from being inoculated. In a series of over 50,000 inoculations performed in Bagdad during an epidemic, it has been shown that persons were attacked in the proportion of one inoculated to twenty-two uninoculated.

**Treatment.**—This is for the most part symptomatic, and pain, collapse, or hyperpyrexia must be dealt with, as in other acute specific diseases, by opium, stimulants, local application of cold, etc. Treatment with anti-plague serum has been tried, but no very striking results have so far been obtained.

## TULARÆMIA

In 1912 a plague-like disease prevalent among ground-squirrels and jack rabbits in California was found to be due to a new cocco-bacillus, which was called *Bacillus tularense*, being named after County Tulare, in which the investigations were made (**28**). Later work has proved that this disease may be transmitted to man in the following ways: (1) To farmers and others in the infected areas by means of a bite from a blood-sucking fly (*Chrysops*). (2) To market-porters and cooks engaged in dressing rabbits by direct infection through cuts on fingers or through the conjunctiva. (3) To laboratory workers investigating the disease. The last has occurred frequently, and in certain cases has followed the cough of an infected animal into the face of the laboratory worker during anæsthetisation. The disease is evidently extremely infectious.

**Symptoms.**—The primary lesion is seen three days after infection and

depends on the site of inoculation. Thus it may take the form of a unilateral ulcerative conjunctivitis, or of a black necrosing mass on the skin which suppurates, leaving a punched-out ulcer. No lesion is seen where infection has occurred through inhalation. The glands in the neighbourhood of the primary lesions enlarge and may suppurate. In all cases the systemic reaction is considerable. The disease begins with fever, malaise, and pains in head and body. The fever may remit after three days, but then slowly rises again, resembles a septicæmia in being of swinging type, and may last for three weeks or more. Prostration is extreme and convalescence may not be complete for months, since milder relapses are apt to occur. Death has taken place from pneumonia.

**Diagnosis.**—So far the disease has only been described among laboratory workers in this country (**28**), and the absence of localising signs made the cases resemble tuberculosis or enteric fever. Among the general population the primary lesion and the nature of the occupation should provide a clue. The only certain method of diagnosis is to inoculate a guinea-pig and observe the characteristic lesions, and to obtain agglutination of killed and preserved cultures of the bacillus with high dilutions of the patient's serum. The disease does not occur in tame rabbits.

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# DISEASES OF THE ORGANS OF RESPIRATION

## EXAMINATION OF THE CHEST

SINCE the lungs are contained almost entirely within the bony thorax, or chest, the diseases of these organs are likely to reveal themselves by modifications in the shape, in the movements, and in the acoustic phenomena yielded by the chest. A consideration of these various physical signs or indications of lung diseases must precede their systematic description.

For purposes of accurate description it is necessary to be able to localise any point on the chest wall. This is usually done by observing in what intercostal space or over what rib the point lies and at the same time measuring its horizontal distance from certain vertical lines, such as the middle line of the sternum, the lateral sternal line, the mid-clavicular line which usually runs through the nipple, the anterior and posterior axillary lines, the line corresponding to the vertebral border of the scapula, and the middle line of the back corresponding to the spines of the vertebræ. The ribs are best counted from the prominent ridge between the manubrium and gladiolus (*angulus Ludovici*), which corresponds to the second rib, and from the twelfth rib behind, which can be readily identified in most persons.

The modes of examination of the chest are inspection, including the use of Röntgen rays, palpation, percussion, and auscultation.

### INSPECTION

By looking at the chest in front, behind, and from above, abnormalities such as various skin eruptions, scars from old empyema operations or gunshot wounds, and enlarged veins may be noticed, and any alteration in its shape and movements can be detected. As regards enlargement of the veins, it is important to distinguish between those that are distended, which may suggest obstruction to the venæ cavæ internally, and those that happen to be more superficial than usual and are of no pathological significance. The chief points to be noticed in a healthy adult chest are as follows: It has a somewhat flat oval form—that is, the antero-posterior diameter is much less than the transverse; its greater breadth is at the lower part; the clavicles are only slightly prominent, with but little depression above, and scarcely any below them; the position of the nipple is on the fourth rib, or on its upper or lower border; the angle (*epigastric angle*), which has its apex at the ensiform cartilage, and is bounded on each side by the seventh and eighth costal cartilages, is from 95 to 105 degrees; the scapula is closely adapted to the posterior part of the thorax; and the spine is straight. In inspiration the chest should expand from 2 to 3 inches in circumference, the two sides should move symmetrically, the epigastric angle should be widened, the sternum thrown forwards, and the lower ribs lifted; and there should be only very slight recession of the lowest intercostal spaces on deep breathing.

By inspection abnormalities in the shape of the chest are noted, and these may be due to the following causes: (*a*) Disease of the *lungs*. In emphysema the chest is more capacious and more circular in shape than normal. In fibrosis of

the lung, as in phthisis, the chest is asymmetrical, owing to local contraction, usually at one or other apex. (*b*) Disease of the *bones*, such as deformities due to rickets and angular and lateral curvatures of the spine. The chest may become asymmetrical, or still show bilateral symmetry although abnormal in shape. Bronchitis and broncho-pneumonia have usually played a part in producing the deformities due to rickets. (*c*) Hypertrophy of the *heart* in young subjects may lead to a local bulging of the chest wall on the left side.

The circumference of the chest is obtained by a tape measure, the transverse and antero-posterior diameters by *callipers*. The actual shape can be obtained by a *cyrtometer*, consisting of two long pieces of soft metal, joined loosely together by one end of each. The point of junction is applied to the spine, and the metal rod on either side is wrapped round the side of the chest at any desired level, so as to take a mould of its shape or curve. The instrument is then carefully removed, without disturbing the moulded curve, and if it is laid out on a large sheet of paper in the position it occupied while applied to the chest, a pencil can be traced round it, and a permanent record of the shape of the chest is thus obtained.

By inspection also, apart from changes in the shape and symmetry of the thorax, we may note the character of the respiratory movements. The normal frequency of respiration in adults is from about fifteen to eighteen in the minute; in children it is much more rapid; it may be thirty or more and varies with the age. In

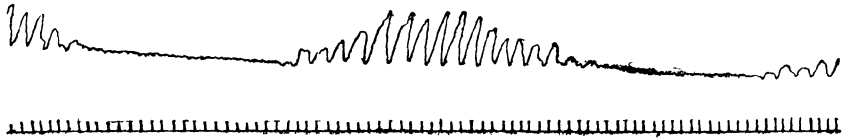


FIG. 18.—Cheyne-Stokes Respiration. The curve reads from left to right, and the time is marked below in seconds. The small undulations during the period of apnœa are due to the beats of the heart.

different forms of disease, both pulmonary and of other origin, the movements may be slower or more rapid, shallower or deeper, feebler or stronger than is normal, and they may be irregular. *Dyspnœa* is a clinical term used to indicate "shortness of breath," whether this is due to one or more of the following: (*a*) increase in the respiratory rate (*polypnœa*); (*b*) increase in the respiratory volume (*hyperpnœa*); (*c*) obstruction to the flow of air into and out of the lungs (*obstructive dyspnœa*). In the last case the difficulty may be most marked during inspiration (*inspiratory dyspnœa*), or during expiration (*expiratory dyspnœa*). If the patient is obliged to sit up in order to breathe with greater comfort, as is the case in many pulmonary and cardiac diseases, the condition is called *orthopnœa*.

It is important also to note whether the breathing is effected more by the upper part of the chest, which is usual in women, or by the lower part, which is more characteristic of men. The inspection must be extended to the abdominal walls, which reflect, so to say, the action of the diaphragm, advancing when it contracts and receding as it relaxes. A disproportionate use of one part of the chest suggests disease in another part. The inspection should be made both during quiet breathing and when the patient makes a forced inspiration.

**Ectoscopy.**—This is a term used to describe a new method of marking the borders of the lower parts of the lungs on the surface of the chest (**1**). If the patient takes a sudden inspiratory sniff with the mouth closed, the contraction of the diaphragm will cause indentations in the costal spaces round the lower part of the chest, because the edge of the lung does not expand sufficiently quickly. If the patient suddenly calls out the word "Kit," the rise of the

diaphragm causes a local expansion in the spaces for a similar reason. The position of maximum disturbance is marked in each space, and the marks are then joined by a line which indicates the border of the lung. It is important to adjust the illumination correctly. There should be a bright light on one side of the room and the walls of the latter should be dark. The patient stands erect with the arms raised. The phenomenon can usually be seen a little better on the right than the left side and is difficult to see with fat people. The method is of value in determining the extent of the lung over the heart, and in the region of the stomach where percussion does not help. The phenomenon is often absent in acute pleurisy or with thickened pleura and retracted lung, but with pleural effusion the phonation movement is almost as large as with healthy lung, and in cases where there is some doubt where to explore for fluid, the position of the maximum expiration movement should be chosen.

**Cheyne-Stokes Respiration.**—This form of breathing is characterised by alternating periods of increased respiration (*hyperpnœa*) and absence of respiratory movements (*apnœa*). The hyperpnœic periods show gradual waxing and waning of the respiratory volumes, as shown in Fig. 18. The whole duration of a cycle may be from twenty to sixty seconds, and the number of respirations in a cycle varies from five to sixty. During the middle of the hyperpnœic period the respiration rate may be as rapid as fifty or sixty a minute. Cheyne-Stokes respiration is probably associated with changes in the excitability of the respiratory centre and generally indicates lack of oxygen. It is, however, possible that changes in the calibre of the cerebral arteries are responsible, periodic contraction of the arteries of the medulla causing periodic apnœa. (**2**). It is occasionally present in normal people during sleep, and it occurs very commonly at high altitudes. It often precedes death by a few hours in a large number of pathological conditions. On the other hand, it has been known to persist for months in elderly persons suffering from myocardial degeneration and arterial disease. The effect of the hyperpnœic period is to wash out $CO_2$ from the blood, so that the respiratory centre is no longer stimulated and the breathing stops. During the pause the $CO_2$ gradually accumulates, while the oxygen in the alveoli is used up. Oxygen want suddenly supervenes, and the breathing starts again. During the hyperpnœa the oxygen in the lungs rises rapidly ; but the respiratory centre overshoots the mark, leading to a washing out of $CO_2$ while the oxygen want has disappeared for the time being. Thus Cheyne-Stokes respiration depends on there being two independent factors that stimulate the respiratory centre : oxygen want and $CO_2$ (Pembrey and Allen). It may be likened to the "hunting of the governor" of an engine in the absence of the flywheel. Other phenomena may accompany the changes in the breathing. Towards the end of apnœa, in the period of oxygen want, the patient becomes drowsy and apathetic and his face livid, while he may become excited in the latter part of the hyperpnœic period. The pupils may dilate in hyperpnœa, and contract again in apnœa. The pulse is often scarcely affected, but Sir F. Taylor observed it cease entirely for thirty seconds in the early and middle periods of hyperpnœa.

In *Biot's respiration*, seen most commonly in meningitis, there are pauses of several seconds, up to thirty or more, occurring more or less periodically ; but there is no waxing or waning of the respirations.

**Vital Capacity.**—As a result of measurements by Dreyer, Ainley Walker, and others, the vital capacity has recently been used as a test for physical fitness. In healthy individuals it increases with the body weight, but is rather larger for those who are accustomed to violent muscular exertion than for those who follow sedentary occupations. The body weight of the patient is taken without clothes, and if the vital capacity observed is less than the corresponding value in the tables, it suggests physical unfitness on the part of the patient. In the case of wasted or over-fat persons such a method would obviously lead to fallacious results, so that the length of the trunk and the circumference of the chest in the nipple line

are measured, and then from further tables the weight corresponding to these values is obtained, with which the vital capacity is compared. The vital capacity is roughly proportional to the body surface.

### RÖNTGEN RAYS

This method of investigation is of great value in recognising the fact of disease in the chest, or in estimating its extent and position: the position and movements of the diaphragm, the presence of pulmonary consolidation, tubercle, new growths, and liquid effusions may be recognised by shadows visible on the screen; and of these photographs may be taken. It is usually best to examine the patient in the vertical rather than in the horizontal position.

### PALPATION

By this is meant the act of laying the hand upon the surface of the chest, either to test its movements or to study the vibrations of its walls produced by the voice or other cause. For the former purpose a hand is laid at the same time on each side below the clavicle, or in the infra-scapular or infra-axillary region, when the absolute and relative amounts of movement can be gauged with some accuracy. For the latter purpose the hand is placed flat upon the chest, and the patient speaks in a loud voice. It is best to use both hands simultaneously placed one on each side of the chest in symmetrical positions; the dorsal surfaces may be used as well as the palmar (Jex-Blake). In health the chest wall is thrown into vibrations which are plainly perceptible to the hand laid upon it (*tactile vocal fremitus* or *tactile vibration*). For this it is necessary that there shall be a normal vibration of the vocal cords, and normal conductivity of the lungs with patent bronchial tubes and spongy lung tissue. The amount of vibration differs in healthy people; it is greatest in adult males with deep sonorous voices; it is least, or it may be absent, in females and children. In disease it is diminished or abolished by anything which obstructs the bronchial tubes or compresses the lungs, so as to convert its spongy tissue into solid, *i.e.* liquid in the pleural cavity and pneumothorax. It is increased when there is consolidation of the lung tissue with patency of the bronchial tubes. In pneumonia it is diminished or absent when the smaller tubes are filled with secretion, but increased if they have been cleared out by coughing.

By palpation also can be recognised the vibrations of pleural friction, of bronchial narrowing (rhonchi), and of some sounds produced in cavities. The corresponding sounds are described under Auscultation.

### PERCUSSION

A sound can be produced from any part of the body by percussion. The thigh produces an *absolutely dull* sound—a mere noise—which possesses only two properties, *loudness* and *duration*. When the chest or abdomen are struck, both of them air-containing cavities, the sound is partly a noise and partly a musical tone; in proportion as the latter is present the structure is said to have *resonance*. The latter depends on there being (1) a cavity in which air can vibrate, (2) walls which are sufficiently elastic, and of the right tension to vibrate in unison with the air, and also capable of conducting the sound externally. The effect that tension of the wall has on resonance can be easily demonstrated by flicking the cheek distended with air and varying the contraction of the cheek muscles. A tone is produced by a regular series of vibrations; its *pitch* depends on the number of vibrations per second; its *quality* depends on the number of harmonics or overtones present with the fundamental note. Like a mere noise, a tone also possesses loudness and duration; but for the same strength of

percussion the loudness and duration are greater in the case of a tone than a noise.

The most musical percussion sounds are called *tympanitic*. They are obtained from the abdomen, and from a chest containing a pneumothorax; the pitch is low in these cases, because the cavities are large. The trachea also gives a tympanitic note, but of higher pitch. Percussion of the normal chest produces *normal lung resonance* where the musical element is less pronounced than in *tympany*, because the elastic walls of the alveoli do not vibrate in unison with the chest wall. The pitch is low, being 70 to 120 vibrations per second (Müller). If the alveolar walls are relaxed, supposing fluid collects in the pleural cavity, the note becomes tympanitic. This is called *Skodaic resonance*. If fluid is present in larger amount, so as to compress the lung and drive the air out of it, the sound will be completely dull; but beyond the margin of the dull area there may be a tympanitic sound, where the lung is only relaxed, and beyond this, again, there will be the normal lung resonance. Fluid itself is a good conductor of sound, as any one may demonstrate to himself by lying in a bath with the ears submerged and allowing the tap to run. The resonance is lost because (1) when there is much fluid present the lung is compressed; (2) even if the lung still contains some air there is faulty transmission of the sound to the fluid (*see* p. 193).

*Hyper-resonance* connotes the stage between normal lung resonance and tympany, *impairment of resonance* that between normal lung resonance and absolute dullness. A hyper-resonant or tympanic note is obtained in emphysema, where the alveolar walls are largely destroyed, and over very large cavities, as in phthisis. Resonance is impaired by solidification or compression of the lung, and by thickening of the pleura. A dull percussion note is obtained over small cavities in the lung, because of the coincident fibrosis. The lung resonance also depends on the thickness of the chest wall. A duller note is obtained with muscularly developed than with spare individuals, and over the back than over the front of the chest.

*Immediate* percussion is practised over the clavicles by percussing with the finger directly. *Mediate* percussion is used for the rest of the chest, the second finger of the left hand being laid along one of the intercostal spaces, and being struck on the dorsal surface of the distal interphalangeal joint by the tip of the second finger of the right hand. It is important to compare corresponding points on the two sides of the chest together. By using the fingers in this way it is also possible to get an idea of *resistance*. When a resonant note is obtained a feeling of resilience is experienced by means of the finger lying on the chest; when the note is dull the chest wall feels unyielding and inert.

The normal lung resonance extends on the right side of the chest from about an inch above the clavicle to the upper portion of the sixth rib; over the whole of the sternum; on the left side from above the clavicle to the upper border of the fourth rib internal to the nipple, and outside the nipple down to the sixth rib, where it passes into the resonance of the stomach. In the right lateral region it extends from the axilla to a horizontal line cutting the eighth rib in the mid-axillary line; on the left side the axillary resonance tends to be tympanitic, owing to the propinquity of the stomach. Posteriorly the chest is resonant from the apices to the lower border of the eleventh rib on the left side, and to its upper border on the right side. The resonance extends a finger's breadth lower than these limits on deep inspiration. The length and loudness of the note are most marked in the second intercostal space in front and over the infra-scapular regions behind. Over the clavicle and sternum it is less full, and of higher pitch; and over the supra-spinous fossæ the note is often deficient, especially in very muscular or fat people. At its lower margin the pulmonary resonance is less marked, and approximates to the dullness of the parts below; it is called *transitional* dullness.

Two special kinds of percussion sounds must be mentioned here; (1) the *cracked pot* sound sometimes heard over a cavity connected with a bronchus (*see* p. 253); (2) *amphoric resonance*, or the *metallic ring* occasionally heard on percussion over a large air-containing cavity, very similar to the *bruit d'airain* (*see* pp. 196, 280). Percussion was first described by Auenbrugger in 1761 (**3**), and he also described the various pathological conditions associated with alterations in the note.

## AUSCULTATION

This is the study of the viscera or other parts of the body by listening to the sounds that are produced within them. It may be *immediate*, when the ear itself is applied to the chest, either bare or with only a towel or handkerchief intervening, or *mediate*, when a sound-conducting instrument connects the chest of the patient and the ear of the listener. The instruments more commonly employed are (1) the *binaural stethoscope*, (2) the straight wooden or metal stethoscope, about 7 inches long, and (3) the *phonendoscope*, in which the sounds are resonated. The first and third have the advantage of flexibility, and can be used in all positions of the patient. Auscultation and its medical bearings were first described by Laennec, who gathered his observations into two volumes in 1819, entitled, "De l'Auscultation Médiate ou Traité du Diagnostic des Maladies des Poumons et du Cœur fondé principalement sur ce nouveau moyen d'exploration," published in Paris. His discovery of mediate auscultation is thus told in his own words (**3**):

"In 1816 I was consulted by a young woman presenting general symptoms of disease of the heart . . . The patient's age and sex did not permit me to resort to the kind of examination I have just described (*i.e.* direct application of the ear to the chest). I recalled a well-known acoustic phenomenon—namely, if you place your ear against one end of a wooden beam the scratch of a pin at the other extremity is most distinctly audible . . . Taking a sheaf of paper I rolled it into a very tight roll, one end of which I placed over the præcardial region, whilst I put may ear to the other. I was both surprised and gratified at being able to hear the beating of the heart with a much greater clearness and distinctness than I had ever done before by the direct application of my ear."

By auscultation of the lungs we determine the character of the breath sounds and of the vocal resonance and the presence of adventitious sounds.

**Auscultation of the Breath Sounds.**—If the healthy lung is auscultated, one hears everywhere, with each respiration, a sound which is known as the *normal breath sound*, or *vesicular murmur*. It may be imitated by blowing softly, with the lips placed in the position to pronounce the German "w" or English soft "v." It is of low pitch, with vibrations of from 70 to 90 per second (Müller). The vesicular murmur is heard during inspiration; but the expiratory act is either quite silent, or is accompanied by a similar sound, much softer and shorter, being limited to the earlier part of expiration.

The production of breath sounds is explained on p. 288 to be due to the formation of a "fluid vein," when the air passes from a narrow opening into a wider space. During *inspiration* the breath sound is formed (1) at the glottis, and (2) at the periphery, as the air passes from the respiratory bronchioles through the alveolar ducts and atria into the air-sacs (*see* Fig. 19). Sound emanating from a source in a uniform medium becomes generally diffused all round, so that its intensity diminishes as the square of the distance; but when formed at the glottis, which is situated in a tube, its diffusion is prevented, and it is conducted down along the tube, by continued reflection from the inside of its walls, as happens in a speaking-tube. However, some diffusion occurs from the smaller tubes into the surrounding lung so that the glottic sound heard on the outside of the chest is weak, though it is reinforced by the sound produced peripherally in the air sacs, etc. The same principle of the speaking-tube explains the use of the

stethoscope in auscultation. During *expiration* the sound is still weaker, because it is due solely to the glottis, and because there is another general rule, viz., that sounds are less readily conducted against the stream of air than with it. In certain parts of the chest, where there is not such a thick layer of spongy tissue between the large bronchi and the chest wall, the vesicular murmur gives place to rather harsher sounds, which continue during the greater part of expiration, and are called *broncho-vesicular breathing*. These parts are the upper end of the sternum, the first costal cartilages at their junction with the sternum, and a diamond-shaped space at the back in the middle line, including the seventh cervical and first dorsal spines. Elsewhere the vesicular murmur is always present as long as the lung is healthy and the air passages are pervious. In children the vesicular murmur is louder and harsher than in adults (puerile breathing). In adults the vesicular murmur over a healthy lung may be harsh and exaggerated when the other one is put out of action (*compensatory breathing*). The breath sounds may be weak in shallow breathing, in emphysema, and when the chest wall is thick. *Interrupted* or *cog-wheel breathing* is where the inspiratory murmur is jerky or wavy, from irregular expansion of the lung, of which the cause may be mechanical obstruction to the entry of air or irregular muscular action from nervousness. It is not of much diagnostic significance.

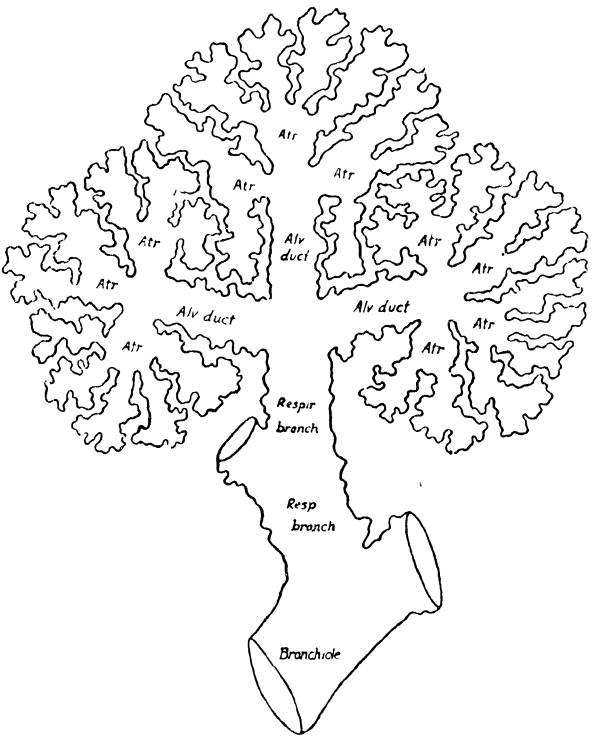



FIG. 19.—Structure of Lung after Miller (**30**). The respiratory bronchioles, alveolar ducts, atria, or terminal air-sacs, all have alveoli lining their walls, through which the exchange of gases takes place

*Diminished vesicular murmur*, deficient entry of air, or absence of breath-sounds, occurs if the air vesicles are obliterated or if the bronchus communicating with them is obstructed or obliterated, or if the air vesicles are displaced from the surface of the chest as is commonly the case in pleural effusion. In the latter case the absence of breath sounds depends on the principle that when sound travelling in one medium, such as air, reaches the boundary surface of another medium, such as a pleural effusion, it is badly transmitted across it, most of it being reflected back again. This is a sign of great diagnostic importance when the breath sounds are normal at the corresponding point of the opposite lung.

*Bronchial breathing* is a double sound produced in the glottis during respiration, modified by resonance in the mouth, pharynx and bronchial tubes. It can be heard by listening over the trachea. The inspiratory and expiratory sounds are

of equal lengths; they are distinctly separate from one another, and are harsh in quality. They may be imitated by placing the mouth and tongue in the position to pronounce the German "ch," and then blowing in and out. As heard over the lung bronchial breathing may be high-pitched (*tubular*), medium-pitched or low-pitched (*cavernous*). The loudness of the sounds is unimportant. Tubular breathing which is heard particularly over hepatised lung has a special blowing or "whiffing" quality. Cavernous breathing has a hollow quality. It is often heard over cavities in the lung, but it does not always indicate that there is a cavity. Bronchial breathing when heard over the chest means that the lung tissue has been modified, mostly by a conversion of the spongy tissue into solid tissue, either by filling up or destruction of the air cells (pneumonia, phthisis), or sometimes by compression from without (pleuritic effusion), if the air passages are not entirely obliterated. The necessary condition is patency of the bronchial tubes with consolidation of the surrounding lung, so that the glottic sound is conducted to the surface, instead of becoming diffused, while the alveolar part of the ordinary respiratory murmur is absent.

*Amphoric breathing.*—This is a double sound heard during inspiration and expiration, more musical than bronchial breathing. It may have a peculiar metallic or ringing character. It may be imitated by blowing softly into the mouth of a narrow-necked glass jar or vase. It is heard over large cavities or a pneumothorax in open communication with a bronchus, and is due to the resonance of the cavity. The pitch and loudness of amphoric breathing are variable.

*Stridor* is a loud sound rather similar to tubular breathing, but it has a characteristic whistling or hissing quality. It is produced by constriction of the glottis, trachea, or one main bronchus. It is audible over the greater part of the chest, and can sometimes be heard by those near the patient without the aid of the stethoscope.

**Adventitious Sounds.**—The word *adventitious* expresses the fact that these sounds are heard in addition to, and at the same time as, the ordinary breath sounds or the breath sounds modified as above. If they are not heard with tranquil breathing, the patient should inspire deeply, when they may become audible. The adventitious sounds are *rhonchi*, *râles*, and *friction sounds*.

*Rhonchi* are more or less musical sounds, due to obstruction of the bronchial tubes by accumulation of mucus, swelling of the mucous membrane, or spasmodic contraction of their muscular fibres. The sounds vary very much according to the size of the bronchial tube and the extent of the narrowing, and are likened to various familiar sounds, such as cooing, groaning, snoring, grunting, or whistling. The lower-pitched, snoring sounds are called *sonorous* rhonchi, and are produced in the larger tubes; the higher-pitched, whistling sounds are called *sibilant* rhonchi, and are produced in the smaller tubes. They may be heard with expiration or inspiration, and are constantly changing in position and loudness. Loud sonorous rhonchi are often audible to those standing near the patient, and constitute "wheezing."

*Râles* are various forms of crackling or rattling sounds, which are produced in the large, medium-sized and smaller bronchial tubes, or in pulmonary cavities by the air forcing its way into fluid secretions accumulated there, and thus causing bubbles to form and burst with a slight noise. They are sometimes distinguished as *moist* sounds from rhonchi or *dry* sounds; but this is undesirable, if rhonchi may themselves be due to the presence of mucus. The râles differ according to the size of the bubbles, and are called *small*, *medium*, and *large*. Râles are also divided into *bubbling* and *crackling*: the latter have a sharp, clear, ringing, explosive character, which is probably due to their occurrence in the midst of consolidated lung, and to consequent special conditions of resonance; the former, or bubbling râles, are dull, not ringing or explosive, and occur mostly in tubes surrounded by normal spongy tissue. Crackling râles are sometimes called *consonating* from their supposed acoustic origin, and bubbling râles *non-*

*consonating* in contrast. A *gurgling* râle is a coarse râle which is produced in the largest tubes.

*Crepitation* is a term which has been used indiscriminately for all râles, but is now generally confined to a very fine râle, so fine as to be suggestive of an origin in dry materials (rubbing of hair close to the ear, or rustling of silk). It is heard in the early stage of pneumonia, in œdema of the lung, and in lung that is forcibly expanded after prolonged collapse. It is probably due to the opening up of respiratory bronchioles, atria and air-sacs, which have been adherent by sticky fluid or from simple disuse. Crepitations and the finer râles are heard only during inspiration; medium-sized and coarser râles may be heard during expiration also.

*Metallic tinkling* is applied to sounds of a tinkling or ringing character, sometimes heard when a patient with a large cavity breathes, or speaks, or coughs. It is usually a musical râle.

*Post-tussive suction* is a sucking sound heard over a cavity (tuberculous or bronchiectatic) following a cough; it is due to the air being suddenly sucked back into the cavity with a hissing sound during the succeeding deep inspiration.

In cases of hydro-pneumothorax or pyo-pneumothorax, if the patient be shaken while the physician's ear is applied to the chest, a *splashing* sound will be heard which proceeds from the air and liquid in the pleural cavity (*Hippocratic succussion*).

*Friction sound*, or *pleuritic rub*, is produced by the rubbing together of two pleural surfaces roughened by inflammation. In its most characteristic form it is a rough, grating, or creaking sound, like that which may be heard on forcibly dragging two pieces of leather over one another, or on rubbing the palmar surface of a finger over a wooden surface. It is best heard during inspiration, but may be heard with expiration also. Some friction sounds resemble râles very closely; but they are localised to a small part of the chest, and are not influenced by coughing. Their loudness varies, when the pressure of the stethoscope against the chest wall is altered. When they arise in the pleura overlying the heart, they correspond with the heart beats, but their loudness varies with the phases of respiration (*pleuro-pericardial friction*).

**Auscultation of the Voice.**—In most people the voice is transmitted through the chest, and can be heard by the ear or stethoscope placed on any part of it; this is called *vocal resonance*. The sounds heard over the normal chest should be compared with those heard over the trachea. In the former situation (1) they are not so loud; (2) they are "muffled," owing to deficient conduction through the alveolar tissue.

*Diminished or absent vocal resonance.*—In children and females with voices of high pitch, the vocal resonance may be slight or absent. In disease its absence is produced by obstruction of the bronchus, or compression of the lung, involving the bronchus.

*Increased vocal resonance. Bronchophony.*—There is naturally a louder vocal resonance at those points where broncho-vesicular breathing is normally heard—namely, the sterno-clavicular articulation and the inter-scapular region. In disease it is caused by consolidation of the lung around bronchial tubes, such as occurs in pneumonia, tuberculous consolidation, and sometimes compression by liquid.

*Pectoriloquy* is the clear transmission of articulate sounds, as distinguished from mere loudness of transmission of the vocal vibrations in bronchophony. It may be recognised when the patient speaks loudly, but it is best observed by asking the patient to whisper, when the laryngeal vibrations are absent. It is observed over pneumonia, and over consolidated fibroid lung.

*Ægophony* is a peculiar *nasal* or *twanging* modification of the voice when heard through the chest. It derives its name from its supposed resemblance to the bleating of a goat. Its most common cause is undoubtedly the presence in

the pleura of a liquid. Ægophony is commonly heard at the middle of the back behind, internal to, or below, or over the lower end of the scapula, particularly when a patient with a pleural effusion has been lying on his back, so that there is a thin but fairly extensive layer of fluid compressing the lung. It is occasionally heard in front. Sometimes it may occur in pneumonic consolidation. The explanation of ægophony, most generally accepted, is that the spoken voice is not a pure tone, but a mixture of a fundamental note and its harmonics of higher pitch. It is well known that low notes are not so well transmitted as high notes from air to liquid, and so it is considered likely that the fundamental note fails to get through the liquid and the harmonics are chiefly heard on the surface of the chest. It is best brought out by asking the patient to utter words containing the vowels "e" and "i," such as "three," or "ninety-nine," which depend on the presence of the higher harmonics.

**Auscultation of the Cough.**—The patient is directed to cough while the physician auscultates the chest. Increased resonance of the cough occurs under the same conditions as increased resonance of the voice (consolidated lung and cavity); moreover, the cough, and the forced inspiration following it, will reveal the existence of râles that are not obvious on ordinary inspiration. In infants the spontaneous cough supplies the information as to vocal resonance, which is given in adults by speaking.

### AUSCULTATORY PERCUSSION

In this process a stethoscope is placed on the chest, and the surface is percussed around it; its chief value is to elicit the *bell sound* or *bruit d'airain* in cases of pneumothorax. While the physician listens with the stethoscope to one part of the chest, presumed to be the subject of pneumothorax, an assistant lays one coin on the chest, and strikes it with another. The noise is resonated in the hollow cavity, and transmitted as a loud ringing musical note through the stethoscope (*see* also p. 230).

# DISEASES OF THE NASAL PASSAGES

## ACUTE RHINITIS

(*Acute Nasal Catarrh, Coryza*)

This disease, familiarly known as a "cold in the head," is a catarrhal inflammation of the mucous membrane of the nose, which often involves also the conjunctivæ, frontal sinuses, pharynx, and Eustachian tubes, and may spread to the larynx, trachea, and bronchi. It is primarily an infection, which is spread by the inhalation of a spray of saliva from an infected person, emitted during coughing, sneezing, and talking. Sometimes at the end of a period of immunity acquired from the last attack patients may reinfect themselves. The cause of a common cold is a minute "filter-passing" coccoid body, 0·2 to 0·3 micron in diameter, arranged singly, in pairs and in groups, and resembling the virus of poliomyelitis (**4**). In quite the early stages very few ordinary bacteria are present in the nasal secretion, but later on pneumococci, *Micrococcus catarrhalis*, *Bacillus septus* (*B. coryzæ segmentosus* of Cautley), and *B. influenzæ* are commonly found, and in more chronic cases there are present, in addition, hæmolytic and non-hæmolytic streptococci, Friedländer's bacillus and staphylococci; but many other organisms have been found in different cases. The chief predisposing cause is congestion and swelling of the nasal mucous membrane. In general this is brought about by a moist atmosphere with the temperature variable, but on the whole cool. It is

this latter fact that has given rise to the phrase "catching' cold." It is under such conditions that epidemics occur. Again, the conditions indoors may be responsible ; people often sit in a warm stuffy room where the air is stagnant, but where the floor is made cold by draughts. The head is warm, and the feet are chilled. Physiologically the head and nasal mucous membrane should be cool and in fresh moving air, and the feet should be warm (L. Hill).

An attack of sneezing or a raw or rough feeling in the throat may be one of the first symptoms ; but these may be preceded by a feeling of indisposition, with chilliness, headache, and loss of appetite. The sneezing is soon followed by the discharge of clear mucus from the nose ; and there is a feeling of stuffiness in the nose, due to swelling of the mucous membrane. At the same time, the eyes are suffused and water freely, there is pain over the eyebrow from implication of the frontal sinus, the throat is sore, taste and smell are impaired, and there may be deafness from closure of the Eustachian tube. Some febrile reaction is present at the same time. If the catarrh extends to the larynx, the voice is hoarse, and there is constant irritating cough ; and its further spread to the lungs will cause the symptoms described below under Bronchitis. Often after one or two days the acute stage subsides, and recovery is complete. On the other hand, the discharge may continue, becoming thicker and more opaque from the presence of pus with the mucus. It may continue thus for a variable period, from two or three days to two or three weeks. During this time the patient is liable to fresh exacerbations of the inflammation.

Such is the typical picture of a coryza ; but in other cases the infection may begin as an acute laryngitis or bronchitis and subsequently spread upwards to the throat and nose. The clinical picture in different people is variable ; but in the individual person the course of the infection tends to be pretty constant. Thus, in one person a coryza always begins as a pharyngitis, in another as a laryngitis, in another as a bronchitis, and so on. People are thus very prone to suffer from repeated infections which chiefly attack one particular part of the respiratory tract. After a time recovery may be longer and longer delayed, and a chronic infection may result, taking the form of chronic nasal catarrh, chronic catarrhal laryngitis, chronic bronchitis, etc., depending on the part attacked in the first place.

It must be remembered that acute rhinitis occurs as a specific lesion in some of the infective diseases, such as influenza, measles, diphtheria, congenital syphilis, glanders, and others.

**Prevention.**—A hygienic method of life with plenty of outdoor exercise and a daily cold bath for robust persons, the avoidance of stuffy rooms, and especially of infected persons, are most important measures. A coryza may sometimes be averted if the throat is painted with 6 per cent. silver nitrate once at the earliest stage of the attack. Oil of cinnamon by the mouth has rather an undeserved reputation for stopping the complaint. Free sweating at night by means of a hot bath, or diaphoretic drugs, often seems to check the disease. Opium in small doses (Tinct. Camph. Co. ℥v.–x.) taken repeatedly may also avert the attack ; but personal experience has shown that a treatment devised by W. Glegg for chronic nasal catarrh is of the greatest value. One or two teaspoonfuls of a mixture of white vaseline and paraffin (1 in 3 or 4) is allowed to run from a funnel down each nostril in turn, while the patient lies on his back. When the mixture is felt in the throat it may be swallowed, or expectorated if it causes looseness of the bowels. This treatment can be carried out once or twice a day, or more often if a cold is threatening.

In many cases nasal and bronchial catarrh, etc., have been prevented by means of vaccines prepared from bacilli and cocci present in the respiratory passages. Stock vaccines prepared by the various commercial houses may be used or autogenous vaccines prepared from cultures of the patient's own organisms in the sputum or obtained by means of sterile swabs from the back of the nose

and throat. In preparing the vaccines it is important to remember that cultivation and incubation should be carried out *immediately* the material has been obtained. It seems at first sight surprising that vaccines prepared from bacteria should be useful, when the infection is due to a filter-passing organism. It is possible that the vaccine acts like a foreign protein in raising the immunity, and on this supposition a stock vaccine should be just as effective as an autogenous vaccine.

**Treatment.**—Personal experience has shown that Glegg's treatment may abort the nasal coryza, even when it is well developed; but it will have no effect on infection which has spread to the larynx and trachea. If the cough be troublesome, a few drops of ipecacuanha wine, with spirits of nitrous ether, or compound tincture of camphor, will relieve. Local remedies sometimes bring comfort, for instance, a mixture of bismuth subnit. ℥vj, morph. hydrochl. 2 gr., pulv. acaciæ ℥ij, 2 or 3 drachms to be snuffed up in small quantities, in the course of a day (Ferrier), or menthol 1 part, ammon. chlor. 3 parts, acid. boric. 2 parts; or the fluid extract of hamamelis may be snuffed up from the hand (Osler); or steam may be inhaled, impregnated with eucalyptus oil by placing five or six drops of the oil in boiling water in a suitable inhaler; or the nose may be irrigated with a solution of tincture of belladonna, ℥j in water ℥j; or a solution of adrenalin chloride (1 in 5,000) may be sprayed into the nostrils.

## CHRONIC RHINITIS

This is seen in two forms. In the one, *chronic hypertrophic rhinitis*, the mucous membrane of the nose and of the lower turbinated processes is greatly thickened, and this thickening may extend to the pharynx and involve the orifices of the Eustachian tubes. It is, at any rate, in most cases, a vasomotor rather than an inflammatory swelling. It may result from sinus disease. The breathing is much obstructed, and takes place chiefly by the mouth; and the sense of smell is impaired.

*Chronic atrophic rhinitis*, in which the mucous membrane is atrophied, is one of the causes of the offensive purulent discharge known as *ozæna*. The mucous membrane is thinned, and crusts collect on the surface, which may be abraded, but is seldom ulcerated. The sense of smell is lost.

**Treatment.**—For the hypertrophic form frequent sprays or douches of antiseptic solutions, containing carbolic acid, boric acid, borax, must be used; and if there is much thickening, it may be lessened by the use of the galvanocautery. Glegg's treatment, as described under Acute Rhinitis, may also be useful. The hygienic surroundings of the patient also require attention. For the atrophic form the treatment is very similar but less promising: crusts must be removed and antiseptics applied directly or by douche or spray. Tonics, such as iron, or arsenic, or cod-liver oil, are helpful.

## EPISTAXIS

Epistaxis, or bleeding from the nose, may depend upon local or general conditions. Among the former are acute and chronic catarrhal and tuberculous and syphilitic lesions and teleangiectases, and traumatic lesions, such as blows and picking. Epistaxis is not uncommon in childhood and early youth from causes that are not always obvious, unless it be simply delicacy of tissue; it is less common in middle age, but is again frequent in elderly people, whose vessels are beginning to degenerate. It is thus related to atheroma. It occurs also at all ages in connection with high blood pressure, Bright's disease, cirrhosis of the liver, cardiac valvular disease, the diseases of the blood, such as anæmia and

leukæmia in their different forms, purpura, scurvy, and some infectious diseases, such as enteric and relapsing fevers, and occasionally influenza.

The bleeding is often from the anterior part of the septum ; if blood flows from the posterior nares, it may trickle down the fauces into the stomach, and be subsequently vomited or passed *per rectum*, or it may cause cough and give rise to a suspicion of hæmoptysis. A pre-existing headache is sometimes relieved by a moderate bleeding in cases of high blood pressure.

Epistaxis, even if abundant, usually ceases of itself, but it may recur so frequently, and thus cause so much anæmia, that treatment becomes essential. It may be stopped by keeping the patient in a sitting posture, raising the arms above the head, and applying an ice bag to the back of the neck. If this is not sufficient, the local application (by means of a plug of cotton wool) of a solution of adrenalin chloride (1 in 5,000) should be tried. Failing this, the anterior or posterior nares should be plugged, and ergot, calcium chloride, or other hæmostatic may be given internally, or ergotin subcutaneously.

# DISEASES OF THE LARYNX

## LARYNGITIS

Laryngitis, or inflammation of the larynx, may be acute or chronic, and arises from a number of causes. Amongst these are—the ordinary conditions of catarrhal inflammation, considered under Acute Rhinitis ; contact with irritating vapours and air charged with dust ; the impaction of foreign bodies or direct injury in other ways ; extension of inflammation from surrounding parts, the pharynx, the bronchi and trachea, or the tissues outside ; the growth of tubercle, carcinoma, and syphilitic gummata ; acute specific fevers, such as those of diphtheria and measles ; and finally Bright's disease. The results differ somewhat according to the cause, and one can readily distinguish a catarrhal laryngitis, an œdematous laryngitis, the membranous laryngitis which is characteristic of diphtheria, and the laryngitis of phthisis and of syphilis.

### ACUTE CATARRHAL LARYNGITIS

**Ætiology.**—This is mostly due to the same conditions as may cause an acute rhinitis, but it also arises from irritating vapours, dusty air, the entrance of foreign bodies, and inflammation spreading from the posterior nares, pharynx or bronchi. It is one of the effects of the poison of measles, and less frequently of other infections.

**Morbid Anatomy.**—The disease consists of swelling and increased vascularity of the mucous membrane of the larynx, with the secretion of more or less mucus, or in later stages muco-pus. Occasionally slight abrasions of the epithelium occur, and less frequently hæmorrhage takes place into the tissue of the mucous membrane or on the surface. In very severe cases there is œdema of the submucous tissues. As a result of the inflammation of the overlying structures certain changes take place in the neuro-muscular apparatus of the larynx, especially paresis of the internal tensors.

**Symptoms.**—The voice becomes hoarse or entirely lost ; there is a tickling sensation in the throat, leading to a husky cough, with expectoration from time to time of small plugs of mucus. Respiration is generally but little affected, but there may, in exceptional cases, be some stridor ; and in children dyspnœa is much more often a marked symptom. Fever may be slight or none. On examination with the laryngoscope the mucous membrane over the arytenoids is swollen and red. The vocal cords may be slightly pink, but usually show very

little change. Some mucus may be seen lying between them. The ventricular bands may be affected.

Children are liable to a form of acute laryngitis (*laryngitis stridulosa*), which is characterised by the sudden development of suffocative symptoms, frequently in the middle of the night. During the day there is only slight cough and huskiness, but some time in the night the child wakes up suddenly in terror, with severe dyspnœa and a barking or husky cough, followed by loud and prolonged crowing inspiration. The voice is husky and feeble, and the features are congested; if the condition continues, the face may become pale and livid, and suffocation seems imminent. Usually, however, in a short time the symptoms become less severe, and the child falls asleep. Either on the same night, after a few hours' sleep, or on subsequent nights, the same attacks of threatening suffocation with croupy inspiration may take place. In association with these attacks there is more fever (white tongue, flushed face, hot skin, etc.) than commonly occurs in catarrhal laryngitis of adults. The attacks are probably due to laryngeal spasm set up by the presence of tenacious mucus in the glottis. The symptoms are apt to recur in the same child whenever it "catches cold"; they are, however, rarely fatal.

The **Prognosis** of acute laryngitis is mostly favourable; it generally subsides in the course of a few days.

The **Diagnosis** is generally simple, especially in adults: diphtheria is more severe, and may be accompanied by membrane on the fauces, by the expectoration of membrane, or by albuminuria.

**Treatment.**—The patient must not speak. He should be placed in a uniformly warm atmosphere, and should inhale steam from a suitable apparatus frequently. This may be charged with tr. benzoin co. (℥j to a pint of water), or benzoic acid, or ol. pini sylvestris (5 minims with 10 grains of magnesii carb. levis suspended in water ℥j), or lupulin (℥ss.). Sprays of menthol (20 to 30 grains in liquid paraffin ℥j), oil of eucalyptus, and creosote are also useful. Demulcent liquids should be drunk freely, or small pieces of ice may be sucked. The irritation of cough should be allayed by opiates. The diet or regimen usual in febrile affections will of course be followed. Local applications by the laryngeal brush seem not to be advisable till the later stages, when astringent solutions may be used like but weaker than those given on p. 202.

For laryngitis stridulosa an emetic is often useful, such as sulphate of zinc (5 to 10 grains) or ipecacuanha (2 to 5 grains of powder, or a drachm of the wine every ten minutes, till vomiting is produced). In addition, hot flannels or a hot sponge should be applied to the throat. In the intervals the laryngitis is to be treated by a warm moist atmosphere (steam kettle) and small doses of bromides and chloral.

### ŒDEMATOUS LARYNGITIS

**Ætiology.**—This may be a result of laryngitis arising in various ways. It sometimes occurs in catarrhal cases, and in the course of Bright's disease. It is, however, frequently the result of *sepsis*, whether local or general; it thus occurs among hospital nurses, students, and others exposed to such influences, and is often set up by inflammations of the pharynx, diphtheria, disease of the cartilages and perichondrium, such as occurs in enteric fever, syphilis, phthisis, and cellulitis of the neck. Injury and the contact of boiling water will also cause it. The localised œdema described as angeio-neurotic frequently occurs in the laryngeal tissues, and is often fatal (*see* Angeio-neurotic Œdema).

**Morbid Anatomy.**—It consists of an effusion of inflammatory serum into the submucous tissue, and the serum contains many leucocytes, so that it may be sero-purulent, or actual pus is diffused through the tissue.

**Symptoms.**—These are often very rapidly developed, and within a few hours the patient may be in imminent danger from the obstruction to respiration

produced by the swelling. Besides the dyspnœa, which is the prominent symptom, dysphagia is also experienced, the voice becomes hoarse and feeble, or disappears entirely, and there is some stridor in respiration. The laryngoscope reveals enormous swelling of the epiglottis, which forms a thick semicircular fold, or shows its two lateral halves much swollen and pressed together; in colour it is bright red. The swelling also affects the ary-epiglottic folds and the ventricular bands, but rarely the vocal cords themselves. The swollen epiglottis can be also felt by the finger, or even seen on depressing the tongue with a spatula. Care must be taken not to set up a spasm of the larynx.

The **Diagnosis** is confirmed by the use of the laryngoscope; in its absence the swollen epiglottis may be felt by the finger.

The **Prognosis** in cases of extensive œdema is a serious one.

**Treatment.**—The object should be to remove the laryngeal obstruction as soon as possible. Bleeding by means of leeches to the neck, adrenalin spray (1 in 2,000), or the same combined with cocaine, 2 per cent., ice sucked, or ice applied to the neck, potassium bromide in 10 or 20-grain doses, and the subcutaneous injection of pilocarpin nitrate may cause the œdema to subside; but if the dyspnœa is urgent, intubation or a low tracheotomy must be performed.

### MEMBRANOUS LARYNGITIS

**Ætiology.**—The most common cause of membranous laryngitis is diphtheria, which either begins in the fauces and spreads to the larynx (*see* p. 68) or attacks the larynx at first, without either then, or later, involving the fauces. It is of interest to note that these primary laryngeal cases are more common in children than in adults, and that they are less often accompanied by albuminuria, or followed by paralysis, than those in which the throat first suffers.

A membranous laryngitis may certainly be produced by traumatic causes or local irritants, such as chemical vapours, boiling water, or impacted foreign bodies. A membranous laryngitis in measles means a superadded diphtheritic infection.

**Symptoms.**—The local symptoms will be like those already described under Diphtheria, but the toxic symptoms of an infectious disease will be absent or little pronounced when the cause is more distinctly traumatic.

**Diagnosis.**—In the majority of children taken with dyspnœa, ringing or "croupy" cough, and inspiratory retraction of the chest wall, without apparent cause, and threatened with suffocation in from one to four days, membranous laryngitis is present; and in the majority of these again diphtheria is the cause: but it is generally impossible to examine with the laryngoscope, and the first proof of the presence of membrane may be provided at or after the operation of tracheotomy. It is distinguished from laryngitis stridulosa (*see* p. 200) by the more gradual development and more uniform progress of the dyspnœa.

**Treatment.**—Membranous laryngitis may be treated as shown under Diphtheria both when it is due to this disease and when it is associated with scarlet fever, measles, or other infectious illness. The antitoxin of diphtheria should be used in the first case.

### CHRONIC CATARRHAL LARYNGITIS

**Ætiology.**—This often follows upon acute laryngitis, especially when the latter is not properly treated with complete rest of voice. It results also from the failure to breathe properly through the nose, when the nasal passages are obstructed by deviation of the septum, turbinal thickenings, hypertrophic rhinitis, or adenoids, and is often due to faulty methods in those who sing and speak in public. It occurs in those who use the voice continuously for long

periods, and with much exertion, like costermongers, schoolmasters, and clergymen. It may be associated with the pharyngitis so often induced by the excessive use of alcohol or tobacco, or infection may spread from the mouth in cases of oral sepsis.

**Symptoms**.—There is hoarseness of voice, accompanied by dryness, irritation of the throat, and tickling cough on prolonged use of the larynx, or there may be loss of voice. The cough is frequent, but there may be only a little hawking up; and the expectorated secretion, which is mostly viscid mucus, is never abundant. The symptoms are often most marked after an interval of rest, and disappear during the effort of talking, until after a time fatigue again ensues. Dyspnœa is generally absent. With the laryngoscope more or less congestion of the mucous membrane is seen; this may be diffused or unequally distributed, and mucus is here and there adherent to the surface. In old cases the mucous and submucous tissues become swollen and thickened, especially over the epiglottis, inter-arytenoid fold, and ventricular bands, and the vocal cords may become thickened, granular, or have nodules upon them (*chronic hypertrophic laryngitis, singers' nodules*). A still more advanced stage, where there is fleshy thickening of the cords, is known as *Pachydermia laryngis*. The mobility of the vocal cords is impaired. The swollen inter-arytenoid fold may project between the cords on phonation so as to hinder their closure.

**Diagnosis.**—This must be made from the history and the laryngoscopic appearances, in which the thickening has to be distinguished from the transparent swelling of œdema and the dull red swellings of tuberculous laryngitis. In cases of long standing a possible connection with phthisis must be carefully considered; in older patients carcinoma may cause a chronic thickening, which is likely to affect one cord only, and to impair its movement.

**Treatment.**—The condition is very troublesome, and requires persistent treatment. Oral, pharyngeal or nasal sepsis should be treated. The patient should protect himself from cold and exposure by suitable clothing, as well as by confinement to the house in bad weather; and he should talk as little as possible. Attention to the bowels and the use of iron tonics and strychnine are desirable; and change of residence to a mild and equable climate may be beneficial, or even necessary. Astringents should be applied by means of the laryngeal brush or of the laryngeal syringe. For this purpose the following solutions may be used: iron perchloride, 10 to 20 grains to the ounce of water; copper sulphate, 10 grains; zinc chloride, 30 grains; silver nitrate, 10 to 30, or even 60, grains; zinc sulphate, 10 grains; alum, 30 grains. One of these should be employed daily for seven days on alternate days during the next two weeks, and so on with gradually decreasing frequency. For cases with excessive secretion turpentine may be locally applied, and carbolic acid (℥ss.—℥j to glycerin ℥j) for cases with long-standing hyperæmia and diminished secretion. Codeia in ¼-grain doses is recommended for the tickling cough. Where breathing through the nose is difficult, operative treatment may be considered.

## TUBERCLE OF THE LARYNX

Of patients suffering from phthisis, or pulmonary tuberculosis, a considerable number have a laryngeal affection, which was formerly described as laryngeal phthisis. This is due to the actual invasion of the laryngeal tissues by tubercle, and it is secondary to the formation of tubercle in the lungs. It is usually known as "laryngeal tuberculosis," and was present in 4·8 per cent. of phthisical patients admitted to King Edward VII.'s Sanatorium, Midhurst, in the first stage of the disease, in 18·3 per cent. in the second stage, and in 31·5 per cent. in the third stage. Men and women have an equal tendency to the disease (**5**). A second form of tubercle affecting the larynx is lupus, which spreads from the pharynx or nose.

## LARYNGEAL TUBERCULOSIS

The tubercles occur as minute collections of cells in the mucous or submucous tissues, forming, perhaps, slight prominences on the surface, leading in time to more or less, often considerable, œdema of the surrounding parts, and later to ulceration. Extending more deeply in severe cases, with the assistance of pyogenic organisms, the inflammatory process leads to deep ulceration, to perichondritis and to necrosis of the cartilages. The most frequent seats of the deposit are in the neighbourhood of the posterior commissure, *i.e.* the inter-arytenoid area, the anterior surface of the arytenoid body and the vocal processes. Next in frequency come the vocal cords. The epiglottis and anterior commissure are involved late in the disease, and rarely at the beginning. Paralysis of the vocal cords may be simultaneously caused by thickening of the right pleura involving the right recurrent laryngeal nerve, or tuberculous bronchial glands pressing upon one or both.

The **Symptoms** are those of a chronic laryngitis, and in cases of ordinary severity consist of hoarseness of voice, frequent husky cough, and pain on swallowing. Sometimes in early stages the voice may be lost entirely from functional failure, in later stages from paralysis of a vocal cord, or extensive destruction; and swallowing may be not only painful, but difficult on account of swelling of the tissues, or from their destruction preventing perfect closure of the larynx. The cough is occasionally severe and paroxysmal, and expectoration is variable, depending rather on the condition of the lungs than on that of the larynx. In a small number of cases, considerable obstruction to respiration arises. In 12 per cent. of cases the larynx was found to be tuberculous without producing any local symptoms at all. In early stages the laryngoscope shows pallor of the mucous membrane, and a decided anæmia of the larynx occurs quite early in many cases of phthisis. When infiltration takes place the parts often assume a characteristic appearance, the ary-epiglottic folds on one or both sides being swollen up into a pale globular or pyriform tumour, the base backwards, the point forwards; and when both are affected the swellings coalesce in the middle line. The epiglottis may form a turban-shaped swelling; and the same thickening may affect the ventricular bands, which are, however, often concealed. Subsequently ulcers form upon the swollen tissues as well as upon the vocal cords, especially in the posterior halves. A frequent characteristic is ulceration with granulations in the inter-arytenoid space.

**Diagnosis.**—This must be made partly from the laryngoscopic appearances, and partly from the condition of the lungs, which are in many cases obviously tuberculous. The pyriform swellings of the ary-epiglottic fold are characteristic of the condition, but when they are absent there may be difficulty in distinguishing it from *chronic catarrhal laryngitis* and from *syphilitic* disease. In the former there are less swelling and more congestion than in tuberculous laryngitis; in syphilis the ulcers are generally larger and deeper, situated upon a more inflamed base, and solitary; the thickening is more irregular, and the disease often unilateral. The ulceration of carcinoma is sometimes difficult to distinguish from that of tubercle; carcinoma is often unilateral, and occurs in older patients.

**Prognosis.**—The presence of laryngeal tuberculosis makes the prognosis of the co-existing phthisis more gloomy (*see* p. 259). Recovery from laryngitis occurred in 25 per cent. of 477 cases at Midhurst. Very often the laryngeal condition progresses or retrogresses as the phthisis progresses or retrogresses; but this is not always the case. The larynx may recover, even though the phthisis gets worse; but with proper treatment the larynx never gets worse if the phthisis is improving.

**Treatment.**—It is essential to treat the disease in the lungs of which the laryngeal tuberculosis is really a part. The most important element in local treatment of the larynx is complete silence on the part of the patient; not even

whispering should be allowed. This may cause great mental strain in some patients. In such cases occasional whispering may be allowed. Where there is much infiltration galvano-caustic puncture with a fine platinum point introduced deeply into the tissue has been successful, but this should be employed only when there is no evidence of *active* disease in the lungs. Three or four punctures may be made at a time, after the production of anæsthesia by instillation of 5 drops of a 20 per cent. solution of cocaine. The puncture may be repeated at not less than two weeks' interval (D. Grant). Other methods of local treatment, *e.g.* insufflations of the larynx with orthoform and other powders, spraying with 10 per cent. cocaine, which the patient must never be allowed to carry out himself, cannot be compared in value with the methods just mentioned, and have now largely been given up. In advanced cases tracheotomy may be considered in order to rest the larynx.

### LUPUS OF THE LARYNX

Lupus, of which a fuller description is given under Diseases of the Skin, rarely attacks the larynx, although the systematic examination of this part in persons with lupus of the skin has shown it to be often present when not in the least suspected.

It occurs as pale or dark red nodules, or papillary outgrowths, isolated or grouped upon a hyperæmic base, appearing first and most often upon the epiglottis, then upon the ary-epiglottic folds, and posterior wall of the larynx, and least often upon the vocal cords. As they increase in size they cause a general thickening of the parts. Later on ulceration takes place, and the ulcers are followed by scars, in which again fresh nodules may appear; but the cicatrisation rarely leads to high degrees of stenosis. Some nodules may be absorbed without ulceration.

**Symptoms.**—There are some soreness, trouble in swallowing, and alteration in the voice, which becomes hoarse or is even lost. Dyspnœa may occur in late stages.

The **Prognosis** as far as life is concerned is good; but occasionally phthisis supervenes, especially if the tongue is involved. As regards recovery the prognosis is not so good, as it tends to heal at one spot and slowly extend at another, as in the pharynx.

**Treatment.**—This consists chiefly in local applications of tincture of iodine, iodoform, nitrate of silver and lactic acid, and the use of the electric cautery, as in laryngeal tuberculosis. However, measures applicable to tuberculosis in general are more important than local treatment.

## SYPHILIS OF THE LARYNX

Syphilis affects the larynx in many ways: in the hereditary form in infancy and childhood; in the acquired form in secondary and tertiary and intermediate stages. The secondary lesions of acquired syphilis are chronic hyperæmia, superficial ulcerations, and condylomas or mucous patches, of which the last are very rare. In the later stages of the disease a diffuse infiltration of the larynx is the most common. Small gummas varying in size from a pin's head to a pea and deep ulcerations are occasionally seen. Laryngeal œdema and perichondritis with laryngeal necrosis occasionally result, and the cicatrisation of ulcers may lead to the union of parts of the larynx to each other, or to the pharynx, so that serious distortions of the larynx or contractions of the glottis ensue. Thus the cords may be united by a web, or the epiglottis may be fixed to the pharynx.

**Symptoms.**—These are not distinctive, and vary much according to the severity of the lesion. They are hoarseness or loss of voice, occasionally cough in

earlier stages, and more or less dyspnœa in later stages. Swallowing is often painful, though the absence of pain when not swallowing is remarkable. Muco-purulent expectoration with blood may accompany extensive ulceration, and in a few cases free hæmorrhage has taken place.

**Diagnosis.**—Syphilis of the larynx must be distinguished from chronic laryngitis, bilateral abductor paralysis, tubercle and carcinoma. In this last the ulcers are generally preceded and accompanied by growth, in the form of nodular excrescences; they may be very large, and the surrounding tissue is inflamed. Dyspnœa and stridor in persons admitting a previous syphilitic infection may arise from paralysis of the vocal cords, from aneurysm pressing on the trachea or laryngeal nerves, or from syphilitic stenosis of the trachea or one bronchus.

**Prognosis.**—The condition may endanger life if an acute laryngitis is superposed. It is particularly resistant to all forms of antisyphilitic treatment, and often requires in course of time a tracheotomy for life.

**Treatment.**—Vigorous antisyphilitic treatment should be carried out. There is a belief, hardly justified, that potassium iodide may cause œdema of the glottis. Tracheotomy should be carried out in order to rest the larynx in the early stages and before cicatricial contraction makes it imperative. In the latter event a tube has to be worn for life; but attempts may be made to dilate the glottis mechanically, or to divide a web by the cutting forceps or dilator, or by the electric cautery.

## LARYNGEAL PERICHONDRITIS

This is mostly the result of phthisis, carcinoma, syphilis, or enteric fever affecting the larynx, or of simple chronic laryngitis. It may also arise from traumatic causes. As a result of the inflammation the perichondrium becomes thickened, pus forms in its fibres and collects between it and the cartilage, which, separated from its nutritive supply, becomes necrosed. The structures superficial to the perichondrium also become inflamed, œdematous, and purulent; thus an abscess is formed, which contains the dead fragment of cartilage. This sequestrum may remain for months or years after the opening of the abscess; on its removal the parts will contract and cause deformity and stenosis of the larynx.

**Symptoms.**—Dull, aching pain, tenderness on manipulation with difficulty of swallowing, and hoarseness of voice or aphonia, are the usual symptoms, varying somewhat according to the cartilage affected, and often very much masked in secondary cases; thus in typhoid fever loss of voice may be the only symptom suggesting an inquiry into the condition of the larynx. If the membrane on the outer side of the thyroid is affected, there will be swelling in the neck and formation of an abscess. If the inner surface of the thyroid or the cricoid or arytenoid cartilage is affected, there is a corresponding œdematous infiltration of the interior of the larynx, which will be visible with the laryngoscope. The mobility of the vocal cords may be lessened by paralysis of the posterior crico-arytenoids when the cricoid cartilage is diseased, and by direct implication of the cord when one arytenoid is affected.

The **Prognosis** is unfavourable; when the primary cause, as typhoid fever or syphilis, is not in itself fatal, troublesome contractions of the glottis ensue after discharge of the cartilage; and pneumonia and gangrene of the lung have occurred from insufflation of the septic secretions.

**Treatment.**—In acute cases, the inflammation may be reduced by the application of leeches or an ice bag to the surface. If an abscess forms, it must be incised—from within if the arytenoid or epiglottis, by incision in the neck if the thyroid or cricoid cartilage, is concerned. Often, however, tracheotomy is required, and before the tube can be dispensed with the contraction of the glottis has to be treated perseveringly by dilatation or other operative measures.

# TUMOURS OF THE LARYNX

## BENIGN TUMOURS

These are papilloma, fibroma, mucous cyst, myxoma, angeioma, and lipoma. The last three are quite rare, and occur especially in children.

*Papillomas* are the most common. They are frequently about the size of a pea, but may be as small as a mustard seed, or in rare cases as large as a walnut. They are pink, whitish grey, or red, have an uneven, or papillated, or warty surface, and grow mostly from a broad base. They are often multiple, and their usual seat is the vocal cord on one or both sides, or the angle between the cords, sometimes the ventricular bands or the epiglottis. They are liable to recur after removal.

*Fibromas*, or fibrous polypi, are of slower growth, and show no tendency to recur. They are round or oval, sessile, or pedunculated, with a smooth surface and bright red colour. Usually of hard consistence, they are more rarely soft, and contain a good deal of serous fluid in the meshes of the fibrous tissue. They are generally single, and arise from one of the vocal cords.

*Mucous cysts* arise commonly from the epiglottis, and are surrounded with an area of injected mucous membrane. They have dense walls, and are filled with thick, white, sebaceous material, or thinner yellowish or brown fluid.

The **Symptoms** of tumours depend upon their seat. If situate upon the vocal cords, the voice is impaired or lost; and impairment of voice is the commonest symptom. If the tumour is sufficiently large, dyspnœa is present. Dysphagia from implication of the epiglottis and cough, generally dry and hacking, also occur. In children the cough may be croupy. Pain appears to be rare.

The **Treatment** is removal by surgical operations, for the details of which the reader is referred to surgical works, or special treatises.

## MALIGNANT TUMOURS

These are mostly carcinoma, but sarcoma also occurs. They are more frequent in men than in women, and appear commonly in advanced life. *Intrinsic carcinoma* of the larynx originates (1) on the vocal cords, more commonly on the anterior and central than the posterior regions; (2) in the subglottic region, more commonly in the anterior part of the larynx. A carcinoma of the cord may remain limited for a long time to the cord and the adjoining side of the larynx, but may spread eventually (*a*) across the anterior commissure; (*b*) to the subglottic region; (*c*) to the arytenoids. Eventually the whole larynx may be involved. In the later stages it ulcerates, vegetations spring up about the margins, and these ulcerate in their turn. The surface is often covered with pus, or sanguineous muco-pus, and occasionally free hæmorrhage takes place. Œdematous laryngitis and perichondritis occur as complications. The larynx is, of course, affected sometimes by carcinoma spreading from the pharynx or the neck (*extrinsic carcinoma* of the larynx).

**Symptoms.**—The earliest symptom is huskiness or hoarseness of the voice. Laryngoscopic examination discloses a tumour. The cord is often freely movable during the earliest stages, but becomes fixed when the growth spreads. In the later stages there may be severe pain, dyspnœa and dysphagia. As ulceration proceeds the breath becomes fœtid, and hæmorrhage may occur. Occasionally the submaxillary glands are implicated.

The **Diagnosis** rests on the age of the patient, the symptoms and laryngoscopic appearances already described. Tubercle tends to attack rather the posterior than the anterior part of the larynx.

The **Prognosis** is relatively favourable if the condition has been diagnosed

in the earliest stage. From a series of fifty-one cases in which the growth was removed after laryngo-fissure there was no recurrence in 80 per cent from one to thirteen years later, but a quarter of these had died from other causes. There was local recurrence in 16 per cent. The immediate operative mortality was very small (**6**).

The **Treatment** consists in removing the growth after laryngo-fissure.

## FOREIGN BODIES IN THE LARYNX

A large number of foreign bodies have at different times found their way into the larynx. Among these are peas, beans, buttons, coins, fragments of bone, shells, pebbles, artificial teeth, portions of solid food, and pieces of children's toys.

The **Symptoms** are divisible into three stages :— (1) The *initial spasm*, which takes the form of a violent fit of coughing, which usually dislodges the object. If this does not occur, the obstruction may be rapidly fatal; but if not, there follows (2) the *quiescent period*, which may last any time from a few hours to many years. The symptoms may be so slight that it is not always known to the patient or to his friends that a foreign body has been introduced. (3) The stage of *inflammation*, due to infection, produces secondary symptoms, hoarseness, pain, coughing, etc.

At any stage a change in the position of the foreign body may cause sudden death.

**Treatment.**—During stage 1 it may be useful to hold the head down, to help dislodge the body. If the symptoms appear dangerous, a tracheotomy should be performed. During stage 2 the body should be located and removed by means of an endoscope. If opaque to the X-rays, these may also be useful in locating the body. In a child up to twelve years old a tracheotomy should always be done before the endoscope is used.

## PARALYSIS OF THE LARYNGEAL MUSCLES

From the peculiar course of the recurrent laryngeal nerve—the chief motor nerve of the larynx—paralysis of these muscles has often a diagnostic importance beyond that of the trouble arising locally. But it may be caused by lesions not only of the laryngeal nerves, but also of the vagus above their origin, and of the medulla oblongata where the nuclei are situated. Thus laryngeal paralysis is a part of bulbar paralysis, results from syphilis and tumours affecting the medulla oblongata, and occurs occasionally in association with tabes dorsalis, general paralysis, syringomyelia, and disseminated sclerosis. The vagus in the neck may be compressed by tumours and enlarged glands, or may be injured by bullet wounds or cuts, whether accidental or in the course of surgical operation. The recurrent laryngeal nerves are in danger in two situations: the thorax and the neck; and the left is the more liable to lesion from its curving round the arch of the aorta, whereas the right goes no lower than the subclavian artery. Either of them may be involved in the fibrous thickening at the apex of the lung in chronic phthisis, but the left is especially liable to be compressed by aneurysm of the arch of the aorta, by mediastinal tumours, by enlarged bronchial glands, and by the dilated left auricle in mitral stenosis. In the neck the two nerves ascending to the larynx lie between the trachea and the œsophagus, and may be involved together in carcinoma of the latter, or compressed by an enlarged thyroid body. Paralysis also occurs, as already stated, in catarrh, and from other local affections of the larynx; as a result of diphtheria, influenza, and other infectious diseases, chronic alcoholism, and poisoning by lead and arsenic; and, finally, as a purely functional failure, or part of hysteria.

## COMPLETE PARALYSIS OF THE VOCAL CORDS

This is the result of lesions dividing or severely injuring the recurrent laryngeal nerve, or the vagus above, or of diseases of the medulla oblongata—*e.g.* bulbar paralysis. Of the nerve lesions neuritis from alcohol and diphtheria must not be forgotten. The paralysis may be bilateral, which is very rare, or unilateral. When bilateral, the cords assume what is known as the *cadaveric position*. They are immovably fixed midway between the positions of *ab*duction and *ad*duction, each cord with a concave margin, leaving a fusiform interval between them, unaffected by inspiration, expiration, or attempts at phonation. The aperture is sufficiently wide for respiration: hence there is no dyspnœa; but phonation is impossible, as the cords cannot be approximated, so that the patient speaks in a whisper, and the acts of coughing and expectoration are imperfectly performed. Some stridor is produced on forced inspiration, probably from the vocal cords, the ary-epiglottic folds, and arytenoid cartilages being thrown into vibration.

When the paralysis is unilateral, the affected cord assumes the cadaveric position, while the sound cord has its full mobility. Again, there is no dyspnœa. The voice may be entirely lost, but often it is produced by the arytenoid of the healthy side being drawn completely over the median line till it comes into contact with the paralysed cord, so that the glottis lies obliquely with its anterior end in the middle line and its posterior end much to the affected side. The voice is then harsh sometimes, and may break into falsetto on an increase of the effort. The cough has often a peculiar brassy or clanging quality.

## PARALYSIS OF THE ABDUCTORS

Although the recurrent laryngeal nerves, supplying as they do all the muscles of the larynx except the crico-thyroid, must contain fibres for both *ad*ductors and *ab*ductors, it is a remarkable fact that coarse progressive lesions of these nerves (compression by tumours or aneurysms) result at first in paralysis of the *abductors* alone; it is only later that the internal tensors (*thyro-arytænoidei*) are affected, and last of all the chief adductors (*crico-arytænoidei laterales*). The abductor fibres form a separate bundle lying internal to the adductor fibres in the recurrent laryngeal nerve of the dog (Risien Russell); but their greater liability to suffer from lesions affecting the whole nerve is apparently due, as shown experimentally, to less powers of resistance to external influences. Abductor paralysis also results from lesions of the medulla where it may be supposed it sometimes depends on a separate affection of the nucleus of the abductor fibres, though adductor paralysis alone never arises under such circumstances. Syphilis and tabes are the most common associates of abductor paralysis arising in this way. It is to be noted that there are no supranuclear lesions of the abductors. Abductor paralysis is probably sometimes the result of a change primarily in the muscle.

The effect of the lesion is that the cord during respiration, not being fully abducted, remains in the cadaveric position, and at first allows ample space for the passage of air; after a time, however, the antagonistic muscle, or adductor, contracts (*paralytic contracture*), and the cord is drawn into a position of adduction. Thus in bilateral paralysis of the abductors the cords are seen to be permanently approximated in the middle line to within one-tenth of an inch of each other; on attempted phonation they meet completely in the middle line; on inspiration they do not separate, but are even drawn a little closer together; on expiration they scarcely move, or only in the reverse sense to their slight movement in inspiration. The important symptom is *dyspnœa*, which results from the permanent narrowing of the glottis; this is generally accompanied by *stridor* on inspiration, which is worse on exertion, and often extremely loud during sleep. The voice is clear, or it may be a little hoarse. Coughing can be perfectly effected.

When only one cord is paralysed, dyspnœa only occurs on exertion, and the stridor is less or absent. On phonation the healthy cord meets the paralysed cord in the middle line, and the voice is normal.

**Diagnosis.**—Abductor paralysis may be confounded with spasm of the adductors, with ankylosis of the arytenoids in the position of adduction, and with perverted action of the cords in which they move inwards instead of outwards during inspiration. When the arytenoid is ankylosed the cord is absolutely fixed, and there is generally some thickening about the joint.

It is important to remember that the lesion which causes unilateral or bilateral abductor paralysis may at the same time cause narrowing of the trachea by pressure (aneurysm, tumour) or by cicatrix (syphilis), and the dyspnœa and stridor due to the latter may be wrongly attributed to the former. Tracheal stenosis usually causes expiratory as well as inspiratory stridor; nevertheless the certain recognition of a tracheal obstruction in the presence of laryngeal stenosis is by no means easy (*see also* p. 214). The diagnosis of the remote cause of the paralysis has next to be made by a consideration of other symptoms, such as those in favour of tabes and central nervous lesions, or of thoracic aneurysm and new growths in the neck or chest. Aneurysm is a very frequent cause of paralysis of the left vocal cord. Wassermann's test and the Röntgen rays may have to be used.

The **Prognosis** is generally serious. Except when syphilis is the cause, there is little hope of recovery; and while some cases may last for years without any change, there is a constant liability to death from suffocation. If the adductors are subsequently paralysed, the obstruction to breathing is diminished, but aphonia ensues. Death may arise from the primary lesion, such as œsophageal cancer or double aneurysm. In long-standing cases the posterior crico-arytenoid muscles become completely atrophied.

**Treatment.**—If the cause of double paralysis is central, or if syphilis is the cause, a vigorous antisyphilitic treatment should be instituted. But if no improvement takes place in a few weeks, and if dyspnœa is constant, or night attacks take place, tracheotomy should be performed, and the tube should be worn constantly.

In unilateral paralysis the risk of asphyxia is much less, and the treatment may be directed mainly to the cause.

### PARALYSIS OF THE ADDUCTORS

This is a functional disorder, and rarely occurs alone from structural lesions. The adductors are the lateral adductors, or *crico-arytænoidei laterales*, and the central adductor, or *arytænoideus proprius;* the inner fibres of the *thyro-arytænoidei*, or internal tensors, also act as adductors of the anterior portions of the vocal cords. In the most common form of adductor paralysis these are all affected. When examined with the laryngoscope, the glottis is seen to be widely open; on attempts to speak the cords scarcely move, but remain still at the sides of the larynx. As the cords cannot be approximated, the patient speaks only in a whisper, no laryngeal voice being produced, though sometimes, with an effort, a momentary contact of the cords may be effected. Coughing, in which the cords are brought together by involuntary reflex action, is generally perfect; and from the open condition of the glottis there is no dyspnœa. This constitutes *functional* or *hysterical aphonia*, which is, however, often started by slight catarrh of the larynx, *e.g.* in the earliest stage of phthisis, or by sore throat, or by other local trouble, both in definitely hysterical persons and in others suffering from anæmia or general weakness. In civil practice it occurs in young women and boys as the result of an emotional shock, the individual being struck dumb with terror. The same explanation must be given for most of the numerous cases of aphonia in soldiers invalided from the War, especially when they were

exposed to shell fire, or buried after explosions, or otherwise directly injured. Some of the cases, in which a child fails to speak after the removal of a tracheotomy tube worn for some weeks, are also due to functional adductor paresis.

Sometimes the adductor paralysis is less extensive; the internal tensors may be alone affected, so as to produce want of contact of the cords on attempted phonation, each cord presenting in its anterior half a concave margin towards the middle line. And sometimes the central adductor is paralysed, in which case the anterior portions of the cords come into contact, and a triangular space is left open behind, between the arytenoid cartilages. These last two forms are not uncommon in the course of catarrhal laryngitis. They may occur together, producing defective closure in front and behind, while the processus vocales are in contact. In these cases the loss of voice is not so complete as in that first described.

The **Diagnosis** of these conditions is easily made with the laryngoscope. Even without this, the voicelessness of the patient, the absence of dyspnœa, cough, and expectoration, and the power to cough at will, are sufficiently distinctive. Should there be, however, any visible evidence of catarrh, the possibility of a tuberculous lesion underlying it should not be forgotten.

The **Prognosis** is favourable, and cases of many years' duration may be at length cured.

**Treatment.**—Functional aphonia is to be considered as a symptom of hysteria, and to be treated as such. In most cases it may be removed by means of the methods of suggestion and re-education. In some instances it is sufficient after a preliminary explanation of the fact that the weakness is not due to any organic disease, but is rather of the nature of a forgotten habit, to induce the patient to cough, and to prolong the noise in the form of an "a-a-h." From this he is led on to the letter "A," and so through the alphabet, beginning with the vowels, when it may be demonstrated to his own satisfaction that he can now phonate properly in the formation of words and sentences. This method must not be used if there is any suspicion of tuberculosis. In other cases the application of a faradic current externally in the neighbourhood of the larynx or to the back of the throat may be useful as a method of crude suggestive treatment. If the symptom is not amenable to such methods or recurs, or if it is associated with other evidences of hysteria, further treatment should be undertaken on the lines recommended under that heading (*see* p. 907).

### PARALYSIS OF THE EXTERNAL TENSORS

The crico-thyroid muscles may be paralysed on one or both sides, but this is not a common occurrence. It arises from cold, or from prolonged use of the voice. The voice becomes gruff, or may be entirely lost. With the laryngoscope the cords are seen to be applied to each other in a wavy line, instead of being perfectly straight and parallel. This condition should arise from lesions of the superior laryngeal nerve, such as the pressure of an inflamed gland; but it is more often seen in connection with diphtherial paralysis, or as the result of a bulbar lesion.

Paralysis of the thyro-epiglottic and ary-epiglottic muscles, which depress the epiglottis, occurs also from lesion of the superior laryngeal nerve, and is accompanied by anæsthesia.

## SPASM OF THE GLOTTIS

In this affection the adductors are spasmodically contracted, and complete closure of the glottis takes place, preventing the entrance of air, and producing asphyxia, or even death. It may occur at all ages, but is especially frequent in infants, in the form now to be described.

## LARYNGISMUS STRIDULUS

*(Spasmodic Croup, Child-crowing)*

This occurs between the ages of three months and two years, and is more common in boys than in girls. It is promoted by imperfect hygienic conditions, and is more frequent among the poor, and in children who are hand-fed, or nursed by sickly and half-starved mothers. In the majority of cases (75 per cent.) there is evidence of rickets, and the disease often occurs in children who have symptoms of tetany. Laryngismus stridulus is also apt to follow whooping cough. Attacks occur by night and day; but a number of causes may excite a spasm, such as crying, sucking, quick movements, milk getting down the larynx, indigestible food in the stomach, the irritation of dentition, and fits of anger; but the attacks often occur without any such obvious antecedent. The child may be in fairly good health when it is noticed to make a slight crowing sound occasionally. This may be repeated at intervals without giving rise to any alarm, but it gradually becomes more frequent. After a while the interruption to respiration, at first only indicated by the crowing, becomes more marked. Breathing ceases, the chest is fixed, the face becomes pale and livid, the head is thrown back, and the facial muscles are slightly twitched. In a short time the spasm yields, and the air enters with a loud crowing noise through the still imperfectly opened glottis; and the child in a few minutes more may return to its playthings. In the severest cases the glottic spasm is accompanied by the *carpopedal contractions* of tetany; the fingers are bent into the hand, the thumb within the fingers, and the hand is flexed on the wrist; the legs are extended, the feet bent on the legs, the soles turned inwards, and the great toe widely separated from the others. General convulsions may be added to these. Occasionally death takes place during a fit, from complete stoppage of the respiration; and as the crowing is really the signal that the spasm is relaxing, it will be seen that in the fatal cases death may occur quite silently.

**Diagnosis.**—The symptoms are very characteristic, and not easily confounded with those of any other disease. The absence of fever, the shortness of the attack, and the completely healthy condition between the attacks, distinguish it from laryngitis. It may be simulated by the presence of a foreign body (*see* p. 207).

**Prognosis.**—Most patients recover completely, but occasionally deaths are reported.

**Treatment.**—This has to be considered in relation to the general health of the patient, and the occurrence of the attacks. The child must be put immediately under the best possible hygienic conditions: fresh air, well-ventilated rooms, and improvements in its food where this is insufficient or unsuitable (*see* Rickets), and attention to the bowels should be secured. Medicinally cod-liver oil, or cod-liver oil with malt extract, is of great value, and potassium bromide may be given three times daily, in doses of 2 to 5 grains, according to the age of the child, and small doses of chloral. If the attacks are slight, sponging the child from head to feet two or three times daily with cold or tepid water, according to the season, often quickly stops them. In the more severe fits, the head should be raised, the surface of the body and face slapped with a towel dipped in cold water, and ammonia or acetic acid held to the nostrils, or the body may be immersed in warm water, and cold water poured over the head and face. The finger should be inserted and the epiglottis pulled forward.

## SPASM OF THE GLOTTIS IN ADULTS

This occurs more frequently in connection with laryngitis, œdema of the larynx, paralytic conditions, or the presence of foreign bodies; it may also be a danger in epilepsy, chorea, tetanus, hydrophobia, and tabes dorsalis. In the last it constitutes the severer form of laryngeal crisis (*see* p. 790); and this may

occur in the healthy larynx, or in one already affected by a paralysis, which is generally of the abductor variety. The entrance of saliva or small particles of food or drink into the larynx may cause most dangerous spasm, and a certain amount is often induced by the application of medicated solutions to the mucous membrane of the larynx. Spasm of the glottis is often the result of hysteria. Allied to this is a functional spasm (*phonic spasm* or *mogiphonia*), brought on in some neurotic persons by the effort of speaking, and relaxing when the attempt to speak is abandoned; it may be confined only to the use of the voice in public, as in singers and teachers.

**Treatment.**—In the first class of cases inhalation of chloroform, amyl nitrite, vapor coninæ, or burning stramonium should be employed, if they can be obtained in time; otherwise tracheotomy may be necessary. The bromides may be given for recurrent attacks.

Hysterical cases require the general treatment of hysteria; and the other functional conditions must also be treated with reference to the general condition of the patient, as well as by breathing exercises, and exercises in voice production.

## CONGENITAL LARYNGEAL STRIDOR

Infants are occasionally the subjects of a laryngeal disorder, in which the breathing is accompanied by a peculiar croaking sound. This is generally first heard soon after birth, is continuous for long periods, perhaps all day and night, but may be absent for a few hours at a time. The croaking takes place with inspiration, and is either a rough sound, or more clear and musical; expiration is silent; the cough and cry are, as a rule, normal. There may be a little sucking in of intercostal spaces, but there is rarely any lividity. In some cases the noise is constant during sleep; in others it is absent. It is generally worse when the child is lively or excited. It subsides as the child grows older, but Sir Frederick Taylor found it still present at two and a half years. The child appears to be in other respects quite healthy.

Both during life and after death the glottic aperture is seen to be extremely narrow, the epiglottis being folded on itself, and the ary-epiglottic folds almost in contact. This, however, is only an exaggeration of the normal condition of the infant's larynx, and Dr. Paterson has shown, by direct observation in five cases, that the stridor was due to the drawing into the larynx at each inspiration, and the vibration in this position, of the arytenoids and lax mucous membrane on the upper edge of the cricoid.

The obstruction caused by the deformity diminishes as the parts develop. No direct treatment is of any avail. Tracheotomy might be necessary in the rare event of life being threatened by asphyxia.

## ANÆSTHESIA OF THE LARYNX

This occurs in diphtheria, in bulbar paralysis, in tabes dorsalis and general paralysis, and from injury to the vagus or superior laryngeal nerve. It is recognised by the insensibility of the laryngeal mucous membrane when touched with a probe, introduced with the help of the laryngoscope. It is often accompanied by dysphagia from particles of food entering the larynx, the result, according to M. Mackenzie, of paralysis of muscles supplied by the superior laryngeal nerve, *i.e.* those which depress the epiglottis and close the upper aperture of the glottis during swallowing. Anæsthesia from diphtheria usually recovers; the prognosis is generally bad in progressive bulbar paralysis and allied conditions, as food is apt to get into the lungs and set up pneumonia.

The **Treatment** should be by means of galvanic and faradic applications. Strychnia may be given internally, and dysphagia may necessitate feeding with the œsophageal tube.

# DISEASES OF THE TRACHEA

## TRACHEITIS AND SPECIFIC INFECTIONS

Inflammation of the trachea arises from circumstances similar to those producing laryngitis. Acute catarrhal tracheitis frequently accompanies laryngitis and bronchitis, but is masked by the symptoms which they produce. Occasionally it exists alone. It then produces cough, often hacking, perhaps violent or paroxysmal, with some amount of expectoration. At the beginning of influenza the tracheitis may be accompanied by substernal pain of unknown origin. With the laryngoscope the mucous membrane may be seen to be congested, and ulcers are sometimes observed. With the stethoscope mucous râles may be heard in the trachea, but the swelling of the mucous membrane and the mucous accumulation are not generally sufficient to cause much dyspnœa. The patient requires treatment similar to that used in bronchitis—warm temperature and avoidance of exposure; troublesome cough may be relieved by insufflations of morphia ($\frac{1}{16}$ to $\frac{1}{8}$ grain); and expectorants, such as squill and ipecacuanha, steam or benzoin inhalations, and the application of mustard to the upper part of the sternum, are of service.

The trachea is attacked by *diphtheria*, spreading from the larynx. Croup, which is no other than laryngeal diphtheria, was at one time supposed to be mainly a tracheitis.

*Tubercle* of the trachea occurs occasionally in association with tubercle of the larynx; ulceration follows the deposit of tubercle in the mucous membrane or submucous tissue. The ulcers are more common on the posterior wall, and usually measure from 2 to 4 mm., but may reach 10 mm. in diameter. The symptoms due to tracheal tubercle are generally masked by those to which simultaneous disease of the larynx or lung gives rise.

*Syphilis*, in its secondary and tertiary stages, also affects the trachea, producing in different cases congestion, condylomas (rarely), and superficial ulcers. The most important change, however, is stricture. The trachea is affected most often at its lower end, less commonly at its upper end; and the stricture may consist simply of a narrowing at one spot, or a considerable length of the trachea may be reduced in calibre. The mucous membrane is raised into bands and ridges, which have been regarded as cicatrices of former ulcers, possibly preceded by gummas; but German pathologists look upon the thickening as a direct result of the syphilis, and the ulceration as secondary. In later stages the cartilaginous rings have been exposed, necrosed, and expectorated or absorbed. The stricture can be sometimes seen below the glottis by means of the laryngoscope. For the symptoms, diagnosis, and treatment of stricture, *see* below under Tracheal Obstruction.

## NEW GROWTHS IN THE TRACHEA

The trachea is remarkably little subject to primary new growths, whether benign or malignant. When present they give rise to dyspnœa, and may be recognised by the use of the laryngoscope or bronchoscope. More frequently carcinoma of the œsophagus or of the mediastinum grows into the adjacent trachea, narrowing its channel and producing symptoms of stricture. When it spreads from the œsophagus, it is preceded by dysphagia; but tracheal symptoms may be the first indication of carcinoma of the mediastinum. Another way in which tumours affect the trachea is by simply compressing it from outside.

As the chief symptoms in all these cases depend on the reduction of the calibre of the trachea, and as this may be due to other causes besides such tumours, it will be well to consider separately the pathology and clinical features of tracheal obstruction.

## TRACHEAL OBSTRUCTION

The causes may be grouped under three heads: (1) compression from without; (2) changes in the walls of the trachea itself (stricture); (3) foreign bodies within it.

*Compression of the Trachea.*—The most common causes are mediastinal new growths, aneurysm of the aorta or large vessels, enlargement of the thyroid body, and malignant tumours in the neck. Carcinoma of the œsophagus may also compress the trachea, but soon invades it, so that perforation takes place between the two tubes. Occasionally in children caseation and suppuration of the bronchial glands may lead to their enlargement, by which the trachea is compressed; and if the abscess bursts into the trachea, pus or portions of caseous glands may be expectorated. A mediastinal abscess arising in any other way (caries of the spine, localised empyema), the dilated left auricle in cases of mitral stenosis, and in children an enlarged thymus, are occasional causes of tracheal compression.

*Stricture.*—The chief cause is syphilis, which has been already considered.

*Foreign bodies* are rarely retained in the trachea, but commonly fall into one or other bronchus, though they may be driven up and down the trachea by the respiratory currents.

**Symptoms.**—The most important are dyspnœa and stridulous breathing; they are often accompanied by cough and the expectoration of thin frothy mucus. The voice is unaffected, or it is feeble because the obstruction weakens the current of expired air. The chest is resonant, but vesicular murmur is faint, or drowned by the noise of the stridor. Other symptoms accompanying tracheal stenosis are due to the lesion which causes it, and these may be at first entirely absent in a case of aortic aneurysm or deeply seated mediastinal tumour.

When the tracheal stricture or compression has reached a certain limit, the patient becomes liable to sudden attacks of aggravated dyspnœa with cyanosis. From a few of these paroxysms he may recover, but in the third or fourth or a later one he will probably die.

**Diagnosis.**—This has to be made (1) between obstruction in the trachea and obstruction in the larynx; (2) between the different causes of tracheal obstruction.

The laryngoscope will show at once the absence of laryngeal disease; the presence of tracheal stricture, or of tumour or aneurysm compressing the trachea, may also be demonstrated by the laryngoscope, or, failing that, by the bronchoscope. This point should be determined, if possible, before the occurrence of the paroxysms above mentioned, in which the use of these instruments may be difficult, and which, moreover, may wrongly suggest laryngeal spasm and lead to a hasty and useless tracheotomy. There are some differences in the effects of laryngeal and tracheal obstruction. One is the fact, noticed by Gerhardt, that in laryngeal obstruction the larynx is moved extensively up and down in the neck during respiratory movements, whereas in tracheal obstruction it moves but slightly. In laryngeal obstruction the head is thrown back; in tracheal obstruction it is often bent forward. If the laryngeal obstruction is due to abductor paralysis, the stridor is chiefly inspiratory, whereas in tracheal obstruction there is generally some stridor with expiration. But in other cases of laryngeal obstruction the stridor occurs with both respiratory acts. Auscultation of the trachea is certainly deceptive, as the loudest stridor is heard over the larynx even when the stenosis is in the trachea. The point is of practical importance, because laryngeal obstruction may be relieved by tracheotomy, but tracheal obstruction rarely so; and it is desirable to spare the patient an operation of this nature when it can do no possible good. But a new growth or aneurysm in the neck or upper part of the chest may produce the two obstructions, namely, one directly, by pressure on the trachea, the other indirectly, by pressure on the recurrent laryngeal nerves so as to cause abductor paralysis.

Apart from the assistance rendered by the laryngoscope or the bronchoscope,

the recognition of the cause of tracheal obstruction must depend upon collateral symptoms. Any source of compression would probably involve other organs and thus cause dysphagia, obstruction of the veins of the head, neck, or arm, pressure on corresponding nerves, and dulness under the sternum, or at the upper part of the chest on one side. On the other hand, stricture due, as already stated, to syphilis must be free from such symptoms; but an aneurysm of the aorta may compress the trachea without at first any other symptom by which it can be recognised. For the diagnosis of mediastinal growth from aneurysm the reader is referred to Diseases of the Mediastinum. The Röntgen rays may in either case render some assistance.

**Prognosis.**—This is very unfavourable, the commoner causes being little amenable to treatment; but the rare cases of abscess compressing the trachea may recover on the bursting of the abscess.

**Treatment.**—The indications are (1) to remove the cause, if possible; (2) to open the trachea below the obstruction where this is in the upper part; and (3) to relieve symptoms and secondary results. A diseased thyroid or hypertrophied thymus may be removed, and enlarged glands or growths in the neck; and abscesses, where accessible, may possibly be opened; but such opportunities are infrequent. If an aneurysm is diagnosed, the treatment for that condition should be employed, and for obvious stricture active antisyphilitic treatment by means of mercury and potassium iodide or salvarsan, especially if a positive Wassermann reaction is obtained. The iodide may be employed in any case which does not present sufficient data for a positive diagnosis as to the cause of the obstruction. In the case of a foreign body, tracheotomy should be performed, and then efforts to dislodge it should be made by inversion of the patient or shaking, or by the use of special forceps through a bronchoscope.

# DISEASES OF THE BRONCHI

## BRONCHITIS

**Ætiology.**—Bronchitis, or inflammation of the bronchi, occurs at all ages, and is the result of infection. It may occur as a primary infection, and it is then probably one form of the "common cold" described on p. 196, and the bacteriology and mode of infection are the same. This type of bronchitis is often associated with, or followed by, inflammation of the larynx and nasal mucous membrane, or the inflammation may commence in the latter, and spread downwards to the bronchi. In fact the section on coryza should be read in connection with bronchitis. Another cause is contact of the bronchial mucous membrane with irritating vapours, or air carrying solid particles, such as dust, fog, or the air of mines and of certain manufactories. Bronchitis may also be set up by the presence of foreign bodies actually in the bronchial tubes; this is comparatively rare, but blood effused into the tubes may act in this way, and it constantly occurs as a result of the deposit of tubercle or carcinoma in the substance of the lung. Certain infectious diseases, already described, are frequently accompanied by bronchitis—namely, typhoid fever, measles, diphtheria, influenza and whooping cough; and it often occurs in Bright's disease.

Bronchitis is especially prevalent amongst infants, young children, and elderly people, whereas young adults and the middle-aged are much less subject to it. Habits of luxury, confinement to warm rooms, and undue wrapping up, render the subject liable to contract bronchitis on comparatively slight exposure; and those in weakly health, or depressed from insufficient food, exhausting occupations, or bad sanitary conditions, easily acquire it. Heart disease, impeding the circulation in the lungs, and previous attacks of bronchitis—all the more if they

have left behind them emphysema or dilated tubes—dispose to the ready occurrence of the disease. Some other conditions of ætiological importance are those which involve constant exposure to the exciting causes, such as residence in towns, in cold, damp, and changeable climates, employment in mines, in wool and steel manufactures, and other such industries. Bronchitis is much more common in winter than in summer.

**Pathology.**—The mucous membrane is the part most affected, but in severe or prolonged cases the submucosa is involved, and rarely the cartilages of the bronchial tubes, and adjacent parts of the lungs. The first effect is increased vascularity and swelling of the mucous membrane, and after a short time a free secretion from the surface takes place. It is a clear fluid containing mucus; but in it there are also leucocytes and shed epithelial cells. In later stages the secretion becomes more opaque and yellow from the presence of increasing numbers of leucocytes. The secretion may also contain cells in a state of fatty degeneration, or cells containing particles of soot or dirt derived from the inspired air.

In fatal cases the tubes are often seen to be filled with greenish pus (*purulent bronchitis*). Sometimes the smallest tubes are affected. If the superficial part of the lung be sliced off, and the exposed section be squeezed, minute drops of pus will be found to ooze freely from the cut surface. The condition is one of *capillary bronchitis*, or *bronchiolitis*. Fraenkel describes a *bronchiolitis fibrosa obliterans* occurring in workmen exposed to irritating air or dust; the bronchioles are obstructed by acute or subacute growth of connective tissue.

If, in ordinary catarrhal cases, the inflammatory process persists long enough, the fibrous coats of the bronchi become thickened, and infiltrated with leucocytes; the muscular fibres are atrophied by pressure; and the cartilages and mucous glands disappear from the same cause. Ultimately, in many cases, the bronchial tubes become dilated, and form fusiform or cylindrical wide channels, often reaching the surface of the lung (*bronchiectasis*).

As a result of bronchitis the lung itself undergoes important structural changes. Acute capillary bronchitis may lead to *lobular collapse* and *broncho-pneumonia*; chronic bronchitis is followed by *vesicular emphysema*, and sometimes by *chronic interstitial pneumonia*. The last three will be spoken of separately.

*Lobular collapse* occurs in isolated lobules when the bronchial tubes leading to them are blocked with mucus, since when a tube is plugged the retained air, being stagnant in contact with the pulmonary capillaries, undergoes absorption, just as air is absorbed which has escaped into the subcutaneous cellular tissue.

There is experimental evidence (**7**), obtained by examination of the arterial blood, that the dyspnœa present in severe cases of bronchitis is due to a retention of $CO_2$ and deficiency of oxygen, probably brought about by bronchial spasm.

**Prevention.**—This depends on avoiding sources of infection and the causes predisposing to infection in individual cases. Susceptible people must avoid others suffering from catarrh. If contact is necessary gauze masks may be worn as described for influenza. Various measures described under the prevention of coryza (*see* p. 197) may be used in suitable cases. Stock vaccines or autogenous vaccines are often useful. The latter may be prepared from the sputum, but cultures should also be taken from the nose, where the organism chiefly responsible may often be obtained in pure culture. The English winter climate with its cold and damp is a very common predisposing cause of bronchitis, but residence in places in the south of England such as Bournemouth, Ventnor, Torquay or Penzance may produce favourable results. There are still more favourable climates abroad, and Mentone, San Remo, Cannes, Arcachon, Canary Islands, Madeira, and the Nile (Assouan) are the places most frequented by patients.

## ACUTE BRONCHITIS

**Symptoms.**—Acute bronchitis begins with some malaise, and a sensation of tightness of the chest; and cough soon occurs. In mild cases the general disturbance may be but slight, and the illness is confined to cough, expectoration of mucus or muco-pus, with very little, if any, dyspnœa. But in severe cases there is slight fever, the temperature rising to 100° or 101°, the appetite failing, the tongue furred, the bowels inactive, and the urine scanty. The cough is at first hard and dry, and is often attended with pain behind the sternum and in the muscles of forced expiration from the strain put upon them. The expectoration is then but scanty, consisting of thin, frothy mucus, with possibly an occasional streak of blood. After a few days the cough becomes easier and looser, and the expectoration is more abundant, more opaque, and yellow and green, from the addition of increasing quantities of leucocytes. In slighter cases the expectoration is generally more in the morning, from the accumulation during sleep, and in towns this sputum is frequently black with pigment derived from the atmosphere. Dyspnœa is often considerable, with marked prolongation of expiration, as there is often spasm of the bronchial muscles; the patient has to sit upright in bed (orthopnœa), and all the respiratory muscles are called into play. After a time the secretion of muco-pus becomes less, the cough is less frequent, and the symptoms gradually subside.

**Physical Signs.**—On *inspection* of the chest of one suffering from acute bronchitis the breathing is seen to be quickened, the chest is symmetrical, and generally in a state of moderate over-distension. The accessory muscles of respiration are seen to be in strong action, and expiration is prolonged. *Percussion*, as a rule, yields a normally resonant note, but there is occasionally slight hyper-resonance from temporary over-distension of the air vesicles; and rarely there is a little impairment of resonance at the base from accumulated secretion or from collapse. *Auscultation* shows that both inspiration and expiration are accompanied by *sibilant* or *sonorous rhonchi*, depending on the size of the tubes affected, or various kinds of *râle*, or both combined (*see* p. 194). The coarser rhonchi are often felt by the hand placed upon the chest, and may be even heard by the patient himself, or those standing near him. The larger or coarser râles are heard with both expiration and inspiration, the finest râles only with inspiration. These sounds are not equally present in all cases or in all stages of the disease. In many cases rhonchi alone are present. In severe cases the sounds are heard, variously mixed, over the whole chest, and may entirely drown the vesicular murmur.

When the smallest bronchial tubes are filled with purulent secretion in the form above described as a capillary bronchitis, the position is one of extreme danger, especially in quite young children. There is severe dyspnœa, great cyanosis or lividity of the face and extremities, and rapid exhaustion. The temperature is little, if at all, raised. The cough is at first frequent, with abundant expectoration of viscid glairy mucus, or muco-pus, or pus. The dyspnœa and lividity are the result of the imperfect aeration which the blood undergoes when the minute tubes are blocked with secretion; and this obstruction is shown by the retraction of the supra-clavicular, supra-sternal, and lower intercostal spaces with each inspiration. The chest is often resonant above, but the percussion note is impaired at the bases. Auscultation reveals small dull or crackling râles over the bases, back and front, almost or entirely masking the vesicular murmur. In later stages the patient becomes more livid and drowsy; he gradually assumes the recumbent position, generally on one side; the pulse is feebler and quicker; inspiratory efforts are less effectual; and the intercostal spaces are more sucked in. Expectoration gradually diminishes; and before death the disturbance of the cerebral circulation is shown in coma, often with a slight amount of delirium. Similar cases were seen during the War in an epidemic form among soldiers.

Clinically Fraenkel's fibrous bronchiolitis closely resembles miliary tuberculosis.

**Diagnosis.**—The diagnosis of bronchitis itself presents few difficulties, owing to the presence of rhonchi or râles. The dyspnœa and physical signs in *asthma* are like those of a very acute bronchitis, but the history of its occurrence and of former attacks will help to distinguish it. In the capillary bronchitis rhonchi are generally quite absent, and these cases are recognised by the lividity, drowsiness, and presence of râles. Rarely *obstruction* of one *bronchus* (*see* p. 230) may cause a stridor which is mistaken for bronchitic rhonchus.

It remains to determine whether an acute bronchitis is a primary infection or secondary to such disorders as whooping cough, measles, typhoid, or other specific fever. In children acute miliary tuberculosis must be thought of.

**Prognosis.**—The duration of an acute bronchitis is from a few days to three weeks or more. In fatal cases of purulent or capillary bronchitis it is from nine to twelve days. Bronchitis complicating other diseases, such as cardiac lesions, the exanthemata, Bright's disease, or typhoid fever presents essentially the same features, and, as it varies in all cases from very mild to the most severe forms, its prognosis must be considered in the same way as in the simpler forms.

**Treatment.**—In cases of moderate severity the patient should be placed in bed in a warm room, and much relief is often given if the air of the room be kept constantly moist by the steam issuing from a "bronchitis kettle." In the first stages of dryness and stuffiness, saline remedies (potassium citrate, liquor ammonii acetatis), with small doses of ipecacuanha ($\frac{1}{2}$ to 1 grain or 5 to 7 minims of the wine), should be given. If there is much tightness of the chest, counter-irritation by means of mustard leaf, or a linseed meal poultice sprinkled with mustard, should be employed. The diet should be light and nutritious. In children, counter-irritants should be used with care, but a thin poultice surrounding the whole chest (jacket poultice) is of great service. In children also an emetic (1 drachm of vinum ipecacuanhæ, repeated in fifteen minutes if necessary) is sometimes of use to get rid of accumulated bronchial secretion, and the same much more rarely in adults. As the cough becomes looser, and the sputum more abundant, expectorants may be more freely used, such as ammonium carbonate (5 grains every four hours), vinum ipecacuanhæ (5 to 10 minims), or tinct. scillæ (15 minims), or a combination of two or more of them. When the cough is very irritating, sedatives may be employed—morphia in small doses ($\frac{1}{8}$ grain or $\frac{1}{6}$ grain), compound tincture of camphor ($\frac{1}{2}$ drachm), heroin ($\frac{1}{10}$ grain to $\frac{1}{20}$ grain), tinct. belladonnæ (10 minims), or potassium or ammonium bromide (5 grains). But they must be used with great care if there is much lividity, as they may dangerously depress the respiratory and cardiac centres under these circumstances. Cases accompanied with much spasm of the bronchial tubes may be benefited by tinct. lobeliæ (15 minims), ether (15 minims), tinct. cannabis ind. (10 minims), or potassium iodide (5 to 7 grains). Chloral in small doses (5 to 7 grains) has also been recommended. In capillary bronchitis, stimulants must be given early; and the drugs of most value are ammonium carbonate, senega (as tincture or infusion), oil of turpentine (20 minims), and lobelia. Sedatives must be given with the greatest caution or entirely avoided, for the reasons given. In all severe cases with cyanosis, oxygen inhalations may render valuable service (*see* p. 234).

## CHRONIC BRONCHITIS

This occurs as a sequel of the acute form, or begins in exposure or chill in those who have had a previous attack. It is common in elderly people, often appearing in successive winters and subsiding with the approach of summer. After a time it may become continuous throughout the year.

**Symptoms.**—The main features of chronic bronchitis are not essentially

different from those of the acute form: cough, dyspnœa due to spasm in the smaller tubes, expectoration of mucus, muco-pus, or pus, with the rhonchi and râles already described. But there is an absence of fever and the constitutional disturbance which occur in acute attacks, and after long continuance secondary results of a permanent kind are produced. In the lung itself, emphysema and dilatation of the tubes (*bronchiectasis*) take place, and these will be described later. But the effects are felt beyond the lung; the emphysema (*q.v.*) impedes the pulmonary circulation, and the right heart becomes hypertrophied; it may eventually dilate, and thereupon the general venous system suffers, so that œdema of the lower extremities, congestion of the liver, ascites, and albuminuria occur. Under such circumstances tricuspid regurgitation often takes place, with its characteristic murmur (*see* Diseases of the Heart). Long-continued and severe chronic bronchitis has a serious effect upon the strength of the patient. In consequence of disturbed sleep, abundant expectoration, and impaired digestion, nutrition fails, and there may be considerable emaciation. In some cases also, in this late stage, febrile reaction of a hectic type may set in. The varieties of chronic bronchitis commonly described are the following:

1. By far the larger number of cases come under the head of ordinary *winter cough*, occurring as above mentioned. The cough is variable, sometimes coming on in paroxysms, generally worse at night; and in the morning also there is often severe coughing to expel the secretions which have accumulated during the period of rest. According to the severity or extent of the affection, the expectoration may be slight in amount, thin, mucous, and frothy, and containing black pigment in the morning; or it may be yellow or yellowish green and muco-purulent, with very little air; or it may be entirely airless, liquid, green pus. The sputa in this case generally run together in the vessel, and do not exhibit the *nummular* character common in phthisis. Microscopically, besides the abundant pus cells, there are epithelial cells containing fat, and non-pathogenic micro-organisms. Blood is occasionally present in the expectoration, usually in the form of streaks, but rarely in masses, or in any considerable quantity.

2. *Dry bronchitis*, or *dry catarrh*, is a form of chronic bronchitis in which there is very little secretion. The cough is frequent, violent, and prolonged, so that extreme congestion of the face occurs; but there is either no sputum at all, or only a small quantity of tough mucus. There is much soreness of the chest and considerable dyspnœa from spasm of the smaller tubes. Thus the bronchitis has caused a secondary asthmatic condition.

3. In *bronchorrhœa* the expectoration is exceptionally abundant, and either thin, clear, and transparent, or thick and ropy; it usually contains but little air. As much as four or five pints may be expectorated in twenty-four hours, large quantities being brought up at a time with comparatively trifling effort.

4. *Putrid* or *fœtid bronchitis* is characterised by very offensive sputum, which is not connected with gangrene of the lung, but mostly occurs in cases where the tubes are dilated. It will be described more fully below.

5. Chronic bronchitis associated with *polycythæmia rubra* (Ayerza's Disease) is the result of long-continued oxygen deficiency, and this leads to increase in the hæmoglobin of the blood, the number of red cells may approximate to 10,000,000 per cubic millimetre, and there may be clubbing of the fingers. The patient is very cyanosed, and in one case the arterial blood was found to be only 79 per cent. saturated with oxygen, instead of the normal 95 per cent. However, the actual amount of oxygen carried by the blood was about normal, owing to the great increase in the hæmoglobin and red cells. The arterial $CO_2$ pressure was also increased, and the patient was very breathless.

**Physical Signs.**—The auscultatory signs are similar to those of acute bronchitis, but as chronic bronchitis of long duration is commonly accompanied by emphysema, the signs of this disease may be also present (*see* p. 231). In cases with much accumulation of secretion the bases are dull; in such cases

also there are abundant crepitations at the bases; but generally the physical signs consist of sonorous or sibilant rhonchi, more prolonged during expiration than inspiration; and of these the coarser and louder may be felt by the hand placed on the surface.

**Diagnosis.**—This is generally simple, the history, with the symptoms and physical signs, being sufficiently characteristic. There is occasionally a difficulty in excluding chronic phthisis, for phthisis is often accompanied by bronchitis. Here the occurrence of febrile reaction, of hæmoptysis, of rapid wasting, and the greater intensity of the physical signs on one side or at one apex, would be in favour of phthisis, and confirmation may be obtained by examination of the sputum for bacilli, by the use of X-rays, or of tuberculin (*see* Phthisis).

**Prognosis.**—Though chronic bronchitis frequently shortens life, many people live to an advanced age in spite of it. It is mostly affected by the season in a marked manner, and patients are often practically well during the summer, and again get ill in the winter; but they are worse with each succeeding winter, and finally may be carried off during an exceptionally severe season, or during the cold fogs of towns, or during east and north-east winds elsewhere. On the other hand, if they can be protected from this unfavourable weather by confinement to the house, or better by residence in a warmer climate, they may keep their bronchitis within limits, and postpone the fatal termination for years. Its ill effects will, however, vary with the amount of secretion and with the rapidity with which the secondary results—emphysema, dilated tubes, and dilatation of the right heart—are developed.

**Treatment.**—In chronic bronchitis, as already indicated, the patient must be carefully guarded from exposure, and kept as far as possible in a uniform temperature of 60° or 65° F.; a moderately nutritious diet should be allowed. The medical treatment consists mainly in the use of different kinds of expectorants. Ammonium carbonate, squill, ipecacuanha, and senega may be given in ordinary cases, but particular indications have to be followed in some instances. Ipecacuanha, potassium iodide, and apomorphine hydrochloride ($\frac{1}{10}$ grain) are of use in cases with hard dry cough, potassium or ammonium bromide where the cough is very irritating. In such cases, opium or morphia may have to be used, and it often gives much relief; but it must be used in small doses at all times, and withheld in cases where cyanosis is at all advanced. Where the expectoration is abundant, the balsams of Peru (20 minims suspended with $1\frac{1}{2}$ drachms of honey) and tolu should be given, or ammonium chloride (5 to 20 grains). In cases where spasm of the tubes is liable to occur, lobelia, Indian hemp, sulphuric ether, spiritus chloroformi and stramonium are of value, and potassium iodide has been recommended for the same purpose. Turpentine, terebene, and copaiba are often beneficial in cases with free secretion. Good may be derived from inhalations of the vapour of ammonium chloride, or of steam, or from the use of sprays charged with succus conii, tr. benzoin co., and iodine or tar in the case of profuse expectoration. Benefit is also obtained from applications to the chest: linseed meal poultices may be employed more or less continuously, and mustard plasters, turpentine stupes, and tincture or liniment of iodine, at longer or shorter intervals. Dry-cupping may also be of value. If the bronchitis can be referred to any constitutional disease, this should, of course, at the same time, be treated—for instance, gout by the exhibition of alkalies and colchicum. Many cases require tonics, such as quinine and cod-liver oil. It is desirable to see that the bowels are freely opened; and in cases of long standing, where the right side of the heart is dilated, the various secretions should be kept free, and the heart's action assisted by digitalis or strychnine, as under corresponding conditions in valvular disease. Where there is dyspnœa, relief may be obtained for the time being by oxygen inhalations (*see* p. 234).

### FŒTID BRONCHITIS

The characteristic feature of fœtid bronchitis is the odour of the sputum. A very offensive sputum is most commonly a result of bronchiectasis, when the secretions accumulate in the dilated bronchial tubes, and undergo decomposition. But it may arise under other conditions—for instance, in acute bronchitis—and the cause is probably infection by organisms, having special biochemical characteristics.

**Symptoms.**—The sputum is abundant and rather thin, and in the sputum vessel it often separates into three layers, of which the uppermost is frothy with mucus, the middle a thin sero-mucous fluid, and the lowest a thick layer of pus containing the bodies known as Dittrich's or Traube's plugs. These are whitish grey or dirty greyish yellow, varying in size from a millet seed to a bean. Under the microscope they are seen to consist of pus corpuscles, detritus, bacteria, bundles of fine acicular crystals of palmitic and stearic acids, and twisted threads of leptothrix. The chemical contents of the sputum are acetic, butyric, and valerianic acids, leucin, tyrosin, sulphuretted hydrogen, and methylamine. The odour is very offensive, putrid, and at the same time somewhat sweet; not only the sputum, but the breath of the patient, is charged with it, so that none can stay near him, and it pervades the atmosphere of the room. The onset of the putrid change may be accompanied by febrile reaction, prostration, and loss of appetite; and the septic condition may extend to the pulmonary tissue, so as to cause broncho-pneumonia or gangrene. Fœtid bronchitis may become chronic, with only occasional attacks of pyrexia, with loss of appetite, nausea, and indigestion, but otherwise no serious impairment of health. Painful swellings of the joints have also been recorded. Some patients recover completely.

**Pathology.**—In fatal cases one generally finds dilatation of the bronchial tubes of old date, with intense injection and ulceration of the mucous membrane.

**Diagnosis.**—The distinction from gangrene is difficult, and all the more so as the two may co-exist. The physical signs of fœtid bronchitis are mostly those of bronchitis with dilated tubes—namely, fine or medium râles; while in gangrene one gets more often signs of consolidation or excavation. The presence of lung tissue in the sputum would be in favour of gangrene, but it is not constantly found in that condition. Very offensive sputum may also proceed from an empyema opening into the lung, and occasionally from an old tuberculous cavity.

**Treatment.**—This must be of a supporting and stimulating kind, while locally we endeavour to lessen the decomposition and diminish the fœtor by antiseptic inhalations. Carbolic acid, creosote, turpentine, thymol, eucalyptol, and tincture of iodine may be used, either inhaled from the surface of hot water, or administered more continuously by means of a respirator.

### PLASTIC BRONCHITIS

*(Fibrinous Bronchitis, Croupous Bronchitis)*

This affection is characterised by the expectoration of casts of the bronchial tubes. The sputum is generally in the form of a rounded mass, covered with mucus or blood, and, when frayed out in water, one sees a more or less perfect branching cast of a portion of the bronchial tube system. The cast is not generally thicker than a goose quill, and varies from 1½ to 2½ inches in length, and only rarely reaches 4 or 5, or even 7, inches. It has a grey or whitish yellow colour, and consists of concentric laminæ, which do not usually fill up the lumen of the tube, so that the casts are not solid, except those from the smallest tubes. Under the microscope the cast has a fibrillated structure, with numerous

embedded leucocytes, streptococci and staphylococci, hæmatoidin crystals, Curschmann's spirals, and Charcot-Leyden crystals (*see* p. 227).

**Ætiology.**—The disease is extremely rare; it occurs in males more frequently than in females, and mostly begins between the ages of ten and forty; it has also been noticed to occur in different members of the same family.

The **Symptoms** preceding the expectoration of the casts may be very slight; indeed, the patient may appear to be in ordinary health; or there may be indications of bronchial catarrh, or a pyrexial condition, with rigors suggestive of pneumonia. Then the patient is seized with violent attacks of coughing, often suffocative in character, with more or less pain or oppression at the chest, and attended at first with no sputum, unless perhaps a little mucus. After a time—it may be a few hours, or as long as two or three days—a bronchial cast is brought up. Relief is generally at once afforded: the cough subsides or disappears. But it commonly recurs in a few hours, and casts may continue to be expectorated, at intervals of a day or so, for several days, when the patient gradually gets quite well. Hæmoptysis occurs in some cases, usually after the expulsion of the cast. The physical signs are attributable to the obstruction of the tube or tubes. The vesicular murmur is commonly deficient; and there may be either slight increase of resonance over the area of lung corresponding to the obstructed tube, or, on the other hand, dulness from collapse of the same portion. The movements of one side of the chest may be impeded if tubes are extensively blocked; and râles, clicks, or flapping sounds are sometimes heard as the casts are becoming loosened.

**Prognosis.**—The disease is rarely fatal, except from complications; but it recurs at irregular intervals over a period of several years.

The **Treatment** hitherto tried has been unsatisfactory. Iodide of potassium in full doses is credited with some success; mercurials, tartar emetic, alkalies, and their carbonates, have been at different times used. Inhalations of steam and tar vapour, and sprays of lime water and of alkaline carbonates, are also recommended; and an emetic is said sometimes to be of value.

## BRONCHIECTASIS

**Ætiology.**—Bronchiectasis, or dilatation of the bronchi, may occur in connection with many diseases of the lungs. It is often associated with some fibrosis of the surrounding lung tissue (*fibroid lung*), or with *emphysema*.

Any gradually increasing and persistent obstruction of a large bronchial tube is likely to be followed by dilatation of the smaller bronchi proceeding from it. Thus aneurysms pressing on a bronchus, carcinoma pressing on or growing into it, and syphilitic stenosis are frequent causes of bronchiectasis. A foreign body impacted in a bronchus almost inevitably leads to bronchiectasis.

Primary inflammatory conditions are usually responsible for bronchiectasis, such as bronchitis, acute broncho-pneumonia, especially in children, and occasionally lobar pneumonia. Pressure on the lung from the outside by pleural effusions or neglected empyemas may also cause bronchiectasis, associated with marked fibrosis of the lung.

Rarely the condition is congenital, and it is not infrequently seen in quite young persons, when the cause may be unknown or forgotten.

**Pathology.**—In cases of partial obstruction, the tubes beyond become distended, because the inspiratory movements are more effective than the expiratory ones, so that air is sucked in and does not completely escape again. In chronic bronchitis the secretion in the tubes may cause obstruction, and at the same time the walls of the tubes are softened by the inflammation and so readily dilate. Where the lung is fibrosed this will help to produce dilatation by traction on the tubes from the outside. The pressure of retained secretion

could hardly be sufficient of itself to produce much dilating effect on the tubes.

In ordinary cases the change affects the medium and smaller tubes; they are dilated into somewhat irregular cylindrical tubes (*cylindrical bronchiectasis*), and can be traced with the greatest ease nearly to the surface of the lung; they are more common in the lower lobes than in the upper. Though the process is usually a chronic one, an acute bronchiectasis of numerous smaller tubes (*bronchiolectasis*) throughout the lung is occasionally seen after an acute catarrhal bronchitis.

In other cases the dilated tubes are ovoid or globular (*saccular bronchiectasis*). In this variety a number of smooth-walled cavities, from the size of peas to marbles, or larger still, are found in the lungs: their walls are thin, and present generally no trace of the muscular tissue or cartilage of the healthy bronchi; a small bronchus may often be found opening into the cavity. Sometimes there are bands running along the walls; sometimes the surface is ulcerated. These cavities are frequently associated with the extensive fibroid changes in the lungs. They are situated much more commonly at the base and the middle of the lungs than at the apex.

Bronchiectasis is often limited to one lung, especially when due to bronchial obstruction, to a foreign body, or to acute pneumonia or pleurisy. If both lungs are affected, either the lesions are not extensive, or one lung is very much more involved than the other.

**Symptoms.**—In cases of moderate cylindrical dilatation associated with bronchitis or emphysema, the symptoms will be lost in those of the primary disease. But in larger dilatations and in the saccular variety the bronchiectasis is the prominent fact in the case, and the secretion from the cavities and the fibrosis and cavitation of large portions of the lung are productive of definite symptoms and physical signs.

The patient need not be emaciated, is generally free from fever, and may be inconvenienced by little besides dyspnœa, cough, and expectoration. When there is increased resistance to the pulmonary circulation, failure of the right heart will cause œdema of the feet, enlargement of the liver, and albuminuria.

The sputum is either purulent and airless, or muco-purulent, or fœtid, muco-purulent and frothy, like that of fœtid bronchitis. When there are one or two large saccular cavities the sputum is often expectorated in a characteristic manner. The secretion collects for some time—it may be two or three hours—in the dilated tubes without exciting cough; then either from its quantity, or because the patient moves about, turns over or sits up in bed, the secretion flows over into an adjacent healthier tube, coughing is excited, and some ounces of muco-purulent secretion are all at once expectorated. In some cases, hæmoptysis is both frequent and moderately abundant.

**Physical Signs.**—These differ according to the character and size of the dilatations, their distribution in the lung, and the amount of consolidation or fibrosis of the intervening lung. In some cases, a large portion of one base, or even the whole of one side of the chest, presents coarse, creaking, and crackling râles, obscuring the respiratory murmur, but without dulness or pronounced limitation of movement. In other cases the condition is similar, but in addition there is, at one or more spots, an area where bronchial or cavernous breathing, with bronchophony and good transmission of whispered voice, is heard. These signs indicate a cavity surrounded by condensed lung tissue.

In other cases the physical signs are present in only one region of the chest, generally either the base or the middle zone, and rarely the apex. There are dulness, bronchial or cavernous breathing, bronchophony, pectoriloquy, and some râles. The breath sounds and the râles vary with the amount of secretion in the cavity, in the same way as do those of tuberculous cavities, and the sound ascribed to *post-tussive suction* may be heard in conditions similar to those of

phthisis (*see* p. 195). In extreme cases, or when fibrosis is advanced, the condition resembles chronic pneumonia. Retraction of the chest takes place, the heart is drawn in a horizontal direction towards the diseased lung, and the opposite lung becomes compensatorily emphysematous. In late stages the right side of the heart dilates, and œdema and venous stagnation ensue.

**Diagnosis.**—The disease is readily confounded with *chronic phthisis*. The chief points of distinction are that in bronchiectasis the physical signs are not confined to, nor most marked at, the apex; the patient is not febrile, nor ill and emaciated in proportion to the extent of the local mischief—indeed, he is often for a long time well nourished—and there are no tubercle bacilli in the sputum. The pronounced cyanosis and clubbed fingers might suggest *congenital disease of the heart;* but the physical signs show that the lung, and not the heart, is primarily at fault. A basal bronchiectasis may be hard to distinguish from an *empyema* discharging into the bronchi. The history may help, and hæmoptysis is in favour of bronchiectasis. Exploration might yield pus in either case.

**Prognosis.**—As compared with phthisis, it is good; patients often live for years with but slow advance in the local conditions; but they are liable to dangerous complications, such as pneumonia, abscess, gangrene, septicæmia, and pyæmia, and the occurrence of hepatic abscess or cerebral abscess. The last is a frequent cause of death.

**Treatment.**—The patient requires support by means of tonics such as iron, quinine, and cod-liver oil, and should be placed under the best hygienic and climatic conditions, on the same principles as in the treatment of phthisis. Locally the object should be to assist the evacuation. For this purpose the patient lies in bed on the sound side and the foot of the bed is raised one foot by means of blocks. The treatment is carried out for half an hour daily at first, and the time is gradually increased to two hours a day. The buttocks may be kept in place by a wedge-shaped cushion. Inhalations of antiseptics (*see* Bronchitis) are of value; the administration of garlic internally by means of capsules containing 30 grains of chopped garlic has been tried; the daily inhalation for fifteen to sixty minutes of the vapour of creosote in a closed chamber (Chaplin), and the intra-tracheal injection of antiseptics.

Several surgical methods of treatment have been tried, including pneumotomy, with or without resection of the overlying ribs, plugging the chest between the pleura and the ribs with paraffin, artificial pneumothorax, and excision of the diseased part of the lung. Resection of ribs overlying the bronchiectatic part of the lung has been carried out, so that collapse may be allowed to take place.

## HAY FEVER; ASTHMA

*Hay fever* is a very severe catarrh, affecting chiefly the nasal mucous membrane, often the bronchial mucous membrane as well, and causing contractions of the bronchial musculature. *Asthma*, used in its broadest sense, is a term applied to a state of affairs in the smaller bronchi which hinders the exchange of air between the larger bronchi and the alveoli of the lungs. We know little for certain about the nature of the obstruction, but arguing from the analogy of the plain muscle of the intestine or bladder, we may assume that the bronchial muscle, which possibly contracts slightly during each normal respiration, may in asthma at one time be tonically contracted, and at another time undergo a series of violent rhythmic contractions and relaxations. Œdema or congestion of the mucous membrane also plays a part in the obstruction. From this definition we see that the term asthma is often used to denote a symptom, or perhaps a syndrome, rather than a disease—a syndrome in fact that may be caused by different agents.

**The Allergic State.**—Hay fever and asthma are often different manifesta-

tions of the same type of disease, which also includes angio-neurotic œdema; some types of urticaria, eczema, and acute dermatitis; certain gastro-intestinal disturbances; migraine, and a few cases of epilepsy. The members of the group have been classed together as the *toxic idiopathies*, or the *allergic state*. The patient is sensitised to a particular foreign protein, which acts as a poison to him when presented in very small amounts, so that a violent reaction is produced. This is very similar to the effect produced by the injection of serum into an individual who has been sensitised to it, and so it has been regarded as of essentially the same nature, *i.e. anaphylactic* (*see* p. 17). According to another and probably more correct view the reaction is not anaphylactic, but is of the same nature as the well-known idiosyncrasy of some people to certain drugs. For instance, 4 per cent. of asthmatics are extremely sensitive to aspirin and have been nearly killed by quite small doses, and in these people the salicylic acid from a dose of aspirin has been found to accumulate in the blood to a much greater extent than normal (8).

Individuals may vary as to the type of allergic reaction produced: some may suffer from asthmatic or hay fever attacks, others may get angio-neurotic œdema or urticaria, and others alimentary disturbances. There is a strongly inherited tendency to the toxic idiopathics, although members of an affected family may vary as to their particular idiosyncrasy and the effect it produces

*Hay fever* occurs to a preponderating extent in the months of May, June and July, because dry grass pollen, which is the toxic agent, is plentiful at this time; but attacks can be produced artificially in susceptible individuals by dried pollen at any time in the year. The susceptibility can be tested by dropping a dilute watery extract into the conjunctival sac when some degree of inflammation follows (cf. Calmette's tuberculin reaction), or by injecting it hypodermically when an urticarial wheal forms at the site of injection. It is found that hay fever patients react in varying degree to pollens of different plants; but in this country grass pollen is the only important agent, because it is very toxic: in the summer there is plenty of it, and it can be carried long distances by wind. In America the pollen of certain *Compositæ* may also cause trouble. The pollen normally produces its effect on the nasal and bronchial mucous membrane after inhalation. If experimentally swallowed by the patient, it causes indigestion and diarrhœa.

*Asthma.*—The animal asthmas form another type of toxic idiopathy. Here the patient may be sensitised to the dandruff of a horse, and gets an attack of asthma if horses are in the neighbourhood, or even if he is in the company of ostlers; in such individuals gastro-intestinal attacks have been described after eating sausages containing horse meat. "Cat" asthma is well known; and there is sensitiveness to sheep, cows, pigs, rabbits, goats, and the feathers of birds, so that an attack may start as the result of sleeping on an ordinary pillow. In Holland it has been found recently that grain infected with mites, much used for feeding animals, is a potent source of asthma. Benger's food, and lead present in a hairwash, and even the smell of paint, may bring on attacks.

Sensitiveness to foodstuffs is another toxic idiopathy, and eating the specific foodstuff may cause asthma or gastro-intestinal disturbances. Asthmatic patients can be tested by inoculation with the proteins of different foods. Positive skin reactions have been obtained with cereals, such as wheat, maize, rice, rye, barley or oats. Eggs, potatoes, casein, lobster, oyster and various kinds of fish, meat of different kinds, spinach, strawberries, apples, and other vegetables and fruit, have all produced positive reactions in different cases.

There are two methods of testing for "skin reactions" in allergic patients. (1) By scarification; the skin after cleaning is slightly scarified, and the solution of the protein applied by means of a platinum loop. If the protein is in powder form, it may be added to a drop of N/10 sodium hydrate which has been placed over the scarified area. An urticarial wheal of at least ¼ inch in diameter indicates a positive result. (2) By intradermal injection; 0·05 c.c. of the required solution

is injected into the skin by means of a very fine needle held to the surface. A small button of fluid must be seen, as nearly parallel as possible, and a control injection with distilled water should be carried out. The front of the forearm is the best place to choose. An inflammatory areola indicates a positive reaction. The intradermal reaction is much the most sensitive of the two, and positive reactions have been obtained with various protein extracts (animals hairs, foodstuffs, pollens) in a very high proportion of asthmatics, while the normal controls gave only occasionally a slight positive reaction. Now the interesting point is that 80 per cent. of asthmatics gave positive results with more than one protein, while 50 per cent. reacted to more than five. This suggests that too much stress has hitherto been laid on the specificity of the reaction to one particular protein, but that there is a group of substances to which all allergic people tend to react, and among these the most active are extracts of human dandruff, for it was found that 90 per cent. of forty-two asthmatics tested reacted to this, while normal people failed to react. This work sheds a new light on the significance of skin reactions in asthma. They are of importance in diagnosing the allergic state, but they do not indicate, except in the rare case of the animal asthmas, etc., what the nature of the poison is that produces the disease (**8**). It would be absurd to imagine that asthmatics were poisoned by their own dandruff.

In the great majority of asthma cases the poisonous substance is unknown, and for the reasons already stated the intradermal reactions give us no help. However the poison, whatever it is, appears to be air-borne. The dust from the house of an asthmatic patient often contains it. It is present in one locality more than another. Thus, in Zeeland there are villages in which 1 per cent. of the population suffers from asthma, and these individuals lose their asthma when journeying up the Rhine. Again, most asthmatics avoid attacks altogether in mountainous regions such as the Alps, though by living there for a time they lose such immunity as they have obtained lower down, and when they return home their attacks may at first be more severe than usual. The freedom from attacks in the Alps is due to the absence of the poison, and attacks may be artificially produced by making the patient inhale dust, etc. specially brought there (**9**).

When we come to consider what the nature of the allergic state is, *i.e.* what it is in the individual that makes him susceptible to certain poisons, when normal people escape, our knowledge does not carry us very far. Certain pathological changes in asthma may be briefly summarised: (1) Substances have been isolated from the blood of asthmatics which stimulate smooth muscle; (2) the uric acid in the blood is very low and there is a failure to excrete uric acid after a meal rich in purins; (3) the "hæmoclasic crisis," which occurs after ingestion of certain proteins, consists in a fall of blood pressure, a leuco penia, a change in clotting time and in the refractive index of the blood to light; (4) there is often a very intense reaction to tuberculin. Dr. Francis's experience (**10**) that certain types of asthma may be cured by stroking lightly the nasal septum in a particular position with the electric cautery, while at the same time there is a fall of 20 mm. of mercury in the systolic pressure, suggests that the vasomotor system is somehow involved, and his experience accords with the close connection that has been found experimentally in animals between the nasal mucous membrane and the bronchial muscles. Asthma has occasionally followed on the removal of nasal polypi and on other extensive operations on the nose. These are some of the factors concerned in the allergic state, which may perhaps be defined as an irritability of various nervous centres, the vagus centre being responsible for the broncho-motor spasm in asthma.

In predisposed individuals there are several factors that may actually cause an attack,—irritation of the nasal mucous membrane, constipation, uterine troubles, distension of the stomach with food, and especially various psychic conditions. Attacks may occur because the patient expects that they will. Thus a patient sensitised to roses had an attack on seeing an artificial rose which was

thought to be real. On the other hand, violent excitement may arrest an attack completely. Attacks are more liable to come on when patients are fatigued, *i.e.* towards the end of the day.

**Asthma and Infection.**—Investigation of asthma by intradermal injection has shown that a very much higher percentage of cases of idiopathic asthma are due to the allergic state than has been generally recognised up to the present time. But in the remainder allergy must probably be excluded. These may perhaps be associated with infection of the nasal mucous membrane, tonsils, teeth, and intestines, and it is possible that the patient is sensitised to the bacterial toxins and that these cause the attacks.

A very common type of asthma is definitely *secondary* to bronchitis. In the first place attacks of asthma similar to idiopathic asthma may begin after an attack of bronchitis, and especially in children, after the bronchitis of measles and whooping cough. In the second place a condition of asthma may gradually supervene when a patient suffers from recurrent attacks of bronchitis, and the asthma may only come on when the bronchitis is at its worst and clear up again when the bronchitis is better. It may be argued that in these cases the patient is sensitised to bacterial toxins; but it is even more probable that the inflammation irritates the nerve endings in the mucous membrane, so as to produce a reflex local contraction of the muscles. Asthma also occurs with phthisis.

*Breathless Attacks in Elderly Subjects.*—This condition, sometimes known as *cardiac asthma*, is described on p. 325. *Renal asthma*, or *uræmic asthma*, are merely terms sometimes applied to breathlessness in uræmia.

**Symptoms.**—In *hay fever* there are acute rhinitis and conjunctivitis with œdema of the mucous membrane, lachrymation and sneezing; in thin-skinned people there are erythema, urticaria, with severe itching. There may be asthma and constitutional effects such as lassitude, mental depression, irritability and headaches. Paroxysmal rhinorrhœa, in which the patient suddenly has a profuse watery discharge from the nose, is probably allied to hay fever.

*Asthma.*—Sometimes there are *premonitory* indications, such as a general sense of discomfort, drowsiness, gaping, itching under the chin, sneezing and coryza, or the passage of much pale limpid urine. But the attack is often quite sudden, commencing in the early morning between two and four o'clock, though the patient may have gone to bed apparently quite well. He wakes up with a sense of dyspnœa, so that he has to sit up in bed, or gets out and opens the window to let in more air. The breathing is soon so difficult that he has to call in the aid of all the accessory muscles of respiration; he grasps with his hands the sides of his bed, the arms of a chair, the mantelpiece, or the edge of a table, to give a firm support for the muscles which pass from the upper extremities to the chest. At first the chest is nearly fixed in a condition of inspiration, and there is very slight movement, and practically no breath sounds are heard on auscultation; later, when the movements are beginning to get somewhat more free, the most noticeable feature is the extraordinary length of expiration, which is accompanied with a loud wheezing, audible at a distance, while the respiration rate is slow. The chest is somewhat over-resonant; the inspiratory murmur is scarcely audible, or accompanied with a little sibilant rhonchus, while with expiration is heard the loud rhonchus just mentioned. With this the patient's distress is very great; the face gets cyanosed, the eyes are prominent, the conjunctivæ suffused, and the whole attention of the patient is absorbed in the attempt to expel the air from the chest. Usually there is no pyrexia. After a time—it may be two or three hours—he begins to cough, and expectorates some thin, transparent mucus, which may be mixed with a little blood; then the breathing becomes easier, the cyanosis is less, gradually the whole trouble subsides, and the patient falls asleep.

The sputum often contains, besides cylindrical or ciliated epithelium, bodies known as Curschmann's spirals. These are yellowish-green or grey particles,

made up of threads of mucus. Under the microscope they are seen to be spirally twisted fine or coarse fibres mixed with eosinophil leucocytes, and there is often in the middle one transparent fibre; they are probably formed in the finer bronchial tubes. Two kinds of crystals are also often found, viz. cubic crystals consisting of calcium carbonate (**11**) and elongated hexahedral crystals known as Charcot-Leyden crystals, which consist of calcium phosphate and are sometimes found in the spirals. The eosinophil leucocytes of the blood are increased in number.

Each attack of asthma may last from two or three hours to as many days; their recurrence, at longer or shorter intervals, is a good deal determined by the exciting causes—that is, a careful patient, who knows how to avoid what will bring on his attacks, may escape for long periods. The duration of the illness is also very variable. Many of those who have it in childhood recover in adult age. The attacks themselves are rarely fatal, and the occasional occurrence of not very severe attacks is not prejudicial to health; but frequent paroxysms induce emphysema of the lungs, and ultimately attendant bronchitis, so that there is constantly more or less lividity, with the round shoulders, barrel-shaped chest, and laboured respiration which are observed in the midst of the paroxysms themselves. Life is thereby shortened, and the tendency to suffer from the severer forms of bronchitis is increased.

**Diagnosis.**—The diagnosis of *hay fever* will be helped by remembering that it occurs in the first half of the summer. The diagnosis in the case of *asthma* is easy, if the history and the character of the breathing and its onset be carefully studied. Sudden attacks of dyspnœa in cardiac disease, thoracic aneurysm, and laryngeal obstruction are those which are likely to resemble asthma most closely. Hysterical attacks may also simulate it. The ophthalmic and skin reactions are widely used to determine whether the patient is sensitive to a particular foreign protein. It is very important to make this test for horse serum before injecting antitoxic serum, as many asthmatics have died from this.

**Prevention.**—In both hay fever and asthma prevention of the attacks is the main object of treatment. In *hay fever* residence in the country during the hay-time should be avoided; if the sufferer goes out, he may wear a veil over the eyes or nose. Susceptibility to hay fever is said to be diminished by the internal use of calcium lactate. Stimulation of certain spots on the turbinals by lightly touching with the electric cautery after cocainisation has been used.

Much work has been done on the lines of vaccine therapy (**12**). Alleviation of symptoms and reduction of sensitiveness may be effected in most cases by the subcutaneous injection of dilute extracts of the pollen to which the person is sensitive. An aqueous solution of 1 part in 1,000,000 has been taken as one unit, 1 part in 500,000 as two units, 1 part in 100,000 as ten units, and so on. Preferably two or three weeks before the expected attack 1 c.c. of a very weak solution is injected, and increasing doses, or rather the same dose, 1 c.c., of solutions of increasing strength, are injected every three or four days until twelve or more doses have been given. Freeman tests the sensitiveness of the individual by ascertaining the strength of solution in units which will give the ophthalmic reaction, and checks his later doses by the same test. A solution of 1 in 1,000 appears to be the maximum strength that is advisable.

As regards *asthma*, the most important thing is to keep in as perfect a state of health as possible. In particular, overwork is to be avoided. Regular exercise and adequate holidays should be taken. Some people can live in London and large cities free from paroxysms who have them at once if they attempt to live in the country. Conversely others can only live in the country, and have asthmatic attacks in town. In the same way sea air may excite attacks in some and cure others. The facts with regard to any patient can only be ascertained by experiment. Patients usually do better on high ground.

Moderation and care in diet are the next points to consider. Food should be

light and easily digestible ; a heavy supper should not be taken ; and particular food should be excluded from time to time, such as cheese, pastry, pork, beer, to see if there is any one offender in this respect. Patients may do better on a carbohydrate-free diet, especially if they are not taking much exercise. If it is found that a patient is sensitised to a particular protein, that substance should be avoided, or if this is difficult, desensitisation with the specific protein may be carried out. However, quite as good results have been obtained by means of non-specific protein therapy. Weekly subcutaneous injections of 1 c.c. of a 1 in 100,000 Koch's T.D.A. tuberculin have been used (**13**). Peptone injections in the form of a 5 per cent. solution of Armour's No. 2 peptone are injected twice weekly intramuscularly or intravenously. The first dose is 0·3 c.c. and this is gradually increased by 0·2 c.c., the maximum dose being 2 or 2·5 c.c. The maximum dose is that which just fails to give a reaction, and the temperature should be taken four or five hours after the injection to see whether it is raised (**14**). Lightly stroking the nasal septum with the cautery, as described on p. 226, is of most value when the nose is free from deformity and polypi and the systolic blood pressure is on the high side (120 to 160 mm.). Under no circumstances should polypi be removed in asthma (**10**). Liquor arsenicalis in 5 mm. doses, bromides, and particularly potassium iodide in doses up to 30 gr. a day are the most valuable drugs to prevent the onset of attacks. Mild purgation with calomel in small doses is also of value, and salol and other intestinal disinfectants have been used.

When asthma is associated with infection other additional methods are available. Coryza should be treated with the paraffin and vaseline mixture described on p. 197. Autogenous vaccines may be prepared from the sputum or nasal secretions (*see* p. 216), or a stock vaccine may be used. Septic foci should be looked for and dealt with.

**Treatment.**—For *hay fever* Morell Mackenzie recommended a spray of a 4 to 6 per cent. solution of cocaine to the eyes and nose. In the case of the nose this was followed by the daily introduction along the floor of the nose of a bougie, smeared with vaseline or oil, and left in for ten minutes at first, and for gradually increasing periods up to half an hour or longer. Adrenin hydrochloride may be sprayed into nose or throat from a solution of 1 to 5,000. Where there is chronic hypertrophic rhinitis, the application of the galvano-cautery to the swollen mucous membrane, after the preliminary use of a 2 per cent. solution of cocaine, seems to be quickly curative.

To stop an attack of *asthma* an injection of 1 to 2 minims of 1 in 1,000 adrenin hydrochloride is the best treatment, though 10 and even 15 mm. have been injected intramuscularly. Atropine $\frac{1}{100}$ to $\frac{1}{50}$ gr. may be injected. Benzyl benzoate in 5, 10, and even 20-minim doses has been given by mouth or hypodermically. It may be taken in capsules or as a spirit. Amyl nitrite is also commonly inhaled, or maximal pharmacopœial doses of Tr. lobeliæ aeth., Tr. stramonii, Tr. belladonnæ, chloral hydrate, the nitrites, or nitroglycerine may be given by mouth. A very common method of treatment is to inhale the fumes from burning a paper saturated with nitre solution and dried, or to smoke cigarettes made of chopped stramonium leaves, or to use other preparations containing stramonium. Inhalation is a bad method of regular treatment, because the bronchial mucous membrane is irritated with resulting bronchitis. Oxygen inhalations may give great relief so long as the oxygen is given in sufficient quantity (*see* p. 234).

## OBSTRUCTION OF THE LARGE BRONCHI

In their relation to the various causes of compression, the two main bronchi closely resemble the trachea, and much that has been said under the head of tracheal obstruction might be repeated here. Aneurysms and bronchial glands

enlarged by malignant growth, or by caseation and suppuration, are the chief causes of compression; less commonly epithelioma of the œsophagus, gummas, abscesses, and even a dilated left auricle may press upon the bronchus. Stricture occurs from primary carcinoma of the mucous membrane, usually columnar-celled, and from syphilitic ulceration and scarring. The special liability of the left bronchus to compression by an aneurysm of the arch of the aorta, under which it passes, is of importance. Obstruction also results from an impacted foreign body. Foreign bodies more frequently fall into the right bronchus, because it is the larger and the dividing ridge between the two bronchi is somewhat to the left of the middle line, and hence objects falling down the centre of the trachea are directed into its right branch. They may be driven into the trachea during coughing, and fall back into the same or the opposite bronchus.

The obstruction of a bronchus is followed by important changes in the corresponding portion of the lung, and the distal divisions of the bronchus. Ultimately in every case the *lung becomes collapsed*, because, when the interchange of air completely ceases, what remains is absorbed by the pulmonary vessels. In a rapidly complete obstruction, as from impaction of a foreign body, this collapse occurs very early; but when the compression takes place slowly, as in the case of an aneurysmal sac, there is at first *distension* of the lung with air, such that the heart may be pushed out of place, and the diaphragm forced downwards (Newton Pitt). A loose foreign body, acting like a ball and socket valve, produces a similar effect.

On the bronchi beyond the seat of obstruction, the certain effect of the narrowing is the occurrence of dilatation, or *bronchiectasis*, which may develop in the course of two or three months. Accompanying the bronchiectasis, there is in course of time *fibrosis* of the lung, and thickening of the adjacent pleura. Pus forms in the bronchiectatic cavities, and though expectorated from time to time, is liable to undergo putrefaction, and contributes to the occurrence of septic pneumonia, gangrene of the lung, acute pleurisy or empyema. Sir Frederick Taylor saw the left lung converted into one large sac containing pus, from the pressure of an aneurysm on the bronchus.

Foreign bodies do not confine themselves to producing mechanical obstruction, but set up inflammatory changes leading to bronchitis, which may spread to the trachea and to the bronchi in other parts of the lung. The most serious inflammation is to be expected quite early when the foreign body is a pea-nut. Complications caused by foreign bodies are abscess, bronchiectasis and bronchial stricture, and occasionally pneumothorax or pyo-pneumothorax in acute cases, and empyema or profuse hæmoptysis in long-established cases (**15**).

**Symptoms and Physical Signs.**—These vary with the degree of obstruction and rapidity of its production; and since the opposite tube is often free, and thus only half the respiratory area is interfered with, the bronchus is often much more completely obstructed, before death occurs, than ever the trachea can be.

Dyspnœa at rest or on exertion is the only constant symptom at first, and occasionally there are stridor and slight hæmoptysis; but obstruction of either main bronchus may lead to the same paroxysms of asphyxia as occur in tracheal obstruction. When bronchiectasis has developed, cough, expectoration of offensive sputum, and febrile reaction become prominent symptoms.

The chief physical sign is the absence or extreme weakness of the vesicular murmur, which is in strong contrast with the increased breath sounds on the opposite side. This, in some cases for a time, may be the only physical sign, for the resonance may be perfectly normal. But in the cases in which distension of the lung takes place there will be hyper-resonance on percussion, with extension of resonance over the cardiac area and evidence of displacement of the heart, so that the resemblance to pneumothorax may be very close. In these cases eventually, and in other cases much sooner, as the air becomes absorbed, there is dulness

at the affected base, with diminished tactile vibration. This may go on to complete absence of breath sounds, voice sounds, tactile vibration, and percussion resonance. If considerable bronchiectatic cavities should be formed, the above physical signs may be, over one or other small area, replaced by tympanitic percussion note, cavernous breathing, crackling, gurgling râles, bronchophony and pectoriloquy.

**Prognosis.**—This depends on the nature of the obstruction and the degree of inflammatory reaction. The lung may become extremely tolerant to a foreign body, as is shown by the fact that cases have lasted for thirty-six years, with persistent bronchiectasis and fibrosis of the lung (**15**).

**Treatment** is similar to that of obstruction of the trachea (*see* p. 215).

**Diagnosis.**—The combination of good resonance with nearly complete absence of respiratory sounds on one side of the chest is very characteristic of obstruction of the corresponding bronchus. When the obstruction is accompanied by stridor, it may be mistaken for bronchitis. Stridor from the above cause is persistent and uniform in character, arising from a single point of obstruction, whereas the rhonchi of bronchitis vary constantly in loudness, pitch, and position.

In compression of the bronchus with a distended lung, pneumothorax has been wrongly diagnosed on account of the hyper-resonance with absence of breath sounds. In these cases a radiogram may show the presence of aneurysm in the case of compression, or the lung retracted towards the spine in the case of pneumothorax.

When, on the other hand, the stenosis causes, as eventually it must do, more or less complete collapse of the lung, the physical signs resemble those due to a partially absorbed pleuritic effusion, and the exploring syringe may be necessary for a final decision.

Where foreign bodies are in question, the history must, of course, be carefully considered. Many are opaque to the X-rays. In suitable cases the *bronchoscope* may be employed.

# DISEASES OF THE LUNGS

## EMPHYSEMA

The term *emphysema* (from ἐν, in, and φῦσα, wind) is rightly used to denote the extravasation of air into the subcutaneous or other tissues of the body (*surgical emphysema*), and into the interlobular or interstitial tissue of the lungs (*interstitial emphysema*). It is much less applicable to the disease of the lung now under consideration, for which, however, in medical parlance it is usually reserved. The alveoli of the lung naturally contain air ; in this disease they are abnormally distended, and may be said to contain too much. So far the name *emphysema* (*vesicular emphysema*) may be justifiable ; but the name *alveolar ectasis*, which has been suggested, is more correct.

**Ætiology and Pathology.**—Several factors are concerned in the production of emphysema. Some of these operate in chronic bronchitis, one of the commonest causes of the condition. (1) Just before the action of coughing there is a high pressure in the lungs behind a closed glottis. This interferes with the blood supply, since the pulmonary blood pressure is low, so that, in course of time, degeneration of the lung tissue takes place. (2) At the end of coughing a deep inspiration is taken, causing distension of the alveoli, stretching of their walls, and narrowing of the capillaries, with interference with the blood supply again. (3) The force exerted by the muscles during inspiration is greater than during

expiration, because the latter action is largely due to the elastic recoil of the lungs. Where the bronchi are partially obstructed with secretion, air may be drawn into the alveoli against the obstruction during inspiration, but cannot get out again during expiration, so that the alveoli are permanently distended. This is the factor that operates in asthma, the obstruction being due to contraction of the bronchial muscles. The obstruction acts as a valve, allowing air to enter, but not to come out again. (4) Prolonged stretching of the alveolar walls has been thought to occur in glass-blowers, and in players of wind instruments, and in those engaged in laborious occupations, who are continually keeping their chests expanded, either to supply a slow regulated stream of air or to serve as a *point d'appui* for the use of the arms. Recent observation makes this doubtful (**16**). (5) In the course of years the elastic tissue of the lungs gradually wears out, producing the *small-lunged emphysema* of elderly people. (6) When a portion of the lung shrinks from disease or becomes infiltrated with inflammatory products or a neoplasm, it cannot expand during inspiration. Consequently the expansion of neighbouring alveoli must be increased so as to fill up the vacant space. This is known as *compensatory emphysema*. By such various means do the septa between adjacent alveoli become atrophied in emphysema. Soon a perforation is established through the septum; then the whole septum is destroyed, and the two alveoli become one. In this process not only the elastic tissue, but also the whole network of pulmonary capillaries contained in the septum, disappear. If this is repeated extensively throughout the lungs, first, all the air spaces are much enlarged, and in many places great blebs of lung tissue simply containing air are formed; secondly, the elasticity of the lung necessary for expiration is reduced much below the normal; thirdly, the vascular area available for aerating the blood is greatly diminished; and fourthly, in most cases the lungs themselves are considerably enlarged.

In consequence of the loss of elasticity, expiration becomes more difficult. Parallel with the increase in size of the lungs, the chest enlarges in width and depth, assuming permanently the shape and position which are characteristic of full inspiration; the mobility of the chest is much diminished, since it ranges only between different degrees of inspiration, instead of between full inspiration and full expiration. The interchange of gases is less complete. This is shown by the values found for the pressure of $CO_2$ in the arterial blood, which are much above the normal. There is, in fact, a $CO_2$ acidæmia, which causes severe dyspnœa. The saturation of the arterial blood with oxygen may also be below normal (**7**).

Another important factor is the *loss of capillary area* in the lungs. From this there results an obstruction to the pulmonary circulation. The tension in the pulmonary artery and right ventricle is increased, the right ventricle hypertrophies, and later there is dilatation of the right side of the heart, so that the venous system becomes engorged, producing congestion and enlargement of the liver, œdema of the feet, legs, and trunk, and albuminuria. In elderly people there is usually also left-sided dilatation of the heart with myocardial degeneration.

**Morbid Anatomy.**—A lung affected with emphysema does not collapse when the chest is opened at the post-mortem examination, but even bulges out through the aperture. It is soft and inelastic, and yields to the pressure of the finger (" pitting "). In different parts of it, especially along the inner and lower edges, may be seen large blebs the size of peas or nuts; and the lung is unusually pale and bloodless, and of a mottled grey colour. On section the larger blebs collapse; and the whole organ is much drier than usual, except in some parts, such as the bases, which may have been the seat of a complicating bronchitis or œdema.

A variety (*small-lunged emphysema*) occurs in old people as a senile atrophic change; the lung is not enlarged, and blebs are not numerous. The septa have

atrophied so that alveoli have joined together, and the lung is shrunken, inelastic, dry, and pale, and presents a less perfectly spongy structure than normal.

The greater development of emphysema in certain parts of the lungs, specially the apices, anterior margins and lower edges, may be accounted for on Jenner's view—that when air is retained in the chest under great pressure, as when coughing or making any great muscular effort, it is the parts of the lung which are least supported by the surrounding structures which will bulge out owing to the air pressure from within.

**Symptoms and Physical Signs.**—The symptoms of emphysema are at first only shortness of breath; the cough and expectoration which are commonly present result from a co-existing bronchitis. The dyspnœa is especially seen on exertion in early stages, when the breathing is quickened and the patient readily pants; later on it may be always present, producing orthopnœa at night. In its worst forms the extraordinary muscles of respiration are in constant use; the clavicles are lifted; and the sterno-mastoids and scaleni stand out at each inspiration, striving to increase the tidal air; expiration is prolonged, laboured, and aided to their utmost by the muscles of the abdomen. The *physical signs* are characteristic. The chest is broad, deep antero-posteriorly, but short; it is often called barrel-shaped, from its enlargement, and from the increase of the antero-posterior diameter giving it rather a circular than a transversely oval shape. The shoulders are raised; the upper ribs are closer together, and the lower ribs wider apart than normal: and the epigastric angle is very obtuse, measuring 105° or more. The elevation of the ribs alters the relative positions of the nipple and the heart's impulse; the nipple is often found on the fifth rib, and the heart's impulse in the sixth space. Percussion gives excessive resonance over the parts of the chest which are normally resonant, and an extension of the resonance over areas which are normally dull. Thus the hepatic and cardiac dulnesses are encroached upon, the right lung being resonant down to the sixth space or seventh rib, and the superficial heart dulness often disappears altogether. On auscultation the breath sounds are very much diminished or scarcely audible, but the expiratory murmur may be much prolonged if there is coincident bronchial spasm, and rhonchi will be audible.

The enlargement of the lungs also affects the signs connected with other organs. Since a larger portion of lung than is normal lies between the heart and the chest wall, the impulse of the heart may be imperceptible in the fifth space, the cardiac sounds are faint, and the fact of dilatation or hypertrophy may be concealed.

In small-lunged emphysema the chest is more nearly circular in its outline, but it is not enlarged; the lungs do not cover the heart; and the heart is not hypertrophied, but atrophied. The percussion note is hyper-resonant, and the inspiratory murmur is feeble, but the expiration is not prolonged.

In both forms the rhonchi of bronchitis are frequently present; in extreme cases there are râles at the bases of the lungs, with an impaired note due to œdema.

**Complications.**—Chronic bronchitis is frequently present, with or without bronchiectasis. In elderly people there is commonly myocardial degeneration with hypertrophy and dilatation of the left side, as well as of the right side, of the heart, and there may be general œdema. There is often arterial degeneration associated with senile arteriosclerotic kidneys. This type of case is more fully considered on p. 325.

**Diagnosis.**—Its recognition depends upon the altered quality of resonance, and especially upon the extension of resonance over the præcordial area, and downwards over the liver. In the small-lunged variety the altered quality of resonance and the dyspnœa are the chief features. The Röntgen rays show a more extensive and lighter area over the lungs than in health, and a lower position and less extensive movements of the diaphragm.

**Prognosis.**—Actual recovery does not occur, only relief of symptoms. The duration of life depends upon the extent of the change, the liability to

bronchitis, and the state of the cardiac muscle. In most cases the final result does not come under several years.

**Treatment.**—This must be directed to improving the general health of the patient, to avoiding all risk of bronchitic complications, and to relieving these when they occur. Thus the patient should have nutritious and digestible diet, should be well clothed, live in warm, well-ventilated rooms, and avoid east winds and the night air. Tonics, such as cod-liver oil, iron, strychnia, and quinine, are used. Attempts have been made to compensate for the loss of elastic tissue. Thus Gerhardt advises assisting expiration by mechanical compression of the thorax; this is done by another person with the hands upon the lower part of the thorax for five or ten minutes every day.

For the accompanying bronchitis expectorants such as ammonium carbonate in doses of 5 to 7 grains, vinum ipecacuanhæ, and the infusion or tincture of senega, should be given. Mustard plasters or linseed meal poultices will afford some local relief. If the heart is failing, digitalis, strychnia, or other heart tonic must be used; and when anasarca is threatened, purgatives such as pulv. jalapæ comp. and diuretics such as squill, acetate of potassium, spirits of nitrous ether, and citrate of caffein, should be given to relieve the overloaded venous circulation.

*Administration of Oxygen.*—Inhalation of oxygen by means of Haldane's apparatus for half an hour or more at a time through the day is of great benefit to these patients. The apparatus consists of (1) a regulator which is screwed into the cylinder, so that the gas is delivered at a low pressure and constant rate; (2) a mask and valves fitted with a small bag to enable the oxygen to be used effectively. It is best to use a current of 2 to 4 litres per minute. There is sometimes a leak between the nozzle of the regulator and the head of the cylinder. This can be obviated by using a small lead washer with central hole cut from very thin lead sheeting. A new washer must be used each time.

The following method of giving oxygen, which is specially valuable in the case of children and is often preferred by adults, requires no special apparatus. The cylinder furnished with the ordinary nozzle is connected first with a Woolf's bottle containing water and then with a rubber catheter which is pushed right to the back of the nose. The connecting tubing should be wide—not much less than $\frac{1}{2}$ inch in diameter. The rate of supply of oxygen is regulated by keeping the bubbling in the bottle at a constant rate. It can be measured by filling a pint measure with water and holding it inverted in a basin of water. On placing the catheter beneath the measure the water is displaced by oxygen. It should take about twenty seconds to collect a pint in the case of an adult. The rate for children may be slower. Instead of the nose the catheter may be pushed as far back as possible through the mouth. The wasteful and ineffective way of delivering oxygen through a funnel should never be used. Oxygen has been given subcutaneously and intravenously; the former plan is useless; the latter might be used if the supply of oxygen were limited or the lungs extremely œdematous, but it is not free from danger. It has been sometimes used because the factors involved in the efficient administration of oxygen through the lungs have not been realised. The treatment of pulmonary disease by compressed air, as in the steel chamber at the Brompton Hospital, depends on the increase in the concentration of oxygen in the inspired air.

## COLLAPSE OF THE LUNGS

(*Atelectasis Pulmonum*)

A distinction is often made between lungs that have never completely expanded (atelectasis) and those that have after expansion partly returned to the fœtal state (collapse). *Atelectasis* is congenital, and is seen in very weakly children, whose respiratory movements are insufficient to draw in the required amount

of air. It occurs the more readily because the lungs after removal from the body at birth are about the same size as the chest cavity, and so there is not the same suction power, compelling the entry of air, as there is in later life, when the lungs are smaller than the chest cavity. *Collapse* is an acquired condition due to failure of air to enter the lungs, and results from (1) obstruction to the entrance of air by the air passages, (2) compression of the lung from without, and (3) immobility and retraction of the chest wall and diaphragm. If any portion of the lung is deprived of respiratory movements, the air will be absorbed by the pulmonary blood vessels, and thus collapse will be produced.

*Obstruction* may arise from chronic enlargement of the tonsils, adenoid growths in the naso-pharynx, much more often from the viscid, mucous, or purulent secretion of bronchitis, especially in children, and as a part of broncho-pneumonia, and in older people from constriction of the bronchus by neoplasm or by aneurysm or some other of the causes previously mentioned.

The causes of *compression* are numerous: in the chest itself it is most frequently due to pleural effusion, but also to enlargement of the heart, pericardial effusion, mediastinal tumours, aneurysms of the aorta, and angular curvature of the spine (kypho-scoliosis); in the abdomen, to the pressure of tumours growing from the upper surface of the liver, especially hydatids, abscess and neoplasm, of sub-diaphragmatic abscesses, hydatid of the spleen, ascitic fluid, and ovarian tumours.

Paralysis of the diaphragm occurs in diphtheria and other forms of multiple neuritis, and paralysis of the intercostal muscles in lesions of the upper dorsal portion of the spinal cord. In wounds of the chest and sometimes other parts of the body, a *massive* collapse of the whole of one lung may occur. There is no necessity for the wound to penetrate into the chest cavity, and the lung opposite to the side of the wound may be affected. The condition may or may not be associated with hæmothorax. A possible explanation is that the paralysis of the chest wall is of reflex nervous origin (Bradford). Massive collapse occurs not uncommonly after acute abdominal conditions, and may be due to the same cause (Pasteur); but another explanation is offered by Elliott and Dingley, who conclude that in cases which they have seen the bronchial tubes are blocked by inflammation, and consequently the air is absorbed from the alveoli, and so collapse occurs. The condition is not due to the general anæsthetic, as it has occurred after spinal anæsthesia.

A similar inhibition of the diaphragm may result from pericarditis.

**Morbid Appearances.**—Lung in a state of collapse or atelectasis has a violet or dark purple-grey colour, and is tough, airless, and dry on section. Isolated patches are seen to be slightly depressed below the general surface. Unless subsequently the seat of inflammation, they may be again expanded by forcible inflation with air.

**Symptoms.**—In congenital atelectasis the child is weakly, more or less livid, with rapid shallow breathing and feeble cry. With each inspiration the lower part of the chest is drawn in, and the intercostal spaces are depressed. Examination may elicit a little loss of resonance at the bases, and occasionally some râles, but feebleness of breath sounds is the chief physical sign. The collapse of bronchitis is rarely extensive enough to reveal itself by auscultation, its distribution being lobular and scattered.

When collapse is more extensive and uniform different stages can be recognised by the physical signs. A very slight degree of collapse may occur at the bases from temporary disuse of the lung as a result of early pleurisy, or from prolonged dorsal decubitus. When the affected area of lung is auscultated, the breath sound is very feeble; if the patient breathes deeply, there is a louder vesicular murmur, and at the end of it fine crepitations, which are due to the fresh expansion of hitherto collapsed air vesicles.

The physical signs of collapse due to obstruction of a bronchus have jus

been considered. In the case of compression by fluid, etc., the early signs may be dulness, diminished tactile vibration, and either diminished or faintly bronchial breath sounds. When complete they are absolute dulness, absence of breath sounds and tactile vibration.

In the massive collapse associated with retraction of the chest wall due to wounds, there is dulness, and there may be deficient breath sounds and absent tactile vibration, but in advanced cases there may be instead loud bronchial breathing and increased tactile vibration. The chest wall is retracted, the intercostal spaces depressed, the diaphragm raised and immobile, and the heart drawn over to the affected side. When a hæmothorax is present on the affected side, although the chest wall is retracted, the heart may be pushed over towards the sound side. This occurrence is strongly in favour of the primary cause being paralysis of the chest wall, because in the last case this persists even though the pressure in the chest on the same side is increased by the hæmothorax. Massive collapse after operations is commoner than is generally supposed. Probably the cases are often diagnosed as pneumonia owing to the physical signs.

The symptoms are dyspnœa, which, however, may be slight when the patient is at rest, cyanosis, and sometimes pain.

**Treatment.**—In most cases of collapse, the primary cause must be discovered and treated. In congenital atelectasis, the treatment must be supporting. The child should be kept warm in a well-ventilated apartment, the chest may be gently stimulated by friction, and proper feeding should be secured. In older children, bronchitis, rickets, or congenital syphilis must be met by appropriate treatment. Lobar collapse after abdominal operations generally recovers in a few days. Elliott and Dingley recommend that expectorant medicines with potassium iodide should be given, that all abdominal bandages should be loosened as far as possible, and that the patient should be encouraged to make full inspiratory efforts, especially of an abdominal type, for five minutes every hour.

## ŒDEMA OF THE LUNGS

This consists of the exudation of serous fluid into the interstices of the lung, and into the air vesicles and smallest bronchi.

**Ætiology.**—Some degree of œdema of the lungs is found in the majority of post-mortems, especially where the patient has been lying for some time in bed before death. For this reason it is most marked at the base and along the posterior borders of the lungs (hypostatic œdema). Apart from this, certain diseases are particularly likely to cause it; these are myocardial disease, valvular disease of the heart, especially aortic regurgitation; and acute and chronic Bright's disease, where it may come on acutely owing to rapid failure of the left ventricle, while the right ventricle goes on pumping blood into the lungs; and an inflammatory œdema generally accompanies acute pneumonic processes. Local œdema may result from the pressure of tumours or of aneurysm on the pulmonary vessels; in diseases involving the lung, like pneumonia or pleuritic effusion, œdema of the formerly healthy lung contributes to the fatal termination. Suffocative poison gases produce œdema of the lungs.

**Morbid Anatomy.**—A lung affected with œdema is bulky, heavy, and exudes when incised an immense quantity of serous slightly blood-stained frothy fluid.

**Symptoms.**—The chest may be at first resonant, but later shows some impairment of the note at the bases behind; here the breath sounds are deficient, and there are heard only abundant fine and medium râles. In the acute form (*acute suffocative œdema*) the patient is taken suddenly with restlessness, dyspnœa, orthopnœa, sense of suffocation, more or less cyanosis, small rapid pulse, and expectoration of large quantities of colourless or blood-stained, frothy, watery

fluid. This may be quickly fatal, or subside in the course of a few hours. There may, however, be no expectoration until some days have elapsed, and, indeed, none at all, in some quickly fatal cases. In the final œdema of pneumonia, the râles are audible over the whole of the hitherto healthy lung.

**Diagnosis.**—Acute pulmonary œdema coming on suddenly in a person, who has been carrying on his usual occupation, must be distinguished from angina pectoris, in which the patient is not restless and there is not much sputum, and from pulmonary embolism, in which the expectoration occurs later and is more sanguineous.

**Treatment.**—This is chiefly to be directed to the primary cause. For the lung condition oxygen inhalations are valuable. Ammonium carbonate may be given as an expectorant. Digitalis is often useful for the heart, and atropine for checking the pulmonary secretion. In very acute cases venesection may be tried.

## PNEUMONIA

Inflammation of the substance of the lung, as opposed to the bronchial tubes, is called pneumonia. As an *acute* disease it leads to consolidation by exudation into the air vesicles of inflammatory products, which are usually absorbed in the course of recovery. In a *chronic* form it causes a dense fibrous transformation of the interstitial tissue, which is permanent. Of acute pneumonia two typical forms can be distinguished from one another by the following features: *Lobar pneumonia* occurs at all ages, but more often in adults, affects large portions of the lung at the same time, and has all the characteristics of a specific infectious disease, with a limited duration, a quick recovery, and sometimes epidemic prevalence. *Broncho-pneumonia* affects chiefly infants, children, and elderly persons, invades several small areas of the lung, and is much less definite in its course and modes of onset and termination. The two forms differ also in their histology and bacteriology. These distinctions are, however, by no means absolute, and some of the difficulties of recognition and distinction will be pointed out after the two have been separately described.

### LOBAR PNEUMONIA

(*Croupous Pneumonia*)

**Ætiology.**—The disease occurs in both sexes, but it is twice as common in males as it is in females, the difference between the two sexes being least marked in the very young, and in old people. It is seen also at all periods of life from infancy to old age, but it is more frequent in adults up to middle age. It occurs much more often in the winter and spring than in the summer and autumn, when the temperature is undergoing sudden changes, when the winds are east or north-east, or when the weather is wet or cold. Habits and occupations which involve exposure dispose to pneumonia, and it is probable that persons of weak health or suffering from mental depression, or those who do not have sufficient nourishment, are more liable to it than the robust and strong. Intemperate habits also dispose to it, and greatly increase its mortality. One attack does not exempt from another; indeed, pneumonia is said to have occurred as many as fifteen or twenty times in the same patient, but more than two attacks are not very common.

Cold or chill often seems to be a determining event, but can act only by favouring the invasion of the micro-organism which is the direct cause. Insanitary surroundings are also sometimes influential in the same way. Cases of direct contagion appear to be undoubted; and many instances are recorded in which pneumonia has spread rapidly through villages, large buildings, or households, precisely like an epidemic fever. Lobar pneumonia also occurs as a complication

or sequela of some other diseases, and especially of mitral disease, acute nephritis, diabetes and some infectious disorders, including influenza ; but it is rare as a complication of tuberculosis. Traumatic pneumonia sometimes occurs from a blow on the chest, if the injured lung becomes secondarily infected.

Pneumonia is, indeed, a specific infectious disease, with the primary seat of infection in the lung ; and the infecting organism is in very many cases the *Diplococcus pneumoniæ* of Fränkel, *Diplococcus lanceolatus*, or *pneumococcus*, which is found in the lungs and sputum, and in severe cases in the blood, thus constituting a *pneumococcal septicæmia*. The four types of the pneumococcus have already been described (*see* p. 72). But lobar pneumonia may also, more rarely, be caused by other organisms, for instance Friedländer's *Bacillus pneumoniæ*, *B. influenzæ*, and *Streptococcus pyogenes*.

**Morbid Anatomy.**—In pneumococcal pneumonia, the part of the lung affected is converted from a spongy structure into a solid mass. In the earliest, or first, stage of *congestion* or *engorgement*, the lung is heavy, reddish brown in colour, exudes a frothy, reddish serum on pressure, and breaks down more readily than in health. The capillaries are dilated and tortuous from distension with blood, and minute hæmorrhages may be present. In the second stage—called *red hepatisation*, from the resemblance which the consolidated lung bears to the liver—the organ is of a dull red colour, finely granular on section, completely airless, solid, sinking in water, but breaking down readily under the pressure of the finger. The contents of the alveoli, which may be detached in fine granular masses, are seen to consist of fibrin, containing some red blood corpuscles and leucocytes. What has actually happened is that the walls of the alveoli have been so distended with fluid exuded from the capillaries that the endothelial cells have given way, and the air spaces have become flooded with fluid that soon coagulates (**29**). The third stage, *grey hepatisation*, is also characterised by its solidity, but the colour is greyish yellow or simply grey, and the surface is less granular than that of the red stage. Microscopically it differs from the latter in that the air cells and alveolar walls are crowded with leucocytes, while fibrinous exudation and red corpuscles are in very small quantity. The change of colour is attributable to the leucocytes in the alveoli. A fourth stage, that of *purulent infiltration*, is also described ; but this is only an extreme condition of grey hepatisation. The lung is softer, yellowish in colour, and yields to scraping or pressure a quantity of yellow purulent fluid, which is provided by the disintegration of the infiltration filling the air cells, the leucocytes becoming fatty and granular. A true *abscess*, however, is exceedingly rare as the result of typical acute pneumonia. It is doubtful whether the stage of purulent infiltration is ever reached in cases that recover ; it is true, recovery, or *resolution*, is sometimes accompanied by physical signs (*redux crepitation*) which indicate that the exudation is softening into fluid. But many patients get well without such evidence, and with so little expectoration that the removal of the exudation can only be explained by its absorption directly by the lymphatics ; in but few cases the amount of sputum is very considerable.

The inflammation of the substance of the lung is accompanied, in a large proportion of cases, by inflammation of the pleura ; this often causes pain at the commencement of an attack, may be recognised by friction sound, and may not be again evident during the course of the disease. Pleuritic lymph may be discovered after death of which there was no evidence during life, and serum or pus may be formed in considerable quantity. The double lesion may be spoken of as *pleuro-pneumonia*, but the name is not generally used except for cases in which the pleurisy is clinically a prominent feature.

**Localisation.**—Pneumococcal pneumonia is nearly always partial, affecting the base more often than the apex, and the right lung somewhat more often than the left. Beginning at the base of one lung behind, it extends upwards to the apex, as well as forwards, or it may extend downwards from the apex, or com-

mence in the centre and spread upwards and downwards. Its progress appears sometimes to be stayed, or checked for a time, at the line of the lobar fissures. Occasionally both lungs are affected, but the disease commonly begins in one earlier than the other.

**Symptoms and Physical Signs.**—Shortly stated, the symptoms of a typical pneumonia are pyrexia, beginning suddenly with rigor, continuing, with a temperature of 103° or 104°, for five to eight or more days, and ending suddenly or gradually, and pain in the side, dyspnœa, cough, and expectoration of viscid sputum stained with hæmoglobin. The physical signs are those which indicate consolidation of the lung—viz. dulness, bronchial breathing, bronchophony, and increased tactile vibration.

*First Stage.*—A rigor or shivering (*69*) occurs, in a large proportion of adult cases, as the first definite sign of illness; the temperature rises to 102°, 103°, or 104°, and there is well-marked pyrexia, with malaise, loss of appetite, vomiting (*33*), furred tongue, and in some cases an eruption of herpes on the lips (*17*). Children often have convulsions, but rigors uncommonly. The symptoms may be at first vague, accompanied perhaps with pain in the head (*35·6*) or pains all over (*9*), or the implication of the lung may be indicated by shortness of breath and severe pain in the side (*60*), attributable to pleurisy. Auscultation at this early period may detect nothing, but sometimes there is heard a fine dry crepitation, which has been compared to the noise produced by rubbing between the finger and thumb a lock of hair near the ear; it is mostly heard towards the end of a deep breath, but sometimes during the whole of inspiration; and it is explained by the separation of the walls of the alveoli, rendered unnaturally adhesive. More often the first deviation from the normal is a marked diminution or loss of the vesicular murmur over the area which subsequently gives the signs of the second stage, or consolidation. The percussion note may be still unaltered, or only slightly less resonant than normal. In other cases a tympanitic note is obtained. This is liable to occur when the pneumonic process is central to begin with, so that there is relaxation of the surrounding lung (*see* p. 191).

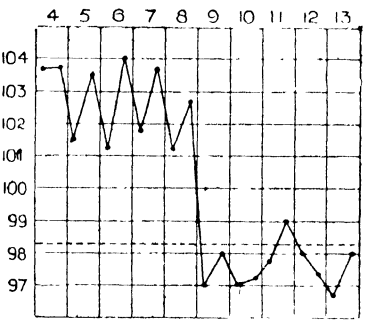


FIG. 20.—Chart of a case of pneumonia, with crisis on the eighth day.

Even as early as this there may be slight cough, with the characteristic *rusty sputum*. This is brought up as a mass of transparent, airless, jelly-like mucus, of a yellow, orange, russet-brown, or even bright red colour, and extremely viscid, so that it adheres to the side or bottom of the vessel with little or no tendency to flow. The pneumococcus may be detected in the sputum by Gram's method of staining; but it is not at first abundant, and the sputum consists chiefly of hyaline mucus, sero-albuminous exudation, some red corpuscles, small alveolar cells, large endothelial cells, and a few polymorphonuclear cells. Hæmoptysis (*1·6*) is an occasional feature.

The physical signs of the *second stage*, or stage of consolidation, are often rapidly developed. There is decided dulness over the part of the lung affected. Over the same area there is high-pitched bronchial breathing, at first soft and distant, but in a short time much louder. If the patient speaks, there is bronchophony, the words uttered being often distinctly heard, and apparently shouted up into the stethoscope; whispered words are also distinctly transmitted. The fine crepitation heard as an early sign may still be audible in portions of lung

(The *italic* numbers in brackets refer to percentage incidence of symptoms in 558 cases of lobar pneumonia among soldiers at Aldershot, 1915–17 (**17**).)

which are being involved by the spreading inflammation; but over areas which give loud bronchial breathing and bronchophony no râles will be heard unless there is associated bronchitis, and then they will be consonating. Tactile vocal fremitus is sometimes increased and sometimes diminished. There is evidence that in nearly all cases the latter is due to a thin layer of fluid in the pleura (**18**). During this development of the physical signs the patient is necessarily confined to his bed, but he often has orthopnœa; his cheeks and forehead are flushed; his eyes are bright, and show a vivid consciousness of his distress; his breathing is quick, and the respiration may rise to 40, 50, 70, or even 80 in the minute. The pulse is quickened, but not in proportion to the respiration; it may be 100 to 120, or somewhat higher; thus the pulse respiration ratio is altered from the normal 3 : 1 or 4 : 1 to 2 : 1 or 1½ : 1. The temperature is maintained generally at a high level, 103° to 105°, with little variation; and the skin is dry, and gives a sense of pungent heat to the hand placed on it. The blood pressure is generally a little below the normal. The cough, which is usually, though not always, present, is not very frequent, is hard, dry, and often painful; and the viscid, rusty sputum is brought up with difficulty. The urine is scanty, high-coloured, acid, and deposits urates; the chlorides are much diminished and may be absent, and there is not infrequently a small quantity of albumin. There is generally leucocytosis with increase in the polymorphonuclear cells, which persists for a long time in severe cases. The patient may become delirious (*4*), especially at night. Lividity or cyanosis is a striking feature if a large area of lung is involved. It has been found in such cases that the saturation of the arterial blood with oxygen is diminished, being 80 per cent. instead of 95 per cent., which is the normal (Stadie). This desaturation is due to the arterial blood being a mixture of aerated blood from the healthy part of the lung and non-aerated blood from the diseased lung. There is not very much of the latter, because the circulation through the diseased lung is slowed.

The general condition of the patient continues very much the same for some days, or more often there is an increase in the severity of the symptoms. The pulse and respiration are quicker, the temperature continues high, the tongue becomes drier and browner, and the delirium at night is more decided. The physical signs are generally observed to alter from day to day, indicating the spread of the consolidating process, so that crepitation and bronchial breathing extend higher and higher up to the chest, until the apex is involved, and the physical signs may be apparent in front under the clavicle.

When the illness is apparently at its worst, improvement takes place, in many cases quite suddenly. On the sixth, seventh, or eighth day, in a large proportion of cases, the temperature, the pulse, and respiration fall, in the course of twelve or eighteen hours, nearly to their normal limits; the tongue becomes moist; and the patient feels himself in all respects better. This *crisis* (*56·4*) is accompanied with profuse sweating. In about half the cases the fever ends more gradually (*lysis*), occupying from four to five days while falling from the acme to normal.

The physical signs gradually clear up after the temperature is normal. *Redux crepitations* are heard in this stage. They are rather coarse crackling or bubbling râles, due to the loosening of the exudation and its presence in the tubes. With the change in the lung, the sputum is also altered, losing its characteristic tinge, becoming yellow or green, muco-purulent, and at the same time less viscid.

In fatal cases death occurs from failure of the heart, or from œdema of the hitherto unaffected lung, or from both combined. All the symptoms are aggravated—the respirations are increased in frequency; the pulse is quick, small, and feeble; the face becomes livid or cyanosed; the physical signs of dilatation of the right ventricle may be observed; the tongue is dry, brown, and cracked; delirium is more or less continuous, and muttering and coma gradually supervene. On auscultation loud, coarse râles are heard on both sides of the chest. As the patient becomes feebler the temperature falls, the skin becomes cold and is

bathed in profuse perspiration. Death commonly takes place during the height of the illness, between the fifth and the tenth days. Occasionally, however, a pneumonia runs a fatal course in two or three days.

**Complications and Sequelæ.**—The former are mostly the result of secondary pneumococcal infections, spreading directly from the lungs to neighbouring structures, or being carried by the blood stream. *Pleurisy,* with formation of lymph or serum, is hardly a complication, as it is present in practically every case. *Empyema* (*20·8*) is not so common, but it should be suspected if fever continues into the third week, with dull percussion note, and disappearance or change of the bronchial breath sounds. Rarely a pneumothorax occurs in the course of pneumonia, from rupture of air vesicles into the pleural cavity. *Pericarditis* (*2*) is frequently associated with empyema on the left side. *Peripheral neuritis, nephritis* (*0·5*), *peritonitis, suppurative meningitis* (*1*), and *arthritis* (*0·5*), are among the rarer pneumococcal complications. Rarely a true *pneumococcal pyæmia* has occurred with suppurative arthritis and pustules and abscesses under the skin, yielding a thick greenish pus containing pneumococci in pure culture. *Malignant endocarditis* (*0·72*) (especially of the aortic valve) has been seen in association with pneumonia, and as a result of its specific organisms. In a small number of cases there is pronounced *jaundice* (*1·3*); a faint icteric tinge is more common. Acute *dilatation of the stomach* sometimes occurs in the course of the illness, and *parotitis* may ensue in severe cases. *Chronic pneumonia, gangrene* and *abscess of the lung,* and *bronchiectasis* are rare sequelæ.

(The *italic* numbers in brackets refer to the percentage incidence of the complications in 558 cases of lobar pneumonia among soldiers at Aldershot (**17**).)

**Diagnosis.**—In the early stages of rigor and high fever pneumonia may be indistinguishable from other *acute illnesses,* such as typhoid, scarlatina, or small-pox. Frequently the pain or distress in one side of the chest will indicate acute disease there, and the absence of breath sounds, or the fine crepitations, at one spot, followed by dulness, bronchial breathing, and bronchophony, will show the nature of the illness. But the pain may be very misleading; it may be so low in the back as to suggest variola; and it frequently extends to the abdomen, or is felt chiefly in the abdomen, so that *appendicitis, peritonitis,* or *cholecystitis* may be first thought of. A careful watch on the pulmonary bases is required to guard against error. In other cases a short cough, with expectoration of rusty sputum, will occur before the development of the physical signs These last may, indeed, be delayed for five or six, or even ten, days, and they may require much looking for and be first found in unlikely places, such as over the scapula, or at the top of the axilla. The absence of rashes characteristic of the exanthemata, the rapidity of respiration out of proportion to the pulse, the flushed face and bright eye, the characteristic sputum, and the presence of herpes about the mouth are useful points in making a diagnosis. An examination of the blood may help, as the presence of leucocytosis excludes typhoid fever, malaria, and influenza. The Röntgen rays are also of value, as the consolidated lung usually casts a definite shadow even in cases where the affected lung is so remote from the surface as to yield no physical signs. In children the pneumonic shadow takes the form of a triangle with its apex towards the hilum and its base along the axillary border of the chest (**23**). The movements of the diaphragm on the same side may be limited, and the right side of the heart is often seen to be enlarged.

When physical signs appear, it has to be determined whether pneumonia or *pleuritic effusion* is present, or a combination of both. The diagnosis of these two conditions from one another will be dealt with under Pleurisy; it will be sufficient to say here that pleuritic effusion, though often accompanied by bronchial breathing, causes more absolute dulness than pneumonia, and weakens or abolishes tactile vibration. When they co-exist the physical signs of the pneumonia are often masked by those of the pleuritic effusion which lies over it, whereas the pneumonia may be signalised by the rusty sputum and the pyrexial

conditions, which are commonly more pronounced than those of pleurisy. As pneumonia very rarely becomes chronic, physical signs of consolidation persisting for weeks with continued pyrexia are almost always due to pus or serum in the pleural cavity. The diagnosis from broncho-pneumonia is considered later (*see* p. 244).

**Prognosis.**—The average mortality of lobar pneumonia is about 18 per cent. It is very low in young children, but increases with age. The disease is more fatal to the intemperate, and to those who have been insufficiently fed. Among the soldiers at Aldershot the mortality was 10·9 per cent. Rather more than half the deaths were due to complications, empyema, pericarditis, etc. The mortality of double pneumonia was 28·7 per cent. Early or violent delirium, failing pulse, lividity and cyanosis, are all symptoms of bad augury.

**Treatment.**—The patient of necessity takes to his bed, and generally in the height of the disease requires to be supported in a semi-recumbent position by means of pillows or bed-rest. Physical rest with freedom from anxiety is one of the most important elements in treatment. Unnecessary medical examination should be avoided. He should have abundance of warm fresh air, in a freely ventilated room, no less than in any other infectious disease. Indeed, an open-air treatment has been advocated and carried out in Sydney, N.S.W., and it is stated that, if the mortality is not less in cases so treated, the average duration of the illness is less, the cyanosis and distress of breathing are less, and the patients sleep better. A diet suitable for a febrile illness should be given (*see* p. 24). Milk will naturally form an important element in it; but eggs and cereals, in the form of milk puddings, Horlick's malted milk, fish or minced chicken, etc., may be added. It is important to consult the patient's taste in such a matter. Beef tea or mutton broth, though not foods in themselves, may be appreciated. It is certain that no drug has any direct influence upon the inflammatory process as such, though much assistance may be obtained by supporting the heart in severe cases, as mentioned below. Treatment on bacteriological lines is sometimes employed; good results have been obtained in the case of pneumococci of Types I. and II. by injecting the corresponding anti-serum. The most important symptom to treat is the cyanosis when it is present. There is no doubt that prolonged anoxhæmia, even of slight degree, affects the heart prejudicially, and is often the cause of death. Oxygen should be given continuously for long periods at a time by a nasal catheter or by Haldane's apparatus with mask and valves (*see* p. 234). A patient rarely objects, when he realises the benefit of the treatment.

In early stages the bowels should be opened, and a free action of the skin should be encouraged by the use of acetate or citrate of ammonium, with small doses of Dover's powder. This last will relieve the pleuritic pain, or opium may be more frequently given in small doses (3 to 5 minims of tincture) with the saline. Local applications such as antiphlogistin, an ice bag or a poultice may also relieve pain. In mild cases this may be all that is required, but in the severer cases delirium and increasing prostration will have to be met. For the former chloral, chloralamide, and potassium bromide may be employed; but when there is much dyspnœa chloral must be given with caution, because of its depressing effect upon the heart and respiration. For the same reason, morphia must be sparingly used in the later stages. The subcutaneous injection of hyoscine hydrobromide ($\frac{1}{100}$ grain) is often useful and safer. For the increasing cardiac failure digitalis is often given, and small quantities of brandy or other spirit, up to the extent of 3 or 4 ounces daily. Where there is right-sided failure, venesection may be required. Where there is much secretion in the tubes, ammonium carbonate (5 to 7 grains every three or four hours) may be given. Recently artificial pneumothorax has been employed, and has been successful, in averting pleuritic pain **(19)**. When the crisis is past, and the temperature has fallen to the normal, the treatment requires simply to be directed to the strengthening of the patient

by the administration of quinine and other tonics, since the sequelæ of acute pneumonia are few and infrequent.

### FRIEDLÄNDER PNEUMONIA

From observations on cases in which lobar pneumonia has been due to the *Bacillus pneumoniæ* of Friedländer, it appears that it is generally a severe disease with a bad prognosis, that the lung in a fatal case oftener presents a blackish-grey than a red colour, and that the section is covered with a slimy mucus. The alveoli contain numerous bacilli and desquamating epithelium. Suppuration and gangrene are much more frequent than in the usual form, and the temperature is more variable. The bacillus of Friedländer may be accompanied by the pneumococcus, and it may be the cause of a lobular as well as a lobar pneumonia.

### BRONCHO-PNEUMONIA

(*Catarrhal Pneumonia, Lobular Pneumonia*)

**Ætiology.**—(1) *Primary Broncho-pneumonia.*—When the pneumococcus in pure culture attacks the lungs in children under five years old, it may produce a typical lobar pneumonia, but it more often attacks the alveoli in patches over the lung. Such cases have been called *primary broncho-pneumonia* (West) or *primary lobular pneumonia.* Except for the patchy distribution in the lungs, these cases resemble the lobar pneumonia just described, and there is no associated bronchitis. There is no necessity to say anything more about them. (2) *Broncho-pneumonia* proper, sometimes called *secondary broncho-pneumonia,* always starts from inflammation in the smaller bronchi, which spreads into the surrounding air vesicles. It occurs commonly in children under three years of age. It is a frequent complication of measles and whooping cough, and also follows other infectious diseases (scarlet fever and influenza, etc.). Ill-nourished town-dwelling children are thought to be more liable to broncho-pneumonia, and it is probable that rickets also disposes to it. Broncho-pneumonia occurs in adults from inhalation of foreign particles, especially septic materials from the throat, into the lungs (*inhalation pneumonia*); it is a common result of the spread of diphtheria down the bronchial tubes to the bronchioles; it is often a terminal event in any long-continued wasting disease, especially in elderly people who have been obliged to lie recumbent for weeks; when it attacks the most dependent parts of the lung it is known as *hypostatic pneumonia.* It may occur after operations under general anæsthesia, and it is a frequent cause of death in those who have been nearly drowned. Carcinoma of the œsophagus may be fatal by invading the lung and setting up pneumonia. But septic particles may reach the lung by the blood vessels, and pyæmia is characterised by its suppurative pneumonic foci. To many of these cases the term *septic pneumonia* is applied.

The bacteriology of broncho-pneumonia is complicated, and a great number of bacteria have been found in various combinations. A specific fever, such as measles or influenza, is to be regarded as exerting a depressing effect on the resistance of the individual, so that the lungs are invaded secondarily by various organisms. In the United States army the hæmolytic streptococcus was found to be the most important agent and the most fatal; but the pneumococcus, *B. influenzæ* and *Staphylococcus aureus* were also present on occasions. Friedländer's bacillus and in cases of diphtheria the Klebs-Löffler bacillus also occur.

**Morbid Anatomy.**—In broncho-pneumonia the consolidation is scattered throughout the lung in the form of nodules, often separate, but tending to coalesce, so as to form larger masses (*confluent broncho-pneumonia*), but even then still to be distinguished by the eye from each other. On section the solid lung is seen to consist of a number of small grey foci surrounded by dark red zones of hæmorrhage, œdema and collapse. The lung is soft and friable, and on squeezing

a bead of pus exudes from the small tubes. The process is sometimes called *splenisation* of the lung. Microscopically in cases of some duration there is infiltration of the lung tissue with wandering cells and a formation of new fibrous tissue in the bronchiolar and adjacent alveolar walls. Hence the name *interstitial broncho-pneumonia* which is often applied to the condition. The streptococci swarm in the bronchioles and thrombosed lymphatics. When the inflammation reaches the surface there is usually some pleurisy. In very acute and fatal cases there is no fibrous tissue formation, and the streptococci are seen invading the alveoli; abscesses may be formed in the lung.

**Symptoms and Physical Signs.**—The former are cough, dyspnœa, and pyrexia; the latter vary with the extent and position of the separate lesions. If the child has already a cough, with rhonchi and râles over the chest, from a preceding bronchitis, the implication of the alveoli may be indicated by a rise of temperature to 102° or 103°, by the cough becoming short, dry, and painful, and by the râles becoming more abundant and taking on a consonating character. But in many cases there are no rhonchi, and the physical signs consist of one or more areas, more or less extensive, in one or both lungs, in which rather sharp crackling râles are heard, with little, if any, change in the percussion note, or areas, also irregular in distribution, over which there is dulness, with bronchial breathing and bronchophony due to the aggregation of a sufficient number of consolidated lobules. Such areas may enlarge or diminish, and spread or clear up as the disease progresses. The sputum consists of mucus or muco-pus with or without streaks of blood, but young children usually swallow it. Exceptionally there may be free hæmoptysis.

The course of the disease is not so definite as in pneumococcal pneumonia. It may end in a week, but often goes on for three or four weeks, or even more. The temperature is generally remittent or even intermittent, and usually falls by lysis; it may be very irregular. The breathing is rapid, and is *inverted*, which is often valuable in diagnosis. There is a quick inspiration, the breath is held for half a second, expiration then occurs with a grunt, and inspiration again follows without an interval. The lower intercostal spaces are depressed during inspiration. There is much cough; the face is flushed, or in severer cases pale and livid. The pulse is quick and small. Delirium is often present. The physical signs frequently alter in the course of the illness, indicating the clearing up of disease at one part, and fresh outbreaks in others; the disease often attacks both lungs. Recovery is mostly gradual, and not sudden, as in pneumococcal pneumonia Convulsions may precede death.

**Diagnosis.**—Broncho-pneumonia may be confounded in its early stages with other *acute illnesses*, characterised by high fever, such as typhoid fever; and the liability of children to marked cerebral symptoms from any acute illness may lead to a diagnosis of *meningitis*. The preceding bronchitis and the predominance of the chest symptoms may prevent a mistake, but an opinion may have to be suspended for a few days. Long-continued broncho-pneumonia may give rise to a suspicion of *tuberculosis*, in which high fever, universally scattered râles, lividity, and cough are prominent symptoms. In *capillary bronchitis* there are dyspnœa, lividity, and râles, but the râles are often confined to the bases; there is no bronchial breathing. There may be profuse purulent expectoration. The diagnosis from pure pneumococcal pneumonia may be rather difficult. In the latter case the signs over the lungs are more uniform, and there may be a crisis. In broncho-pneumonia there is bronchiolitis, as indicated by consonating râles of varying coarseness in both lungs, and since the patches do not resemble one another in the state of development of the inflammatory process, the breath sounds will change between puerile and fully developed bronchial breathing of varying pitch.

**Prognosis.**—Though this form of pneumonia is much more fatal than the pneumococcal variety, the prognosis in any given case must depend upon the

general progress of the symptoms. Cases that are apparently desperate often recover, and an unfavourable opinion should be given with some caution. In the broncho-pneumonia of old people, and in that which is due to the inhalation of solid particles, the prognosis is more grave.

**Treatment.**—The treatment may be conducted on the same general principles as in the case of ordinary pneumonia. The room should be well ventilated, with free access of air to the patient; open-air treatment has been tried with success. Oxygen should be given continuously for long periods if there is respiratory distress or lividity (*see* p. 234). Tr. belladonnæ in doses of 3 to 5 minims may be given to quite young children in order to dry up the pulmonary secretion. Injections of atropine are also sometimes given. Expectorants are not generally approved of for young children, as they have no power of coughing up sputum. Severe cases often require stimulants to be administered rather freely—*e.g.* for a child three or four years old 20 minims of brandy every hour; or 1 or 2 minims of liquor strychninæ may be injected two or three times daily at this age, and smaller quantities in infants.

## ABSCESS OF THE LUNG

Abscess may be a result of pyæmia, and of acute pneumonia. It may be determined also by foreign bodies entering the bronchi; and new growths, such as carcinoma, and probably syphilitic gumma, occasionally suppurate. It may occur after tonsillectomy from the inhalation of septic material into a bronchus. The differential diagnosis of abscess from consolidation of the lung is difficult until the abscess has burst and discharged pus. Then the cardinal signs are (1) purulent sputum, which may be foul; (2) cough and explosive expectoration; (3) elastic tissue with alveolar arrangement in the sputum; (4) circumscribed dulness on percussion; (5) X-ray appearance of a cavity with a fluid level. When the fluid contents of the cavity have been coughed up there may be the usual signs of a cavity, such as tympanitic resonance if the cavity is large, cavernous or amphoric breathing, metallic tinkling, and pectoriloquy. The multiple small abscesses of pyæmia are not recognisable as cavities; indeed, their presence is usually masked by the surrounding consolidation, or pleuritic effusion.

**Prognosis and Treatment.**—Less than 10 per cent. of cases recover with purely medical treatment, which is described under Bronchiectasis. Various surgical measures have been advocated, with 50 to 60 per cent. of cures; the simplest is to resect a rib and drain the cavity. Recently some success has been obtained with artificial pneumothorax.

## CHRONIC PNEUMONIA

### (*Fibrosis of the Lung, Fibroid Lung*)

**Ætiology.**—This form of lung disease is comparatively rare except in children, the great majority of chronic inflammations of the lung tissue in adults being associated with tubercle. The cases in which a chronic inflammation is independent of tubercle, and to which the names *chronic pneumonia* and *cirrhosis* or *fibrosis of the lung* are given, arise only rarely from a preceding acute lobar pneumonia; but broncho-pneumonia is a more frequent antecedent. Chronic bronchitis, bronchiectasis, and chronic dry pleurisy appear to be the causes in other instances.

An important class of cases, in which chronic pneumonia succeeds to bronchitis, is that occurring in various manufactories, and known as *pneumokoniosis*. Here the repeated inhalation of an atmosphere laden with the dust of coal, metal, stone, cotton fibre, fluff, etc., provides a lifelong source of irritation. The disease has received different names according to the particular irritant concerned

—*anthracosis* (coal dust), *siderosis* (steel), *silicosis* (stone). Silica workers are specially liable to secondary tuberculous infection.

**Morbid Anatomy.**—The characteristic feature of the lung affected with chronic pneumonia is the excessive development of fibrous tissue in its substance. At first the lung is traversed by bands of fibrous tissue, arising in the interlobular septa; in late stages the whole lung may be converted into a dense mass of fibrous tissue, of various shades of grey from the presence of pigment, tough in consistence, and creaking under the knife. In cases of dust disease, the lung is coloured black, red or grey, according to the nature and quantity of the particles inhaled. With the growth of the fibrous tissue contraction takes place, and the lung may be reduced to two-thirds or half its natural size; the excavations commonly occur partly from dilatation of bronchial tubes, and partly from ulcerative processes in the lung itself. Nearly all cases are accompanied by a chronic pleurisy, and the lung is fixed to the chest by a thick fibrous layer. The contraction of the lung leads to displacement of organs, and, as usually only one side is affected, the mediastinum is pulled in that direction.

**Symptoms and Course.**—The disease is generally chronic, and patients in whom it is recognised have usually complained for some months or years: but that the formation of fibrous tissue may begin very early is shown by a case in which the alveolar exudation was being organised into connective tissue at the end of three weeks (Kidd). The patients are short of breath, and have cough and expectoration, which vary with the extent of the cavities in the lungs (*see* Bronchiectasis). When there are large cavities or much-dilated tubes, the cough may be paroxysmal, with abundant and perhaps fœtid expectoration. The patient is often thin, but may be well nourished, and is at any rate for a time free from the fever, night sweating, and general constitutional disturbance observed in phthisis. Hæmoptysis is often present. Some of the local conditions have been already indicated. The disease is commonly unilateral; the corresponding side of the chest is retracted, the shoulder depressed, and the angle of the scapula tilted outwards; the impulse of the heart is shifted towards the affected side, and the healthy side of the chest is hyper-resonant. The affected side expands but little; it is dull on percussion. The respiratory sounds are feeble or distant; and if large cavities or much-dilated tubes (bronchiectasis) are present, the breathing may be hollow or tubular with metallic or bubbling râles. Such cavities are more often situated about the middle level of the lung than at the summit, as in phthisis. Tactile vocal fremitus is commonly diminished. Thickening or *clubbing* of the finger ends (*see* p. 672) is often pronounced. Eventually there may be right-sided heart failure.

**Diagnosis.**—The condition has to be distinguished from phthisis, from chronic pleurisy with effusion, and from malignant growth in the chest. From *phthisis* the absence of fever and constitutional disturbance is the chief distinguishing feature; the disease is often rigidly unilateral, whereas phthisis rarely reaches an advanced stage in one lung without affecting the other; and tubercle bacilli are not found in the sputum. *Pleuritic effusion* of old standing with retracted chest may closely resemble the fibroid lung, and exploration with a needle may be required to clear up the diagnosis. Pleuritic fluid, whether simple or purulent, is generally accompanied by feverishness, and, on the other hand, crepitation and râles will be much in favour of chronic pneumonia. *Intrathoracic carcinoma* is likely to be associated with cachectic appearance, with irregular retraction of the chest, with pains, with extensive consolidation, and signs of pressure or displacement of the heart or other organ; but one or more of these indications may be absent.

**Prognosis** is ultimately bad, but the course may be very slow, extending over ten or fifteen years. Death may take place from failure of the right heart, or from the gradually increasing exhaustion which follows profuse discharge, or from metastatic abscesses, in particular cerebral abscess.

**Treatment.**—The patient should be placed under the best possible climatic and hygienic conditions. He should have bracing air in the summer, but a warm climate in the winter; avoid exposure to chills at all times; and have nourishing diet and tonics, such as quinine, iron, and cod-liver oil. Cough, expectoration, and other symptoms should be treated as they arise in the same manner as directed under Phthisis and Bronchiectasis.

## GANGRENE OF THE LUNG

This is a comparatively rare disorder, which may arise, however, in a variety of circumstances. It is one of the terminations of acute lobar pneumonia, especially in cases dependent on Friedländer's bacillus, and it occurs sometimes in phthisis; it may result from the invasion of the lung by adjacent diseases like carcinoma of the œsophagus, abscesses, and suppurating hydatid cysts, from the pressure of aneurysm on the root of the lung, and from injuries to the chest setting up pneumonia or empyema; as a result of foreign bodies lodged in the bronchus, and from the presence of secretions retained in dilated tubes; from the passage into the lung of particles from septic diseases in the mouth, throat, larynx, œsophagus, or mediastinum, such as carcinoma of the tongue or larynx, sloughing of the tonsils, diphtheria, carcinoma of the œsophagus, or tuberculous bronchial glands; from particles of food drawn into the lung by accident, or during vomiting, especially in persons who are drunk, insane, comatose, or suffering from laryngeal paralysis; or from impure water inhaled during immersion. Gangrene of the lung is also sometimes caused by septic particles brought to it by the blood vessels, as in various pyæmic processes, after otitis, bedsores, puerperal disorders, etc. Thus it is seen that it is always in association with infection by some micro-organism, which may be a streptococcus, staphylococcus, pneumococcus, tubercle bacillus, *Bacillus coli communis*, or one of the higher bacteria (*see* p. 10). Such infections are common without producing gangrene; and gangrene, when it occurs, seems to be determined by the presence of putrefactive organisms, such as *Bacillus proteus*, *Bacillus fluorescens putridus* and *leptothrix*.

The conditions which have been regarded as predisposing to such invasion are marasmus, old age, intemperance, diabetes, general infectious diseases in their typhoid and adynamic stages, and some paralytic and mental disorders.

**Morbid Anatomy.**—The affected portion of lung is of a dirty, greenish-brown, or black colour, soft, readily breaking down or even diffluent, and often emitting an offensive odour. It is generally surrounded by consolidated pneumonic tissue, into which it may gradually pass, or from which it may be more or less sharply separated off by a line of demarcation; thus in some cases the lesion is diffuse, in others definitely circumscribed. The gangrenous tissue may break down, and be expectorated, so as to leave a cavity with ragged, shreddy walls; and occasionally such a cavity opens into the pleural sac and causes pyo-pneumothorax.

**Symptoms.**—Gangrene of the lung occurring, as it often does, as a secondary lesion, just before death, may be readily overlooked. On the other hand, its symptoms may stand alone, or overshadow those of the primary lesion. Fœtid expectoration and fœtid odour of the breath are the most prominent. The latter may be very penetrating; it is carried to a great distance, and makes it almost impossible for other persons to live in the same room with the patient. The sputum is dirty grey or greenish brown, or black, from altered blood; and either fragments of gangrenous lung tissue are found, or the microscope detects the typical elastic fibres (*see* p. 257). Occasionally hæmoptysis takes place. Cough, pain in the side, and irregular, and mostly intermittent, pyrexia are also present. The physical signs are those of consolidation and cavity proportionate to the extent of lung disease—viz. dulness, bronchial or cavernous breathing, broncho-

phony, and medium or coarse râles; but their value in diagnosis must depend a good deal on the preceding disease, if any. The illness may begin with rigor and pain in the side, or with hæmoptysis, or with recurring attacks of fever and fœtid expectoration; in most cases these are soon followed by prostration, with quick small pulse, dry tongue, and death at no great distance of time. Some cases, however, last for months or years, with much variation in the intensity of the symptoms, but without escaping a fatal termination. And in a few cases, with probably a very small patch of gangrene, recovery actually takes place.

**Treatment.**—This is similar to that of fœtid bronchitis or bronchiectasis. Antiseptic inhalations (creosote, carbolic acid, menthol, eucalyptus oil, thymol) or the antiseptic respirator should be frequently used. Guaiacol (3 to 5 minims) and oil of turpentine (10 to 16 minims) may be given internally. Successes have been obtained with arsenobenzol and novarsenobenzol. A gangrenous cavity is sometimes amenable to the surgical treatment of antiseptic incision and drainage; and the operation should be considered when a certain diagnosis can be made, and the associated conditions are in themselves not necessarily fatal.

## PULMONARY TUBERCULOSIS

Tuberculosis of the lung occurs in several forms. In one there is a general distribution of minute tubercles throughout the organ, arising acutely, and determined by the carriage of the tubercle bacilli by the blood from some other part, such as a bronchial or cervical gland, or a joint or kidney, or less commonly from a focus of chronic disease in the lung itself. This acute form is often part of a general miliary tuberculosis, in which tuberculous meningitis is usually the most striking feature (*see* p. 129). The other forms of pulmonary tuberculosis have this in common, that the affection of the lung usually makes the chief part of the clinical picture.

*Phthisis* (*φθίω*, I waste) or *consumption* is a chronic disease of adults or later childhood in which tubercles are formed and multiply in one small part of the lung, usually the apex, and spread with very varying degrees of rapidity to other parts of the lung. It is thus at first entirely local. The later changes are assisted by the action of other organisms, especially the *Pneumococcus*, *Streptococcus*, and *Staphylococcus pyogenes*. In apical phthisis there is not usually caseation or calcification of the regional lymph glands. It probably starts as a direct infection of the lung due to inhaled tubercle bacilli. Much discussion has taken place as to why the apex is primarily affected. Keith has pointed out that this part of the lung is less ventilated than other parts, because the top of the thorax is relatively immobile, the greatest movement taking place at the diaphragm and over the lower part of the chest wall. Since the ventilation is least at the apex, it may well be that the circulation is also least, so that the bacilli find a favourable nidus. Another explanation, not a very probable one, is that the infection begins in the tonsils and spreads along the cervical lymphatics direct to the apex of the lung across the pleura.

*Hilum phthisis*, or *peribronchial phthisis*, is also a chronic disease of the lungs, which occurs rather uncommonly in adults; but it is the usual form that chronic pulmonary tuberculosis takes in young children. Tuberculous infiltration of the bronchial glands and of the lymphatics draining into them from the surrounding lung is the chief lesion. It has already been explained that the lung provides the primary focus in these cases (*see* p. 127). Tubercle bacilli are taken up by the lymphatics of the lungs, which become gradually choked or obliterated in the process, beginning at the hilum and gradually extending outwards towards the surface of the lungs. Hilum phthisis may give rise to a more acute tuberculous process at any place in the lungs. Usually, however, it heals in course of years, and it has been suggested that the pulmonary scars found so frequently at post-mortem are the results of this disease of childhood, now

extinct (Opie). In this case the apical phthisis of adults is a reinfection in later life, although whether the patient who has once had hilum phthisis is specially sensitive to a fresh infection or specially resistant is open to doubt. At the same time it is believed by many that apical phthisis is an exacerbation of older trouble in childhood owing to lowering of resistance. The reinfection hypothesis is the most likely.

*Tuberculous broncho-pneumonia* is an acute tuberculous process occurring in children or young adults, in which caseous tuberculous foci, with commencing cavity formation, are scattered through the lungs. In children it commonly results from the ulceration of a caseous gland into a large bronchus, with rapid dissemination by inhalation of the highly infective material throughout the lungs. The apex usually is not specially affected. *Acute pneumonic phthisis*, which has been called "galloping consumption" or *Phthisis florida*, is a still more acute process, and is described later.

The ætiology of pulmonary tuberculosis has already been discussed in the section on tuberculosis in general (*see* p. 125).

**Morbid Anatomy of Phthisis.**— Tubercles form and develop in the lungs in the most typical way, with their giant cell systems, and their tendency to caseate and break down (*see* p. 128). In the common apical lesion the process starts in the wall of a small terminal bronchus. Caseation occurs, and the tuberculous mass breaks down; the material is discharged through the bronchus, and a minute cavity is formed connected with it. Meanwhile the process spreads into the neighbouring lung tissues, which become consolidated partly by caseation and partly by cellular inflammatory exudate. The solid areas are dark in colour, with small white caseous tubercles in bunches scattered through them, and usually a fair amount of consolidation takes place before much cavity formation is seen. Cavities or vomicæ are formed by a mixed process of caseation and suppuration. Adjacent cavities run into one another, and ultimately the lung may be extensively hollowed out. In their earlier stages the walls are often formed of caseous deposit, but in old vomicæ they are quite smooth. They are often traversed by bands, or trabeculæ, which contain pulmonary vessels. The vessels resist the destructive process, whereas the bronchi are generally ulcerated in proportion as the cavities enlarge, and into each cavity one or more bronchi open. The contents of vomicæ are caseous matter, *débris* of lung tissue, and pus. The latter predominates in the older cavities; the quantity is very variable, and it may be so small, under certain circumstances, that no expectoration takes place for considerable periods. It is only rarely that decided putrefaction takes place in phthisical cavities.

While the apical lesion is progressing infective material from the cavities may be aspirated into the opposite lung and into lower parts of the same lung and set up numerous smaller lesions of essentially the same character. In cases where the patient has been infected with enormous numbers of bacilli, or where the resistance is low, the spread of all these foci into the surrounding tissues takes place before the caseous matter can be discharged. The whole lung becomes completely solid, being filled with partly caseous and partly gelatinous-looking material, the latter representing parts of the lung not quite so completely destroyed (*caseous lobar pneumonia, acute pneumonic phthisis*). In other cases at post-mortem large cavities are in process of formation, their walls being extremely ragged. If the patient dies in a less advanced stage, the lungs will show numerous discrete caseous and gelatinous areas with early cavity formation (*caseous broncho-pneumonia*), since here the areas have not had time to coalesce to produce the diffuse pneumonic condition.

But in the majority of cases this process of destruction does not have full play. The inflammatory changes present varying changes of activity in different cases; and the mischief may be stopped for long periods one or more times in its course, or may even become abortive at an early date, and go no farther. The

development of *fibrous* tissue is the important agent here. It is rarely absent in any but the most acute cases, and in the chronic cases it forms a large proportion of the remaining tissue of the diseased lung. In the consolidated lung there are numerous bands running in the course of the interlobular septa, surrounding the bronchi, the blood vessels, and the cavities, and forming a dense layer under the visceral pleura (fibroid phthisis). The fibrous tissue is frequently deeply pigmented, and is mixed here and there with caseous masses. By its contraction it tends to diminish the size of the cavities, and opposes some resistance to destructive processes; and in some favourable cases a small deposit of tubercle may be ultimately converted entirely into a mass of pigmented fibrous tissue, which, indeed, replaces a similar amount of healthy lung, but is otherwise harmless. In these cicatrices it is not uncommon to find calcareous particles, from the deposit of calcium salts in the caseous material; and around such a cicatrix may arise the condition known as *compensatory emphysema* (*see* p. 232).

When the process is advanced the pleura seldom escapes. The formation of tubercle in the pleura is not common; but inflammation of the membrane is the result of the extension of the pulmonary change, whether of consolidation or excavation, to the surface. The *pleurisy* is often chronic or subacute; if acute, the area invaded at one time is but small. The final result is the formation of a thick layer of membrane over the affected portion of lung, commonly uniting the organ firmly to the wall of the chest. This adhesion of the lung has an important influence, for, if the process of excavation advances to the surface at a point which is not adherent, the vomica may ulcerate through, and discharge its contents into the pleural cavity, leading, on the one hand, to an acute pleurisy, generally of the purulent variety—*empyema;* and, on the other, to the entrance of air into the pleural sac—*pneumothorax*, or pyo-pneumothorax.

Another important result of the destruction of tissue is *hæmorrhage:* in earlier stages this follows from congestion alone; in later stages the vessel walls are directly invaded by tubercle, and hence may be eroded, or they may be weakened and dilate so as to form aneurysms, which may reach the size of a pea or bean, and ultimately give way at the thinnest part.

**Situation of the Lesions.**—As already indicated, the above changes follow one another with very varying rapidity, and the spread of the disease through the lung is equally irregular as to absolute time. But the situation of the lesions and the order of their invasion are subject to some very constant rules.

The first deposit of tubercle takes place one or two inches below the apex of the upper lobe; and fresh deposits occur at intervals of weeks, months, or years, lower and lower down. This invasion of fresh parts of the lung takes place by direct contiguity, by lymphatic channels, and largely through the bronchi; infective particles are inhaled into them, and thus start fresh centres of disease. By the time that tubercle forms at the lower levels the first lesion may have led to considerable consolidation; and later on, when tubercle is being deposited towards the base, the middle part of the lung will have solidified, and the apex may contain a large cavity.

Again, the progress of the disease, while unequal in any one lung, is unequal in the organs on the two sides. As a rule, before a patient dies of phthisis both lungs are affected, but rarely to the same extent; a large area is often involved on one side before the other is attacked; and so, in an advanced case, it is common to find the most extensive disease at one apex, and the most healthy tissue, or the only healthy tissue, at the opposite base. The law of the extension of lesions from apex to base may be supplemented by the rule that the apex of the lower lobe is often invaded soon after the apex of the upper lobe, and before the lower part of the upper lobe; and in testing the truth of this observation clinically it must be remembered that the lower lobe occupies the greater part

of the back of the chest, reaching as high as the third dorsal spine, or the spine of the scapula, and that the greater part of the front of the chest corresponds to the upper lobe. A primary lesion of the lower lobe (primary basal phthisis) is very rare.

In *fibroid phthisis* the affected lung is found to be contracted to one-third or one-quarter its normal size, firmly adherent to the chest by a thick dense fibrous layer, and presenting a quantity of dense white or grey fibrous tissue, which contains caseous or cretaceous deposits, vomicæ, and dilated bronchial tubes. Tuberculous fibroid change may be present to a smaller extent in the opposite lung.

**Clinical History of Phthisis.**—Phthisis may run a rapid or a slow course. The description which follows will mainly apply to a case which lasts from six months to a few years.

The onset is variable. Many cases begin with cough and expectoration of muco-pus or pus for which no cause can be given, or which is referred to some chill or exposure. Other cases begin with hæmoptysis or spitting of blood. The patient may have been apparently in good health, when sometimes after an effort, but quite as often when still, or walking or doing something which involves no strain, a tickling is felt in the throat, the patient coughs, and is surprised and alarmed to find that what he spits is blood. Thereupon he may expectorate a few drachms or an ounce, or even half a pint. This may remain the only symptom, and an examination of the chest may reveal nothing. But after a time, with or without a fresh loss of blood, cough and expectoration supervene, and the case develops like others. In a small number of cases the first apparent departure from health is an acute pneumonic process in one upper lobe, which only partially clears up, while cough and expectoration persist, and the case takes on all the features of phthisis; and in others the first recognisable illness is a pleurisy with effusion, which may even appear to recover completely, and yet be followed by the usual pulmonary changes. Lastly, in some cases indigestion with loss of appetite, frequent vomiting, and emaciation, are prominent symptoms for some time before the special indications of a lesion of the chest are apparent.

The disease is also very variable in its course in different cases. Patients with the earliest symptoms, whether hæmoptysis, or cough, or wasting, placed under favourable conditions of climate and hygiene, may completely regain their health; and it has long been known that in persons killed by accident, or dying of disease unconnected with the lung, cicatricial and pigmented patches, with perhaps calcareous deposits, are found in the apices which can only be regarded as the remains of former tubercles.

If, however, the infection is well established before being submitted to treatment, the result cannot be so satisfactory. Thus the disease may be fatal in three or four months, or it may last twelve or fifteen years before finally killing the patient; and in this time its progress will be very unequal, often quiescent for months or a year or two, and then making great strides, with hæmoptysis or much fever. While the more rapid cases are fatal chiefly by the extent of lung involved, the cases of longer duration threaten life by a number of complications, some of which are lesions of the lung itself, such as hæmoptysis, empyema, and bronchitis; while others involve distant organs, such as tuberculous meningitis, ulceration of the intestines and diarrhœa, nephritis, and lardaceous disease of the viscera.

**Local Symptoms.**—These will now be described somewhat more in detail.

*Cough.*—This is a very common symptom and generally, though not always, present as long as the disease is in any degree active. It is mostly easy at first, sometimes not much more than a clearing of the throat; it becomes harder in the latter stages; and with extensive cavities it occurs in prolonged attacks, painful to the patient, distressing to those about him, and lasting perhaps more than a minute, until at length some sputum is brought up. With laryngeal complication the cough acquires a hoarse or husky quality.

*Dyspnœa.*—Shortness of breath is often early noticed, and becomes very

marked as more and more of the lung is diseased, and so the surface available for interchange of blood gases is diminished.

*Expectoration.*—In the early stages this is not different from the sputum of bronchitis—that is, it is either simply mucous or muco-purulent; and this is accounted for by the bronchitic processes which frequently accompany phthisis. But sometimes comparatively early, and always in later stages, the sputum becomes purulent, of green or greenish-yellow colour, opaque, and quite free from air bubbles. If it is very fluid, the individual sputa may run together and lose their separate form; but the sputa of phthisis often keep separate long after expectoration, and, from the round, flat shape that they assume in the sputa vessel, they are called *nummular*, or coin-shaped. This is no doubt due to the accumulation of the secretion in cavities in the lungs, and hence it constantly occurs in phthisis, but may also be present in cases where the cavities are produced by dilated bronchi (bronchiectasis). The microscopical examination for tubercle bacilli is described later.

*Hæmoptysis.*—When hæmoptysis occurs as the first sign of phthisis, the blood is generally bright red and frothy; it is expectorated in variable quantities, and, as a rule, for some hours or days the patient continues to spit pellets of blood which have a darker and darker colour, become gradually less frequent, and then cease entirely. There may at this time be no other sputum. In later stages, when the disease is well established, the muco-purulent or purulent sputum is often streaked or stained with blood. A few streaks in the sputum may proceed from small vessels in the bronchial mucous membrane, but more characteristic of phthisis is the intimate mixture of bright blood with the sputum, or the discharge of pellets of coagulated blood frequently during the day. From time to time may occur more abundant hæmorrhages, like those first described, in which the blood comes up apart from the ordinary secretion; and if a large vessel is ulcerated, or, what is more often the case, if a small aneurysm in a cavity ruptures, several ounces or a pint or two of blood may be discharged within a short time, and death may follow rapidly. Hæmoptysis tends to occur rather more frequently at night than in the daytime, possibly owing to the cold (**20**).

**Physical Signs.**—From the clinical point of view three stages of apical phthisis are described (Turban-Gerhardt classification). In Stage I., which represents early cases, the disease is limited to a small area of one or both apices. In Stage II. there is consolidation, the disease affecting the whole or the greater part of one lobe. Stage III. represents still more extensive disease and all cases where there is considerable cavity formation.

The physical signs are best described according to these stages. In the *first* stage they may be very slight, and vary considerably in different cases. The eye or the hand may detect a slight impairment of mobility on the affected side. For this purpose the relative movements should be watched during tranquil and during full, yet gentle, respiration. Careful percussion of the apex may give slight impairment of note as compared with the opposite side. There is normally a band of apical resonance, 4·5 to 5 cm. ($1\frac{3}{4}$ to 2 inches) wide, running over the shoulder (*Krönig's isthmus*). This may be narrowed in disease. An impaired note may also be obtained just below the clavicle, on the clavicle, or in the supra-clavicular fossa. The patient should be sitting in a relaxed position. Auscultation often gives a diminution of the vesicular murmur and the presence of fine or medium râles during inspiration. These are heard best towards the end of the first inspiration after coughing. Sometimes there is nothing but the deficiency of vesicular murmur, but this, if associated with impaired resonance or mobility, is of much importance. The expiratory murmur may be loud and prolonged, as in bronchial breathing, and this may be associated with an increase of vocal resonance. It is, however, very important to remember that prolonged loud expiratory murmur with loud vocal resonance is not uncommon upon the right side in healthy individuals, and especially in females. And, as a rule, repeated

examinations at short intervals are needed before one can with confidence state that there is evidence of apical phthisis from the physical signs in an early case. Impairment of note and râles are the most trustworthy signs. However, sounds resembling very closely crackling râles are sometimes produced in the sterno-clavicular joint. Irregular, jerky or wavy breathing—the so-called cog-wheel respiration—is of no diagnostic significance.

In the *second* stage (consolidation) the physical signs are in many respects similar to those of the second stage of pneumonia. According to the extent of lung involved, there is more or less impairment of mobility of the affected side; and when the progress has not been unusually rapid, there is obvious depression of the supra-clavicular and infra-clavicular regions, caused by contraction of fibrous tissue, or perhaps by the earliest destruction of tissue, producing cavities as yet too small to be recognised by physical signs. On percussion there is increasing loss of resonance as the case goes on; but the dulness is rarely so absolute as that which occurs over a pleural effusion. On auscultation bronchial breathing of different qualities and pitch is heard, and the voice and cough are loudly bronchophonic. Râles are often present; they are mostly of sharp clicking or consonating character.

It is in the *third* stage (excavation), when the disease has existed some time, has seriously involved one lung, or has already begun to attack the other, that the modifications in the shape of the chest are most obvious. On the most affected side the chest takes on the type of extreme expiration. It is flat, long, and narrow; the shoulder is depressed and sloping; the lower angle of the scapula is displaced inwards; while the upper ribs in front are wide apart, the lower ribs are crowded together, and the epigastric angle is reduced to its smallest size. The crowding together of the ribs gives a characteristic X-ray appearance, called "roof-tiling," as shown on the left side in Plate III. (*see* p. 257). The nipple tends to lie high in relation to the ribs—for instance, in the third space—while the heart may strike the fifth rib, instead of the fifth space, as if the ribs had glided down between the skin and the viscera. In addition to this general change in the chest, there is retraction of the upper part of the chest and a corresponding impairment of movement. At this point, which we now suppose to be the seat of cavities, we find that the percussion note is variable. It must be remembered that the cavities form in lung which has first become solid. Now, absolutely solid lung gives dulness on percussion, and a lung entirely hollowed out into one large cavity gives a hyper-resonant note; the percussion note over excavated lung must, therefore, vary with the size of the cavity, its nearness to the part of the chest percussed, the amount of consolidation around it, or between it and the point struck, and the degree to which the ribs are fixed by pleuritic adhesions. The note may, therefore, be quite dull, or of tympanitic resonance. If there is a large cavity in free communication with the bronchial tube, and the patient's mouth is open, percussion will often elicit the *cracked pot sound*, or *bruit de pot fêlé*, which is somewhat, but not exactly, like the sound produced by striking the two clasped and hollowed hands upon the knee to delude children with a hope of pence. There are two elements in its formation: (1) the presence of a cavity filled with air; (2) the rapid passage of the air out through a narrow opening on percussion. The resonance over a cavity is raised in pitch when the patient opens his mouth (Wintrich), and during inspiration, while it falls in expiration (Friedreich); and the pitch varies with change of position of the patient (Gerhardt). On auscultation over cavities one may obtain bronchial, cavernous, or amphoric breathing, according to the varying degree of excavation and condensation around. It is only truly amphoric when the cavity is very large indeed. The vocal resonance is increased (bronchophony), and there is increased clearness of the articulated sounds (pectoriloquy). In extensive cavities, when the patient speaks, there is heard, in addition to the loud vocal resonance, a kind of *whispering echo* of the same, apparently produced by reverberation

from the walls of the cavity. Crackling râles of large size and *metallic tinkling* are even heard in cavities. The auscultatory sounds are best brought out on deep inspiration or on coughing, when sometimes the sound of *post-tussive suction* may be heard. A cavity probably does not give distinctive signs as compared with consolidation, unless it has reached at least the size of a walnut. At the same time it must be pointed out that a cavity has been found *post mortem* when there were no signs of it during life.

In cases of long standing, in which the left lung is mainly affected, the contraction of that organ allows the heart to come more fully into contact with the chest wall, and in the second left intercostal space may be observed the pulsation of the conus arteriosus of the right ventricle; the closure of the pulmonary valves may be then felt with the finger, and the second sound is heard with greater distinctness than is normal.

**General Symptoms.**—The more important are pyrexia, with night sweating; loss of flesh and strength; anæmia; and the evidences of imperfect aeration of the blood.

*Pyrexia.*—From the earliest days of phthisis fever may be present, but it generally bears some relation to the activity of tuberculous processes in the lung, so that, if the mischief becomes inactive from time to time, the fever may for a corresponding time be absent; but it is often present continuously for months. The temperature is commonly higher in the evening than in the morning, and is either remittent or intermittent in type. In the former it may be 99° to 100° in the morning, and 102° or 103° in the evening; in the latter it is 98·4° or even lower in the morning, and reaches 100° to 103° in the evening. The lower degrees of fever are often not appreciated by the patient; the higher are accompanied with the discomfort and malaise common in pyrexia, and especially in the advanced stages of the disease, with profuse sweats and even slight chill before the sweating. Actual rigors, however, are exceptional, and the most common event is for the patient to sleep more or less quietly in the early part of the night, so far as the cough will let him, and to wake up in the early morning to find himself drenched with perspiration. These are the "colliquative sweatings" of older writers. Some night sweating is not uncommon even in early stages.

*Loss of Flesh and Strength.*—Emaciation is the rule in phthisis; it may be one of the earliest symptoms, and may give a note of warning when the cough has been thought to be a mere bronchial catarrh; towards the end of a chronic case the emaciation is extreme. If, as a result of treatment at any time, the patient improves, he commonly puts on a little flesh, or at any rate remains stationary. Exceptionally nutrition is maintained fairly well even when the physical signs show that there is a considerable and even apparently active lesion. The muscular power is soon enfeebled, and the patients lose energy, becoming languid and unfit for prolonged exertion, whether of mind or body. The mental condition in many patients, however, is one of great hope and confidence; even when helpless in bed they often fail to realise how ill they are, and look for complete recovery could they but once get rid of the cough.

*Anæmia.*—The loss of nutrition is represented also in the blood, and the patient is commonly pallid, both in the early and late stages.

*Imperfect Aeration of the Blood.*—This shows itself by lividity of the face, especially in acute cases, involving a large area of lung. In chronic cases in which the right side of the heart has become somewhat dilated there is more obvious cyanosis, which is due chiefly to retardation of the venous circulation, so that more oxygen is removed from the blood in the capillaries than usual. Another condition which is usually attributed to venous stasis is that of the *clubbed fingers*. The finger loses its tapering form, and the last joint becomes thickened, especially from palmar to dorsal surfaces, but also transversely. The nail is unusually convex from base to tip, and seems to curve over the end of the finger. The appearance is more pronounced on account of the wasting of the rest

of the finger. It is a common feature of phthisis, but not peculiar to it. The same change may be seen in the toes. (*See* Hypertrophic Pulmonary Osteo-arthropathy.)

**Complications.**—It will not be necessary to do much more than enumerate the various complications which occur in phthisis, as they are described in different parts of this volume under the particular organs concerned. Many of them result from the formation of tubercles in other parts of the body. They are more common in the long-standing chronic cases.

*Respiratory Organs.*—Tuberculous disease of the larynx (*see* p. 203) is common in phthisis, and considerably aggravates the patient's distress. In exceptional cases the symptoms of laryngeal mischief are obvious before those of the pulmonary lesions, but it is doubtful if the tubercle is ever actually deposited in the larynx before it is formed in the lung.

*Pleurisy.*—This is so common as to be almost a part of phthisis. An old phthisical lung is usually adherent to the chest by a thick fibrous layer, and pleuritic lymph forms nearly always over tuberculous lesions that approach the surface. Sometimes liquid is poured out, and this is mostly a sero-fibrinous effusion, less often an empyema.

*Pneumothorax.*—This occurs in a very small proportion of cases, but phthisis is by far the commonest cause of pneumothorax. If the liquid contents of a cavity escape into the pleura, an acute pleurisy may be set up, effusion takes place, and there will not only be a pneumothorax, but a *pyo-pneumothorax*, or less often a *hydro-pneumothorax*. Pneumothorax itself does not usually increase the risk; in fact, some cases have shown remarkable improvement as the result.

*Circulatory System.*—The heart becomes smaller in chronic phthisis, as has been shown by X-ray measurements of its width carried out by means of the ortho-diagraph. X-rays may also show a rather elongated appearance. In acute cases with anæmia there may be fatty degeneration. In chronic fibroid forms the right ventricle is dilated and thickened to a moderate extent. The arterial erosions and small aneurysms of the pulmonary artery in cavities in the lungs have been already mentioned.

*Femoral thrombosis* is frequent in the last days of patients suffering from phthisis; it is more common on the left side.

*Anorexia, indigestion, nausea,* and *vomiting* are common accompaniments of phthisis more or less during its whole course. A capricious appetite and a distaste for fat in every form have been noticed even before definite symptoms in the lungs. In the last stages sickness or loathing for food is so marked that it is one of the chief difficulties of doctor and nurse to get the patient to take anything at all. The most extraordinary things are fancied by the patient at one moment only to be rejected directly they are put before him.

*Diarrhœa* is common in late stages; it may be due simply to catarrhal conditions, to tuberculous ulceration of the ileum, or to lardaceous disease. The stools are variable, sometimes yellow in colour, undigested, and containing a little mucus or blood. Large hæmorrhages are not often seen.

*Peritonitis* is very rarely the result of perforation of a tuberculous ulcer. More often it is due to tubercles in the peritoneum, but it is not a common complication.

*Lardaceous disease* of the liver, spleen, kidneys, and intestines was found in 20 per cent. of fatal hospital cases some years ago; probably it is less now.

*Fatty liver* is somewhat more frequent.

Tuberculosis of the *epididymis* and *vesiculæ seminales*, and of the *uterus* and its *appendages*, occasionally occurs, but these lesions do not usually form prominent complications at the bedside. *Fistula in ano* is occasionally associated with phthisis.

*Addison's disease* of the suprarenal capsules is very rarely associated with phthisis.

*General tuberculosis* is one of the fatal complications of phthisis. The meninges,

lungs, liver, spleen, and kidneys are the organs commonly affected (*see* p. 129), Tuberculous meningitis is a cause of death in a small number of cases. Its course is often very rapid ; perhaps the earlier indications are lost in the general symptoms already present.

*Bones and Joints.*—Tuberculosis of joints, caries of ribs with subcutaneous abscesses, caries of the spine with psoas abscess, and other similar lesions may co-exist with the pulmonary disease.

**Fatal Termination.**—Death takes place in the following ways : exhaustion, hæmoptysis, meningitis, perforative peritonitis, and uræmia, if the kidneys are lardaceous.

Exhaustion, or, to speak in more modern terms, the disturbance of all the functions and finally of the circulation, from poisoning by the toxins of tuberculous and pyogenic organisms, and from deficiency of lung substance, accounts for the larger number of cases. The wasting is explained by the pyrexia with its increased metabolism, by the expectoration, diarrhœa, and the deficient supply of nutriment from anorexia, nausea, and vomiting.

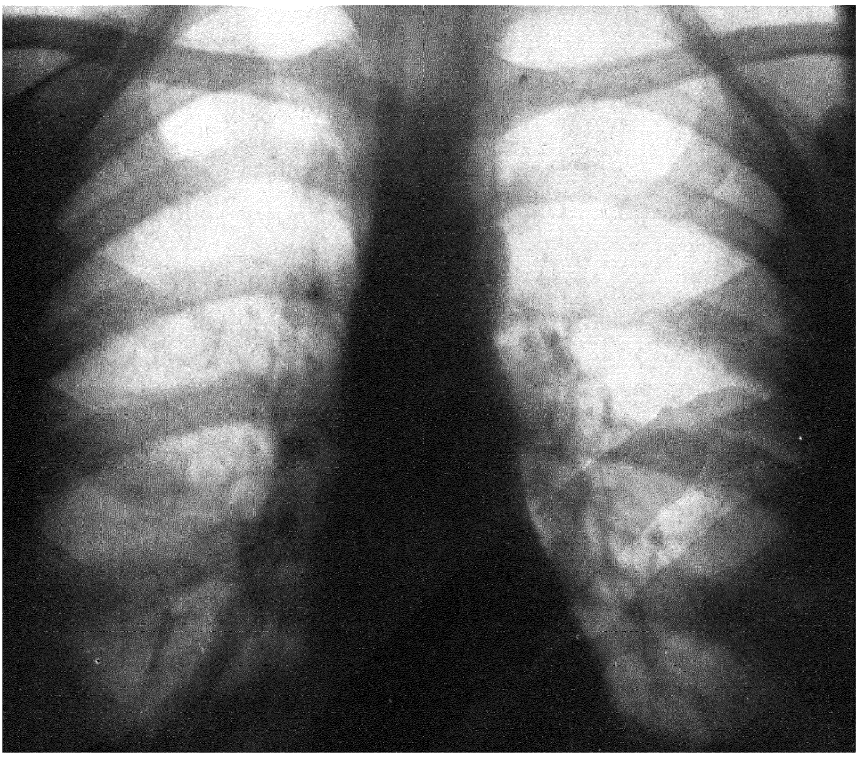
**Other Forms of Pulmonary Tuberculosis.**—*Pneumonic Phthisis (Scrofulous Pneumonia).*—This begins very much like an attack of acute pneumonia, with pain in one side, high fever, chills, and night sweats, cough and expectoration. The physical signs also are those of pneumonia ; but they are most marked at the apex, and spread downwards. Dulness, bronchial breathing, and bronchophony are accompanied by coarse mucous râles, consonating râles, and loud clicks. Often the condition is much more marked in one lung than in the other. The mischief extends rapidly : the pyrexia is severe, there are profuse sweats, appetite is entirely lost, and prostration becomes extreme. The indications that the lung is breaking down are more and more marked ; the temperature assumes an intermittent type ; the sputum is purulent, and contains *débris* of lung tissue. The illness is often fatal in the course of from five to twelve weeks, either by exhaustion or by hæmoptysis, which is always abundant if it occurs at all.

*Hilum Phthisis.*—This is a chronic form of disease. The patient, usually a child, sometimes an adult, complains constantly of a tired feeling. The temperature is often slightly raised, being perhaps 99° F. in the morning and 100° F. in the evening (rectal). There is an impaired note over the root of the lung behind, on one or both sides, between the vertebral borders of the scapulæ, and Krönig's isthmus may be diminished. Râles are seldom heard. In adults there may be no alteration at all in the percussion note. In contrast to these very indefinite physical signs, the X-rays show increase in the normal root shadows, probably due to lymphatic infiltration, and an extensive fine mottling all over the lungs. It is only when the process, which is at first central, reaches the surface, that râles may be heard over the chest in various situations, particularly over the outer surfaces of the lungs.

*Fibroid Phthisis.*—This is a very chronic form of apical phthisis, often affecting one lung only. Clinically the case is distinguished by the evidences of contraction of the diseased lung; the chest is sunken; the heart is displaced to the affected side ; the opposite lung may extend its resonant area in the same direction ; the spleen and stomach if the left lung is diseased, or the liver if the right, may be drawn far up into the chest. The physical signs of cavities are chiefly at the apex, as in other cases of phthisis ; but impaired resonance, bronchial breathing, and bronchophony are perhaps present over the whole of the affected lung. If the other side is involved, it is only at the apex. There is often not much cough or expectoration ; there is no sweating, and the temperature is normal. In the later stages there may be failure of the right side of the heart, with shortness of breath, dropsy and cyanosis.

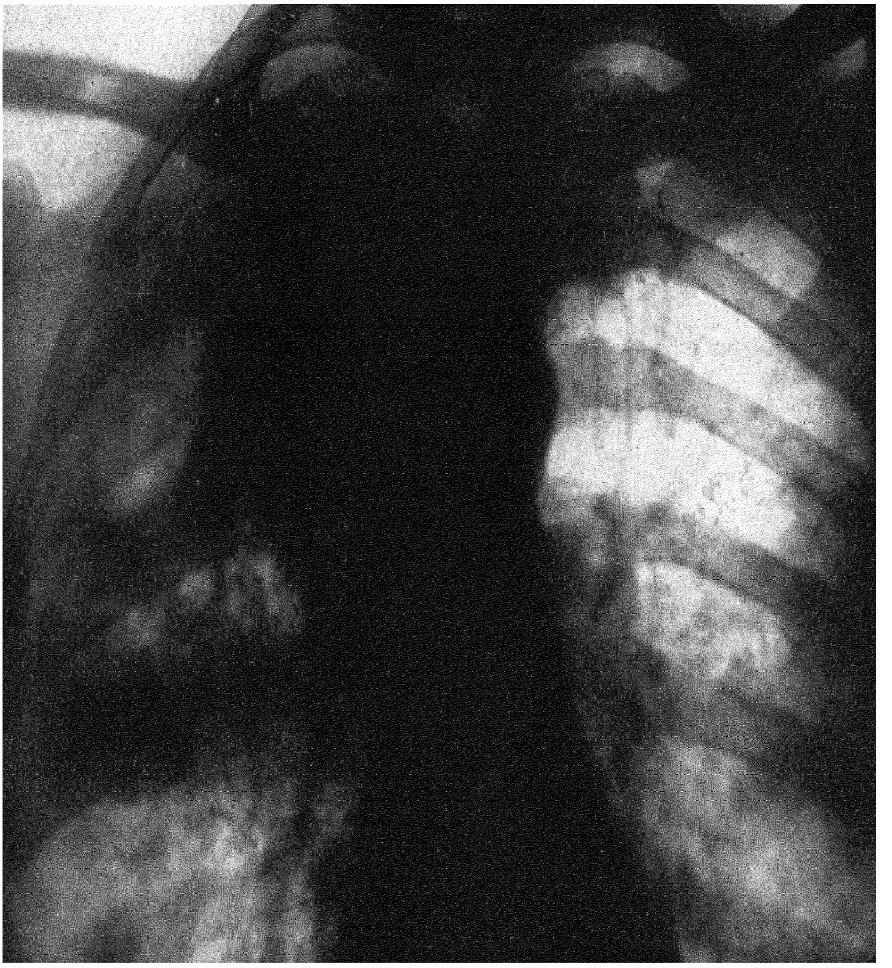
**Diagnosis of Phthisis.**—In advanced cases the symptoms and physical signs make the diagnosis obvious. Except in the earliest stages, and in periods

# PLATE I.



Radiogram of Upper Part of a Normal Chest. The root shadows, which consist of the larger bronchi, vessels and lymphatics, are shown on each side. Outside this, the lung is clear. However, in a good plate a very finely mottled or marbled appearance can be seen in the intercostal spaces outside the roots due to the healthy lung tissue. This is scarcely shown in the reproduction. The central shadow due to the heart and great vessels should also be examined. Above there is a bulge due to the aortic arch. This should be compared with Plate IX., in which an aneurysm is depicted. Below the aortic arch on the left are the pulmonary artery, left auricular appendix (not distinguishable from the surrounding structures), and left ventricle; on the right, the superior vena cava, and right auricle. The right diaphragm is higher than the left. (From a plate taken by Mr W. Lindsay Locke.)

[*To face p.* 257.



**Radiogram** showing Right Apical Phthisis. A cavity is seen, and round it there is dense infiltration. The part of the lung below has a mottled black and white appearance, due to extension of the tuberculous process. At the tip of the left apex there is also some tuberculous infiltration: but the lung beneath this is clear. Lower down, again, in the lower lobe, there are shadows suggesting further infiltration. Extending upwards from the left root are two lines representing the walls of a large ascending normal bronchus. The shadow at the left root is also perhaps slightly more opaque than normal (compare Plate I.). (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p* 257.

## PLATE III.

Radiogram showing an Extensive Tuberculous Infiltration of the whole of the Right Lung and of the Lung in the neighbourhood of the Left Root. The process is of the hilum variety. On the right side there is no special infiltration of the apex, while the left apex is clear. The base of both lungs just above the diaphragm is clear. The mottling on the right side suggests that the lung contains numerous active foci separated from one another by more or less healthy tissue. On the left side the infiltration is denser. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 257.

of quiescence or arrest, the sputum will show *tubercle bacilli*. For their detection they require to be stained, and to be submitted to a microscopic power of 350 or 400 diameters. The Ziehl-Neelsen method of staining is now commonly employed. A cover glass is smeared over with a thin layer of sputum, which is dried by gently warming and then fixed by passing the slide three times through the flame of a spirit lamp. A solution of 1 part of fuchsin in 10 parts absolute alcohol is added to 100 parts of a 5 per cent. aqueous solution of phenol. This is heated till the steam rises; and the cover glass is floated on it, film downwards, for three or four minutes, rinsed in water, and immersed in a 20 per cent. solution of sulphuric acid until it is decolorised. It is then washed in water, and counter-stained by means of a nearly saturated watery solution of methylene blue, again washed quickly in water, dried, and mounted in xylol balsam. The sputum may also be injected into a guinea-pig for diagnostic purposes, and the animal is examined for miliary tuberculosis after three weeks.

The fragments of *elastic tissue*, which are present with sputum in the later stages, may be seen with the microscope either by teasing out the little rough nodules which are sometimes found, or by boiling the sputum in liquor sodæ for twenty minutes, and examining the sediment. Elastic tissue is found in any case where there is active destruction of lung tissue.

It is much more difficult to be certain of the presence of phthisis in its early stages. Apart from the more obvious symptoms of cough, expectoration and wasting, which may all be absent, valuable indications are obtained from (1) a feeling of tiredness complained of by the patient; (2) changes in the rectal temperature: in very early cases the rectal temperature in the early morning is lower than that of the average normal subject, *i.e.* below 97° F. Later on, at 7 a.m. it is constantly above 98·4° F., and in the evening above 99·5° F. an hour after exercise. It must be remembered that in women the rectal temperature is normally slightly raised for a week before or occasionally after the beginning of menstruation. (3) There may also be a history of pleurisy.

The first *physical signs* of any value in diagnosis are impaired resonance at one apex with diminished vesicular murmur, or diminished vesicular murmur with râles on inspiration or on coughing. The *Röntgen rays* may give valuable help in the diagnosis of hilum phthisis, but in apical phthisis the physical signs are often well marked by the time a dark area at the apex is seen (*see* Plates I., II. and III.). However, there may be early limitation of the movement of one diaphragm. Further there may be increased mottling spreading out from the roots, but not extending up to the apex, even when early apical signs are alone present.

Recent observations on the diminution in the vital capacity of physically unfit persons point to this method as being valuable in the diagnosis of early phthisis (*see* p. 189).

The various *tuberculides* when recognised on the skin suggest some internal tuberculous lesion (*see* p. 951).

*Tuberculin in Diagnosis.*—Koch's *old tuberculin* is employed for purposes of diagnosis in three ways at the present time: (1) subcutaneously in order to produce a general *reaction;* (2) by application to the conjunctiva (Calmette's *ophthalmic reaction*): (3) by inoculation (Von Pirquet's *cutaneous reaction*).

The *reactions* produced by the introduction of tuberculin into the system are of three kinds: A *local* reaction is the inflammatory change which takes place at the site of injection. It is seen in Von Pirquet's test. A *general* reaction consists of headache, malaise, rise of temperature to about 101°, coming on twelve hours after the injection, and sometimes nausea and vomiting. It occurs in test (1). A *focal* reaction is a change occurring in the focus of tuberculous disease, *e.g.* in the lung, and consists of subjective signs, such as dyspnœa, cough, or pain in the chest, and objective signs, such as increased quantity of sputum, with perhaps the appearance of blood and increase of the physical signs, such as

dulness and râles. As a rule the local reaction is induced by a smaller dose than the general, and the general by a smaller dose than the focal reaction. It is obviously undesirable to produce a focal reaction.

1. For the subcutaneous method the patient should be free from fever. The tuberculin, O.T., is diluted with a 0·5 per cent. solution of carbolic acid; and an amount equal to ·001 c.c. (or 1 c.mm.) is injected into the back or buttock. If no rise of temperature takes place, double the dose is injected on the day but one after; if there is again no reaction, the dose is increased to ·005 c.c. (or 5 c.mm.) given three days later; and if this gives a negative result, a dose of ·01 c.c. (or 10 c.mm.) is given; and failing a result, tuberculosis is excluded. For children and weakly persons the initial doses may be half of those given above. In very advanced cases reactions are not always obtained, probably because the patient has lost the power to react.

2. A few drops of a 0·5 per cent. solution of tuberculin (precipitated in alcohol and redissolved in sterile distilled water) are allowed to fall on the conjunctiva near the inner angle of the eye, and the eyelids are kept apart for a few moments. If the subject is tuberculous in any part of the body whatever, the conjunctiva of the lower lid and the caruncula will begin to redden in three hours, the injection increases in six hours, the caruncula is swollen, tears flow freely, and the eye is covered with slight exudation. The reaction reaches its maximum between the sixth and the thirteenth hours, and subsides entirely in two, three, or more days. This is considered to be a good test for active tubercle.

3. In order to test for the cutaneous reaction, four solutions of tuberculin are used, of the following strengths: 1, 4, 16, and 64 per cent. The skin is inoculated with these solutions in four places, in the same way as in vaccination for small-pox (*see* p. 42); a fifth inoculation is made with some sterile distilled water as a control. Reaction is shown by hyperæmia with some œdema within twenty-four hours; in forty-eight hours the spot is reddened, slightly raised circular and palpable; on the third day the spot begins to fade, and on the fourth day it is gone. If the test is positive, the size of the raised spots increases with the strength of the solution, in such a way that as the strengths are 1, 4, 16 and 64, *i.e.* in geometrical progression, the diameters of the spots are in arithmetical progression, for instance, as 1, 2, 3, and 4. In general a reaction of some kind is obtained with any one who has had a tuberculous focus in the body at some time, even if it has completely healed. The presence of some active focus is suggested if the size of the spots increases regularly with the strength of the solution when the test is carried out quantitatively as above. A satisfactory reaction is not always obtained in very advanced stages of the disease or in debilitated subjects. The *old tuberculin* reacts equally well for bovine as for human tubercle.

*Diagnosis by the Complement Fixation Reaction.* -The use of the Bordet-Gengou reaction for diagnosing tubercle, like the Wassermann reaction for syphilis (*see* p. 114), has recently given good results according to some workers. An emulsion of living tubercle bacilli is used as antigen. This, when mixed with serum from a tuberculous patient, fixes complement and so prevents the hæmolysis of red cells by a sensitised hæmolytic serum (McIntosh and Fildes).

Before *hæmoptysis* is regarded as indicative of tubercle it must be clearly made out that the blood really comes from the lung, and not from the stomach; the descriptions of a patient are often very unsatisfactory or misleading. Blood from the lungs should be coughed up, and should be bright red and frothy; it is often preceded by a tickling in the throat, and not by the sense of nausea, which is more common with hæmatemesis. Further, if the blood has come from the lungs, the patient will usually spit up blood mixed with sputum for twenty-four or forty-eight hours after the free hæmorrhage has taken place. *Anæmic* girls often speak of finding blood on their pillows on waking. This is,

no doubt, from the gums or cheeks, is generally diluted with saliva, and may be mixed with buccal epithelium. *Malingerers* may produce blood in a similar way. In *purpura*, blood may be expectorated actually from the lung, but the cause will be readily distinguished by the associated symptoms. Hæmoptysis is frequent in mitral stenosis in young people. Although phthisis, alcoholism, and cirrhosis of the liver are often associated, bleeding from the lungs may occur in the course of *cirrhosis* quite independent of tuberculosis, and it is not infrequent, especially in older people, as the result of abnormally *high arterial pressure*.

Phthisis is sometimes masked by *bronchitis*. The accentuation of the physical signs at one or other apex is important, as well as the history of the illness, hæmoptysis if present, and the detection of bacilli in the sputum. The possible confusion with *bronchiectasis* has been mentioned (*see* p. 224). *Empyema* is accompanied by fever, sweating and emaciation; and if it bursts through the lung, there will be cough and purulent sputum. The physical signs will usually be at the base.

**Prognosis.**—In scarcely any disease are cases so different as they may be in phthisis. Discovered in its earliest stage, it may be cured so completely that no trace of it can be detected clinically; it may in other cases be fatal in a few months; or it may last ten, twenty, or even fifty years with obvious physical signs and symptoms throughout. The variable elements are the virulence of the infection and the capacity for resistance on the part of the patient, and it is difficult to estimate which will gain the upper hand until the patient has been under observation or treatment for some time. Improvement may occur at once under treatment, or, at any time in the course of the illness, the protective powers of the patient may be so increased as to cause arrest of the process for a long period. And in no case should hasty predictions be made as to when the end will take place. However, certain factors are known to influence the prognosis. Thus the after-histories of phthisis patients who had been discharged from King Edward VII. Sanatorium at Midhurst showed from three to seven years after discharge 15·6 per cent. of deaths in Stage I. (Turban-Gerhardt), 38·0 per cent. in Stage II., and 70·1 per cent. in Stage III. When tuberculous laryngitis was also present the figures were 42·9, 63·3, and 78·3 per cent. respectively. This complication made the prognosis much worse, especially in the early stages (**5**). The presence of other complications is also unfavourable. The prognosis is much better if as the result of treatment either there is no sputum or tubercle bacilli cannot be found in it. The prognosis also depends on the amount of care that the patient can take of himself after discharge. It is in general much worse in the case of the industrial classes than in the well-to-do. Pregnancy has an unfavourable influence on the disease, a fact which is first noticed after the confinement.

**Prevention.**—Fresh air and good food, as recommended for a patient with phthisis, are advisable for children born of phthisical parents. It is by such means that they can best increase the resistance of their tissues against the bacillus. Phthisical persons about to marry should be informed of the risk that their offspring may develop the disease. There is also a definite risk to the healthy partner from infection. With a phthisical patient living in a house, the danger of infection to the other healthy occupants must be guarded against. The patient should sleep in a room by himself. Underclothes and bed linen should be scalded before being washed. There should be ample ventilation in the house. The sputa should in all cases be ejected into antiseptic fluid (5 per cent. carbolic acid solution), and they should finally be rendered innocuous by exposure to boiling water for ten minutes. Tuberculous mothers should not suckle their infants.

**Treatment.**—In the treatment of phthisis chief reliance must be placed on the improvement of the body and its tissues in every possible way, so that it may

be enabled to resist the inroads of the disease, or rather that the tissues may become less fitted as a soil for the bacillus, and less readily excited to inflammation in its different forms. This indication is met by providing for the individual perfect hygienic surroundings, fresh air, and sufficient good food. But the most important treatment for an early case is absolute rest until there is no longer any pyrexia, and for chronic cases one day in bed every week is a golden rule. The patient should as far as possible lead an easy life, free from care. In a woman pregnancy should not be permitted, and should be terminated at an early stage if it has begun.

Treatment with the above objects should be taken *as soon as possible* after the evidence of tuberculous infection is confirmed. The methods available may be divided into two groups: *general*, namely, sanatorium treatment with or without change of climate and possibly followed by a sea voyage; *specific*, namely, tuberculin treatment, graduated labour, and artificial pneumothorax. Symptomatic treatment must also be carried out as required.

*Sanatorium Treatment.*—The first object of sanatorium treatment is to teach patients enough about the disease, so that they may live the rest of their lives under conditions favourable to recovery. The second object is to provide for a minimum period for three months, which however ought to be considerably longer, conditions which favour the beginning of their recovery. Patients are kept warm but not too warm, since cold stimulates metabolism; they are sheltered from rain and cold winds, and live night and day practically in the open air. The day is spent as much as possible outside the building, either in the open or in shelters screening the patient from the wind, and when possible exposed to the sun. The bedrooms and day rooms are thoroughly ventilated, and the rooms are constructed so as to prevent any accumulation of dust. The patients are well fed, having three meals daily of plain but varied food, while care is taken to see that they eat a sufficient quantity of it. Exercise is only permissible when the morning temperature is normal, and the evening temperature not above 99·5° (rectal); the exercise will probably raise the temperature for a time; if it does not fall to a normal value after an hour's rest, the exercise should be stopped. The patient begins by taking a slow walk, and the amount of exercise is gradually increased. In any case, he rests one hour before meals and one hour after; and violent exercise and indulgence in exciting games or recreations are prohibited. The clothing of the patient must be suited to the temperature of the air. Treatment on this system has benefited many patients temporarily, but it must be submitted to for very much longer than the period of three months for which it is sometimes prescribed. In fact, a carefully controlled series of results among poorer class patients showed that three months' sanatorium treatment was not at all beneficial, presumably because in his normal surroundings the patient had become more or less adapted mentally and physically to fight the disease, but after a stay in a sanatorium he lost this adaptation and did not regain it on going back to his ordinary surroundings and work (Ward). At the same time a short stay will be of value to teach the patient the principles of his treatment, in order that he may at his own home carry them out as far as circumstances will permit. In particular, rest, ventilation, the collection and destruction of sputum and plenty of nourishing food should be insisted upon, and the patients should learn how to take their own temperatures and stay in bed so long as it is raised. In this connection the tuberculosis dispensaries are valuable.

A development of the sanatorium is the tuberculosis *colony*, where patients can live with their families more or less permanently under favourable conditions in the country, following an occupation that partly covers the cost of the treatment (**21**).

*Change of Climate.*—The places usually selected are South Africa, New Zealand, high altitudes in Switzerland, such as Davos, Wiesen, and Maloja, or the English east coast. The patient may in one or other of these places obtain a dry, cool,

and bracing air, which he can enjoy for several hours daily outside the house, without the risk of catching cold; and he may spend the whole of the winter, avoiding the cold, damp, and fog of that season in the greater part of England, and returning to his home in the summer, when the weather is more tolerable. With the advent of winter he must again seek the climate which he has found suitable. A dry climate is especially desirable for phthisis patients who have a secondary bronchitic infection. In more advanced cases milder climates may be beneficial, such as that of the Riviera or the English south coast. "Sun-bathing," or heliotherapy, or treatment with ultra-violet light, such as is carried out in surgical tuberculosis, may be used as an additional stimulus in a case which is becoming cured, but in which the progress is slow. It should only be very slowly begun by gradually increasing the surface exposed, beginning with the extremities. Sun-burn must be avoided. The aim should be to produce an erythema, which is followed by pigmentation. Where the disease is active, with slight fever on exertion, hæmoptyses, etc., heliotherapy should not be used (**22**).

*Tuberculin Treatment*.—This aims at immunising the body against tuberculous infection by the production of anti-bodies.

Apparently perfectly healthy persons are not affected by the injection of tuberculin: but the previous presence in the body of the tubercle bacillus renders the tissues sensitive, and to them the tuberculin acts as a poison, causing the reactions already described; hence its use in diagnosis. By the repeated injection of tuberculin and the continuous formation of anti-bodies tolerance is gradually established, so that larger and larger doses can be borne without reaction; and finally the body may become immune not only to tuberculin introduced from without, but to the tuberculin toxins produced by the pulmonary lesions themselves (auto-tuberculin).

Koch's new tuberculin, or "T.R." (tuberculin Ruckstand), is perhaps the tuberculin most generally employed. It consists of triturated human bacilli deprived of all soluble matter and forming an emulsion of which 1 c.c. contains the active bacterial matter of 10 milligrammes of tubercle bacilli. As extremely small doses may have to be given, the tuberculin requires dilution by a ½ per cent. solution of normal saline with ·25 per cent. of carbolic acid.

The present practice with regard to the dosage of tuberculin is to keep it just below the point at which a *reaction* is obtained. It should only be employed for afebrile cases, and the temperature should be carefully observed all through the treatment as a test of the reaction.

The initial dose varies with different physicians, but it is safer to give a very small dose at first, such as ·001 c.mm. of whatever preparation is used, double this quantity, ·002 c.mm., in three or four days, double this again, or ·004 c.mm., after a similar period, and so at similar intervals, or twice a week, until a slight reaction, either local or general, is observed, namely, some thickening at the site of injection, or fever, headache, malaise, etc. These symptoms generally occur within twenty-four hours and subside in about the same time. After three or four days again the last dose may be repeated, when the reaction, if it occurs, will be less, and soon the same dose fails to produce any reaction at all; that is, tolerance to that quantity is established. The dose should now be increased by much smaller additions every half-week until a reaction again occurs. In time another reaction will result, and the dose is again maintained at the same figure until tolerance is established, and then increased on the same system, until as much as 10 c.mm. is given at a dose. With the larger doses the interval may be a week instead of three or four days. The final dose adopted by different authors is as variable as the initial doses, but the maximum dose which can be safely given is considered by some to be 0·1 c.c.; others would not go beyond ·075 or ·05 c.c. This final dose may be repeated at longer intervals for some time. The duration of the treatment is from six months to eighteen months or two years.

It can only be proved that tuberculin is of value by observing a series of cases treated by this means and a similar control series treated at the same time without tuberculin. In two series consisting respectively of eleven and twelve such afebrile cases, rather better results were obtained with the tuberculin-treated cases than with the others; but it is impossible to draw conclusions from so small a number (Batty Shaw). Many physicians are strongly opposed to its use, and think that as much harm as good may be done by it. In some important institutions and sanatoria it has been completely abandoned after full trial.

*Graduated Labour.*—Provided the temperature is normal throughout the twenty-four hours, carefully graduated exercise, as recommended by Dr. Paterson, may be beneficial. Each case must be treated on its merits, and the work at Frimley has ranged from a half-mile walk daily to six hours' spade-work or load-carrying in the day. Beginning with the lightest exercise, the patient is gradually put to heavier work. However, it must be remembered that it is rest and light work rather than heavy work that a phthisis patient really requires, and it is less dangerous to give him too little than too much exercise.

*Artificial Pneumothorax.*—This treatment was described in 1821 by Carson, of Liverpool; but it is only within the last few years that it has been widely adopted. The chief reason that prevents tuberculosis of the lungs from healing is that the tissue is kept expanded by the negative pressure in the thorax, and any cavities that form cannot close up. If air is injected into the thoracic cavity, the lung will collapse, so that help is given to the natural healing processes of the body.

The operation is especially suitable in cases in which one lung is extensively diseased and the other is relatively sound. In the past it has been used chiefly for advanced cases, but the tendency at present is to use it more widely; in fact, for any case of one-sided disease where the process is extending. There is statistical evidence that the treatment is beneficial (Saugman). Hæmoptysis is a special indication for immediate induction of a pneumo-thorax on the side of the hæmorrhage.

The apparatus consists essentially of a trocar and cannula connected on the one hand with a water manometer, and on the other with a receiver filled with air, so that a measured quantity can be passed into the chest. The skin and deeper tissues are anæsthetised with 0·5 per cent. novocaine. The puncture is made either directly or after a slight incision through the skin; and the fact that the needle is in the pleural cavity is recognised by the oscillations of the manometer fluid, which, of course, shows a negative pressure. These oscillations should be from 4 to 6 cms. of water; and if the oscillations are no more than 1 or 2 cms., the needle is probably not in the pleura. When it is certain that the needle is in the pleural cavity, the air is driven in to the extent of 300 c.c. to 500 c.c., or until the manometer oscillates about the zero point. After a week more air may be injected.

Refills should be given according to the absorption of the gas, in amounts from 500 to 800 c.c. and at weekly or fortnightly intervals to begin with, though later on the intervals may be longer, as absorption will be less. They should be continued for three years or longer. It is usually unnecessary to anæsthetise the skin for the refills. The final pressure should never be greater than + 10 cms. of water. The chief accident in the past has been due to air embolism; but this cannot occur if the needle is really in the pleura, and that is the object of having the manometer. To be on the safe side, it is perhaps advisable for a first injection to use oxygen. When some 200 c.c. have been passed in safety, it is removed, and air is substituted for it. Quite rarely unpleasant effects are noticed which are ascribed to the pleural reflex, viz. dyspnœa and palpitations, pallor, and sometimes even loss of consciousness. Sometimes the mediastinum is weak and readily displaced to the opposite side. There may be discomfort and dyspnœa. The condition can easily be diagnosed by X-rays, and here emphasis should be laid on the necessity for screening every case before and throughout the treat-

ment. "Ballooning" of the pleura towards the healthy side sometimes occurs; but if without symptoms, it does not matter. About half the cases develop a clear effusion during treatment. When this begins, treatment should be less active, as the pleura is inflamed. The fluid need not be removed unless the temperature rises, and if removed, it should be replaced by gas. An extension of the pneumothorax treatment suitable for quite early cases consists in admitting into the pleural cavity quite a small volume of air, which is found to collect round the infiltrated part of the lung and cause a partial collapse, while the healthy part remains expanded. A partial pneumothorax can be induced on both sides. Refills must, however, be carried out frequently (**24**).

Where a complete one-sided pneumothorax has been successfully induced, 70 per cent. of the patients have succeeded in getting back to work, but the main difficulty in the treatment is the existence of adhesions, which hinder collapse, and in this case there are only 33 per cent. of successes. It is impossible to diagnose adhesions until the attempt at injection is made. Sometimes they will break down of themselves after repeated refills; but when this is not the case, attempts have been made to divide them. They are localised by introducing a thoracoscope into the pleural cavity, and then cauterised by a platinum wire inserted through another cannula. This method is not suitable where there are large surface adhesions, and for this case *thoracoplasty* has lately been carried out with success. In this operation the hinder portions of the ribs are removed extrapleurally from just in front of the angles to as near the transverse processes of the vertebræ as possible. In complete thoracoplasty parts of all ribs are removed from the first to the tenth; but in cases where the disease is limited to the lower lobe a few of the upper ribs may be left intact. Light ether anæsthesia is to be preferred. Forty-five per cent. of cases have become subsequently fit for work after this operation (**25**). In *apicolysis* the parietal pleural round the apex has been separated from the ribs and pushed in, and the space has been filled with a graft of subcutaneous fat from the abdomen or plugged with gauze. When there is extensive apical cauterisation thoracoplasty has been combined with apicolysis.

Various remedies have from time to time been considered specific in phthisis, but without much evidence. Among these may be mentioned creosote 10 to 100 minims in capsules or dissolved in cod-liver oil, guaiacol 10 to 60 minims in capsule, guaiacol carbonate 20 to 90 grains in cachet or wafer, daily; antiseptic drugs by inhalation (*see* p. 224); pneumosan, or amyl-thio-trimethylamine, a bluish purple volatile liquid, which is injected into a muscle, *e.g.* the deltoid, twice a week, or every other day, or even daily; garlic juice (succus allii); "nascent iodine," thought by some to be produced by giving by mouth potassium iodide with meals, and chlorine water between meals; sodium morrhuate.

**Symptomatic Treatment.**—During treatment in a sanatorium drugs are avoided as much as possible, and it is generally found that the symptoms disappear with the improvement of the patient. It is in all cases essential that the digestion should not be upset by the medicines administered.

*Cough.*—The rapid diminution of cough has been constantly observed in the open-air treatment. Only if it is painful, frequent, or keeps the patient from sleep, it may be treated by small doses of opium or morphia in combination with expectorants; for instance, tr. camph. co. with tr. scillæ, or liq. morphinæ hydrochlor. with vin. ipec., or syr. papav. with camphor water, or a few minims of chlorodyne. A morning cough, which gets rid of accumulated secretion, may be usefully promoted by a little ammonium carbonate. The patient should resist coughing as far as possible. If it is preceded by a tickle in the throat, painting the lingual tonsil with iodine may be effective.

*Night Sweating.*—This can be generally checked by 1 minim of liq. atropinæ sulph. given in a little water at night, or 2 or 3 grains of oxide of zinc in a pill, with $\frac{1}{6}$ grain of extract of belladonna. Arseniate of iron ($\frac{1}{5}$ grain), or picrotoxine ($\frac{1}{50}$ gram), or tincture of nux vomica may also be used. Night sweating usually

means too warm clothing at night or excessive auto-inoculation by the patient with his own toxins—too much exercise, for instance—and reflects on the management of the case by the physician.

*Hæmoptysis.*—The patient should be kept in bed in the semi-recumbent posture; but he should be allowed to change his position when he likes. The most effective hæmostatic is the intravenous injection of 20 c.c. of a 15 per cent. solution of calcium chloride. A simple plan is to give 5 to 15 grammes of sodium chloride by mouth. Sodium bromide may be substituted if the patient is nervous. Bleeding often ceases after inhalation of a few drops of amyl nitrite. In some dangerous cases of hæmoptysis, the induction of pneumothorax has been successful. Hypodermic emetine injections have been successful when the bleeding is slight and continuous (Flandin). The traditional method of treatment is to place an ice-bag on the chest and inject morphia; but this has the disadvantage that the blood is kept stagnating in the lungs and so helps to cause an acute spread of the tuberculous process. It has been found that hæmoptysis is largely independent of the patient's movements (**26**).

*Diarrhœa.*—For this one must carefully regulate the diet, and use the vegetable astringents, mineral acids, opium, sulphate of copper in $\frac{1}{4}$-grain doses, or carbonate of bismuth.

*Pleuritic Pains* are frequent, and are often relieved by antiphlogistine or thermogene wool or by painting the surface of the chest with tincture of iodine. Anodynes internally may be necessary. Many believe that pleuritic effusion delays the progress of the disease in the corresponding lung, and postpone tapping until pressure is extreme. An empyema requiring evacuation may be aspirated.

*Excessive Expectoration.*—This condition may be treated by antiseptic inhalation. Eucalyptol and thymol may be used, or Lees' solution, consisting of carbolic acid 2 parts, creosote 2, tincture of iodine 1, spiritus ætheris 1, and spiritus chloroformi 2. Six or eight drops of this are poured on the inhaler every hour. The patient wears a "respirator" over the mouth or nose for one, two, or three hours at a time, and breathes the vapour into his lungs. Drainage with the head down and feet raised may be useful (*see* p. 224).

## SYPHILIS OF THE LUNG

Apart from the ulcerations of the bronchi, with resulting stenosis, which have been shown to be due to syphilis, the lung tissue itself may exhibit the effects of the disease in various forms. One is that of the ordinary *gumma*, which is extremely rare in adults, though more common in infants, and gives rise to no recognisable clinical symptoms. Another is the so-called *white pneumonia* of syphilitic infants. The lungs are enlarged, white, dense, and firm; their section is smooth and opaque; they are sometimes resistant, at others easily broken down. The microscope shows a diffuse cellular inflammation of the lung, with thickening of the alveolar walls, and desquamation and fatty degeneration of the pulmonary epithelium. This condition may affect the whole lung, or one part may be uniformly altered, while the other contains only isolated areas. In another variety the alveoli are lined by cubical epithelium, and the connective tissue is replaced by a fibrous stroma infiltrated with cells from the cubical alveolar epithelium. Spirochætæ have been found in these cases. As these lesions are found chiefly in still-born children, they have but little clinical importance.

The extent to which syphilis may affect the lung in adults otherwise than by gumma has been the subject of much discussion. Destructive changes with bronchiectatic cavities may take place as a result of bronchial or tracheal stenosis; and diffuse fibrosis, sometimes with cavities, has been found in syphilitic subjects, associated with marked pulmonary symptoms. The lesions occur especially at the root and central part of the lungs, extending outwards along the bronchi and vessels, and the cavities are often connected with obstructed bronchi. But these

conditions are rare, and most cases of destructive disease of the lung occurring in syphilitic persons are due to tuberculosis.

## OTHER INFECTIONS OF THE LUNG

Besides the different forms of infective inflammation of the lung described under the heads of pneumonia, phthisis or tuberculosis, and syphilis, there are lesions induced by the organisms of other infectious diseases, glanders, plague, anthrax, actinomycosis, and aspergillosis, of which accounts have been given.

## POISONING BY IRRITANT GASES

In April to May, 1915, the Germans used cloud gas attacks by liberating chlorine from cylinders so that it drifted with the wind. In December, 1915, phosgene ($COCl_2$) was also used. In the autumn of 1916 lethal gas shells were used instead of cloud gas attacks. They contained phosgene, chloropicrin ($CCl_3NO_2$) and other *suffocative* gases. In July, 1916, "Yellow Cross" shells, containing "mustard" gas, which is dichlorethyl sulphide ($(C_2H_4Cl)_2S$), were used, and shortly afterwards "Blue Cross" or "Red Cross" shells, containing organic arsenic compounds, such as diphenylchlorarsine. *Lachrymatory* gases were also used in shells, *e.g.* xylyl bromide and chloropicrin.

*Suffocative* gases acted chiefly on the lung alveoli, causing acute œdema, thrombosis of the capillaries and disruptive emphysema.

**Symptoms.**—There was first of all irritation of the eyes, which, as a rule, quickly wore off. The lung symptoms came on from three to twelve hours later. The respirations were rapid; there was cough and severe pain in the chest and a varying quantity of expectoration; often the patients were semi-comatose. There were two groups of cases: (1) those showing venous engorgement with cyanosis and deep respirations, the pulse being full and of good tension; (2) the more serious "grey" cases, showing collapse, with pale leaden faces, with rapid, weak pulse and shallow respirations. In patients who recovered the œdema fluid disappeared in a few days; but bronchitis and broncho-pneumonia often came on, and emphysema persisted.

"*Mustard*" *gas* is really an oily liquid which is scattered on the ground or on to the clothes and slowly evaporates. Nothing was usually noticed for some hours except a faint smell of mustard. There then developed severe conjunctivitis, vomiting with epigastric pain, widespread erythema of the skin with vesication leading to severe burns, inflammation of the mucous membrane of the respiratory tract, which caused the most dangerous symptoms. The whole surface became ulcerated and covered with a fibrinous membrane, it was secondarily infected, and if death did not come on at once broncho-pneumonia supervened.

**Treatment.**—In treating the immediate pulmonary complications of irritant gas poisoning, the most important principles are—(1) to keep the patient at rest, so that the metabolism of the body shall be as low as possible: for this purpose chloral may be used if necessary; (2) to keep him warm; (3) to give oxygen continuously by means of Haldane's apparatus (*see* p. 234), because oxygen want is the most important element in causing death.

**Later Effects.**—Many patients who have been gassed develop the "effort syndrome" (*see* p. 356). The following symptoms have been observed, shortness of breath on exertion (*70*), persistent cough with sputum (*54*), pain or tightness across the chest (*25*), palpitation and occasional giddiness (*14*), morning vomiting or nausea (*12*), headache (*9*), neurasthenic symptoms (*7*), soreness of eyes (*5*). The blood may show polycythæmia. In this connection Haldane, Meakins and Priestley have observed that these patients sometimes lose the power

of breathing deeply, and can only take rapid *shallow* respirations after exercise. They believe that these patients suffer from want of oxygen because the ventilation of the deeper alveoli is defective, so that the blood from these parts is imperfectly aerated. Treatment in an oxygen chamber relieves the symptoms more or less permanently, and the polycythæmia diminishes (Hunt and Dufton).

(The *italic* numbers in brackets refer to percentage incidence of symptoms in 83 cases in the Oxford area.)

Various pulmonary sequels to mustard gas poisoning have been described, many of them being of a relapsing character, *i.e.* bronchitis, emphysema, asthma, œdema, pulmonary abscesses, and "pseudo-tuberculosis" with wasting, fever, bronchitis and apical râles, but with no tubercle bacilli in the sputum. There is fibrosis of the lung. These conditions are due to secondary infection after the injury due to the gas. Finally, true tuberculosis of the lungs may be found, probably in those cases where the disease has been previously arrested.

## NEOPLASMS OF THE LUNG

Malignant disease of the lung may be either primary or secondary. The latter is more common, and occurs mostly in the form of nodules scattered irregularly through the substance of the organ, or forming plates covering the surface of the pleura. The original seat of the growth is rather commonly in the breast, or stomach, or the bones, but it may be anywhere. The lung is also sometimes invaded directly from the mediastinal glands, or by an epithelioma of the œsophagus. Primary growths of the lung are classified in three groups according to their origin: (1) from the bronchial epithelium, (2) from the bronchial mucous glands, (3) from the lung substance. In the first group the tumour is chiefly localised in one of the larger bronchi, which becomes obstructed; the growth does not spread diffusely but tends to break down and form bronchiectatic cavities. It is columnar-celled. With the second group, which also tends to be localised round the bronchi, there is abundant mucous secretion. The third group causes rapid and diffuse infiltration of the lung with destruction of the surface where the normal exchange of gases takes place. The cells vary from being columnar in shape to flattened, but are usually cubical, suggesting an origin from the bronchioles or alveolar epithelium. As a neoplasm of the lung grows adjacent portions of the lung may become pneumonic or gangrenous, or by involvement of the pleura an effusion is caused, which compresses the lung. Very rarely a growth involving the lung starts in the pleura.

**Symptoms.**—These vary with the position and distribution of the malignant growths—that is, according as they are seated in the main bronchi, or are disseminated throughout the lung, or form one continuous mass or infiltration, involving a large portion of the organ.

1. Carcinoma involving the bronchus usually causes obstruction, the symptoms of which have already been described (*see* p. 229). Sometimes primary carcinoma of the mucous membrane causes death by profuse hæmorrhage.

2. When the lung is the seat of numerous nodules of growth, scattered indiscriminately through it, the patient suffers at first, at any rate, but little discomfort, and the physical signs are not very distinctive. Percussion resonance is normal, and the only change that may be observed is a diminution of the respiratory murmur all over the chest. In other cases the carcinoma nodules are more numerous, or set up bronchitic changes in the neighbouring bronchi; and more decided, though scarcely characteristic, symptoms may be produced. These are dyspnœa, very rapid breathing, lividity, frequent cough, and mucous expectoration; and on auscultation numerous rhonchi and râles are heard over the whole chest. The condition bears some resemblance to miliary tuberculosis, but the

temperature may be normal. In other cases pleuritic friction sounds are heard in patches scattered widely over the lung.

3. Uniform infiltration of the lung is insidious in its course, and produces cough, dyspnœa, and expectoration of mucus, which is sometimes tinged with blood, and sometimes mixed with larger quantities; occasionally it has a dark colour, and resembles currant jelly; but hæmoptysis is not very common. Pain is not generally a prominent symptom. The physical signs are those which must result from the infiltration of the lung with a solid material, at the same time that the bronchial tubes are filled up or blocked by the new growth. There is dulness with absence or deficiency of breath sounds, of vocal resonance, and of tactile vocal fremitus. There is thus a general resemblance to pleural effusion, which is often very deceptive. If the growth is considerable, the resemblance is increased by expansion of the chest wall and displacement of the mediastinum; but a simple infiltration of the lung without large masses may lead to contraction of the side affected, and resembles rather cirrhosis, or chronic pneumonia, or phthisis, or chronic pleurisy with partial absorption of fluid. Exceptionally, from the breaking down of the growth or as a result of bronchial obstruction and bronchiectasis, cavities are formed which produce characteristic physical signs; but the disease is usually too acute for this. Often the growth is accompanied by pleuritic effusion; this may be a purely serous liquid, or it may contain blood from rupture of the vessels of the new growth. The bronchial, cervical, and axillary glands become enlarged; and extension to the mediastinum may lead to symptoms of pressure, such as œdema of the head, neck, chest, and upper extremities, dilatation of superficial veins, abductor paralysis of one vocal cord from pressure on its recurrent laryngeal nerves, obstruction of the trachea or one bronchus, or dysphagia from pressure on the œsophagus (*see* Mediastinal New Growths).

Neoplasm of the lungs, no less than that of other organs, is accompanied by progressive emaciation and loss of strength, and ultimately, in the course of from six to twelve months, death takes place, generally from exhaustion. In the arterial blood the $CO_2$ pressure is increased and the saturation with oxygen diminished before the end. The temperature is often normal, but it may be diminished before the end (**7**). The temperature is often normal, but it may be pyrexial. Sometimes, but not always, this is explained by an accompanying septic process in the bronchus or the pleura. The pyrexia is not so marked as in rapidly advancing tuberculosis. A pneumothorax is not so very uncommon.

**Diagnosis.**—When growth is known to exist in other organs, or when a carcinoma of the breast or of the jaw has been removed by operation, the presence of unaccountable dyspnœa should make one think of its occurrence in the lung; and in cases where the pulmonary symptoms are most prominent the presence of large hard glands in the neck, or a tumour of the testis, or a rigid spine from implication of the vertebræ, may sometimes give the required clue. Extensive infiltration of the lung is most easily confounded with *pleuritic effusion*, and in elderly persons with the symptoms and physical signs of fluid the possibility of carcinoma should not be forgotten. Exploration with the needle, or a trocar and cannula, or aspirator, may be necessary, and particles may then perhaps be obtained for microscopic examination. The sputum sometimes provides similar evidence. But if the exploration be negative, it may be that the needle has entered a lung collapsed from a carcinoma obstructing the root, and in this case bronchiectatic cavities may afterwards develop, with offensive purulent sputum. On the other hand, if liquid be found, this does not exclude a carcinoma of the root of the lung; and this may well be suspected, if the liquid returns again and again after aspiration while the temperature remains normal. Such a fluid should show, after centrifuging, only endothelial cells, and no leucocytes or lymphocytes (*see* p. 276). Blood-stained liquid is suggestive of neoplasm, though its commonest cause is tuberculosis. As implied above, a febrile temperature

does not exclude carcinoma. The Röntgen rays may, of course, give valuable help (*see* Plate IV).

The **Prognosis** is bad, and the **Treatment** must be directed to relieving pain and cough procuring sleep, and supporting with good nourishing food. Deep X-ray therapy may be tried or radium may be employed in massive doses. This treatment is most successful with sarcoma. A liquid effusion accompanying the growth may be aspirated, but it will probably return quickly.

## HYDATID OF THE LUNG

This parasite affects the lung in two ways: first, a cyst may form in the lung apart from, or even without, its occurrence in any other region; secondly, the lung may be invaded by a cyst in an adjacent organ rupturing through the parts which separate them. This is most common in cases of hydatid of the liver.

A *primary hydatid of the lung* is very rare in this country. It forms a globular cyst, with all the characteristics of the parasite as seen in the liver (*see* Hydatid of the Liver), but it is not generally surrounded by such a dense cyst of connective tissue. It is rather more frequent at the base than at the apex. It may be the only cyst in the body, or there are others at the same time in the liver, spleen, brain, or elsewhere.

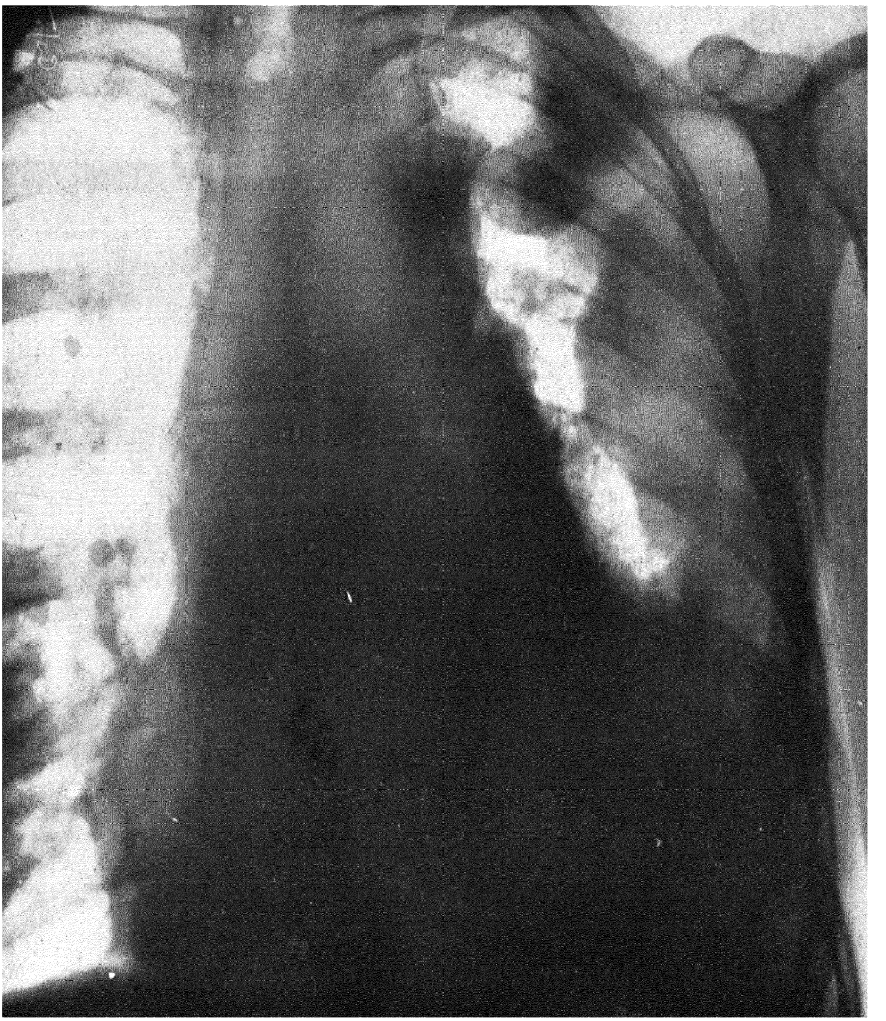
**Symptoms.**—A cyst may attain a large size without producing symptoms. The earlier symptoms are due to changes round the cyst. (1) It may rupture into a bronchus, and there is a sudden rush of salt-tasting, watery fluid with a sense of suffocation. The fluid contains cyst *débris*, and hooklets may be found. This may result in a cure. After rupture of a cyst the signs of a cavity may develop. (2) Hæmorrhages may occur without other premonitory signs. It is difficult to exclude phthisis, but the small degree of constitutional disturbance and the fact that the other lung is quite healthy are in favour of hydatid disease. (3) In half the cases there are attacks of pneumonia and pleurisy, often recurrent over a period of years. Empyema may occur, and exceptionally gangrene.

The final symptoms are observed when the cysts have enlarged to such an extent that one lung is largely involved, and there is perhaps a little pleural effusion. There may be pain and cough, but the patient is not as a rule very ill. The breath sounds are deficient, and there is corresponding dulness, and there are accompaniments in the surrounding lung. The X-ray appearances are characteristic, opaque masses of circular outline being seen; but they may be somewhat obscured if pneumonia is also present. If a cyst has ruptured into a bronchus it will appear as a dark ring containing air and fluid, like pneumothorax.

**Diagnosis.**—A *primary* hydatid is most likely to be mistaken for phthisis, especially if the cyst is situated at the apex. A girl with the symptoms of a cerebral tumour had hæmoptysis, and was thought to have tubercle of the brain and pulmonary phthisis; but a hydatid cyst was found in the brain and another in the lung. The patient's serum may be tested for precipitins and specific anti-bodies (*see* Hydatid of the Liver), and the blood tested for eosinophilia and basophilia. Diagnostic puncture may be used if the cyst is small, but later on it is too dangerous (Foster). *Secondary* hydatid is generally recognised by the preceding hepatic trouble, and the appearance of bile and hydatid skins in the sputa.

**Treatment.**—No internal treatment can kill the parasites in the lung. If the diagnosis of a cyst sufficiently near the surface could be made with confidence, it might be treated by the surgical methods applicable in hydatid of the liver. Hydatid of the liver opening into the lung is commonly beyond the reach of

PLATE I



Radiogram of Chest showing secondary deposits in the Left Lung. The shadow at the base with concave upper border suggests a small pleural effusion. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 268.

surgical interference, and the treatment must be symptomatic, and in the main supporting.

## PULMONARY EMBOLISM AND THROMBOSIS

The nature of embolism and thrombosis is dealt with in the section on Diseases of the Blood Vessels, but the special liabilities of the pulmonary circulation to this accident may here be described.

The pulmonary artery and its branches are in direct communication with the systemic venous trunks through the right ventricle and auricle; and accordingly micro-organisms, particles of coagulated blood, or of any other kind, which become loose in the veins of the body or limbs, must be carried into the right cardiac cavities, and thence into the pulmonary artery, where they will be impacted in a larger or smaller branch according to their size. A large old laminated clot may block one of the main branches. In some cases the embolism is a long thrombus from a medium-sized systemic vein which is folded up in a ball.

Pulmonary thrombosis is commoner than pulmonary embolism, occurring in 85 per cent. of thirty-five consecutive autopsies where these lesions were present. Blood clots are found in the branches of the pulmonary artery in all parts of the lungs and there is evidence of their ante-mortem formation in the commencing organisation of the clot or deposition of pigment. The diagnosis of "pulmonary embolism" or "pulmonary thrombosis" usually implies that there is a serious lesion producing widespread effects in the lungs with urgent symptoms, and placing the patient's life in immediate danger.

Pulmonary embolism and thrombosis may result from thrombosis of the femoral vein, such as occurs in typhoid fever, or phthisis, or fracture of the femur in older persons. They may result from acute infections, such as pneumonia, and from surgical operations, especially operations on the pelvic viscera, because in these cases large veins, like the ovarian vein, may be injured by clamps, and there are often septic foci in the neighbourhood. Micro-organisms have in many cases been found in the clots. The effects of the emboli vary with their size. If one of the main branches of the pulmonary artery is blocked, rapid death is a necessary consequence. When the embolus is smaller there is time for changes to take place in the lung, and hyperæmia, petechial hæmorrhages, œdema, and collapse with emphysema around it, are found.

In auricular fibrillation which is particularly associated with mitral stenosis small thrombi may form in the auricles owing to their failure to contract, and those from the right side may pass into the lungs and block the smaller arterioles and give rise to local hæmorrhages in the lungs, which are known as *pulmonary infarcts*. Thus a conical portion of the lung wedge-shaped in longitudinal section, its base to the surface of the lung and its apex internally, becomes solid, firm, dark red in colour, and airless; and under the microscope the air vesicles are seen to be filled with red blood corpuscles. The base of the cone bulges on the surface of the lung beyond the surrounding vesicular tissue, and in a short time the surface may present early pleuritic changes. These infarcts are most common in the lower lobes and often involve the lower edge to a considerable extent, when the description of them as conical or wedge-shaped hardly applies. They are commonly about 1 inch in diameter, but sometimes reach a much larger size.

Other bodies may act as emboli besides these venous and cardiac thrombi, namely, particles of growth, and rarely a small hydatid cyst, but quite commonly also pyogenic micro-organisms. When the latter are carried, either alone or with particles of thrombus, into the lung, the conical infarcts soon become abscesses, and are often accompanied by broncho-pneumonia of the adjacent tissue, or by serous, sero-purulent, or purulent pleurisy.

*Fat embolism* of the pulmonary capillaries is a result of injury, which allows the passage of fat into the vessels. Globules of fat are very commonly seen microscopically in the lung capillaries after death from surgical injuries.

**Symptoms.**—The symptoms of embolism of the pulmonary artery and its main branches vary with the size of the vessel obstructed and the degree of obstruction. When a large thrombus from the femoral vein is impacted in the pulmonary artery or one of its main branches, death may be absolutely sudden; the patient may start up from bed in alarm and fall back dead, or there may be a few minutes' dyspnœa with cyanosis, or, on the other hand, syncope or convulsion. If the obstruction is less complete, the condition may be one of syncope, or syncope with asphyxia, or with rigors, pain in the chest of varying severity, a sense of suffocation, dyspnœa, developing perhaps into Cheyne-Stokes' respiration and, finally slowing down and ceasing. The face may be pale and livid, the jugular veins distended, and the hands cold and clammy. On auscultation the breath sounds are harsh and exaggerated. The onset of symptoms is sudden. In pulmonary thrombosis the symptoms are similar in character but the onset, rapid as it may be, is gradual.

The symptoms of the occurrence of an infarct will also vary with the size of the vessel obstructed, and the nature of the thrombus or particle impacted. If it is a relatively large vessel, the symptoms may resemble those above described, but will be of less severity. With a sufficiently large infarct there may be breathlessness, palpitation, and even rigor. The extravasation of blood into the lung tissue often reveals itself by the expectoration of blood, or hæmoptysis; and the blood may be in moderate quantity, or in small separate blood sputa, or may only render the mucous sputa blood-coloured or rusty. Pain in the side will result from the concomitant pleurisy, and after the event there may be some febrile reaction with or without rigors. Only if the infarct is very large will it give rise to an area of dulness, and suppression of breath sounds; but some crepitation is possible. If an extensive area of dulness is found in a cardiac case in which infarcts are suspected, it must be remembered that the mixed condition of congestion and œdema, known as *red induration* and *brown induration*, is a common result of valvular disease, and often co-exists with infarcts.

Septic infarcts are frequent in pyæmia, and indeed are the characteristic post-mortem lesion of that disease in its acute form. The foci are commonly quite small, and incapable of giving rise to definite physical signs other than some crepitation. But the pleurisy and effusion which so often accompany them will give the usual signs, and febrile reaction of septic type with rigors and increasing prostration will be present.

Fat embolism, when present in sufficient amount, may be one of the causes of surgical shock, owing to obstruction of the pulmonary circulation. The symptoms are dyspnœa, prostration, red frothy sputum, quick pulse, cyanosis, and râles over the lungs.

**Diagnosis.**—The diagnosis of pulmonary embolism or thrombosis depends very much upon the antecedent data, such as the known existence of venous thrombosis or sepsis, associated possibly with some abdominal operation. The existence of heart disease may suggest pulmonary infarction. Of all the causes of hæmoptysis, heart disease is the next most frequent to pulmonary tuberculosis; the knowledge of this fact goes a long way to a safe diagnosis.

**Prevention.**—It is suggested that after abdominal operations, if there is no peritonitis, and after labour, early movement of the legs and pelvis should be permitted, so as to prevent stagnation, which predisposes to thrombosis (Symonds). Deep breathing should be carried out systematically to prevent stagnation in the lungs which leads to thrombosis. If the patient is blanched blood transfusion should be carried out before operation. The patient should be allowed to drink plenty of water to avoid concentration of the blood.

# DISEASES OF THE PLEURA

## PLEURISY AND EMPYEMA

The chief features of pleurisy, or inflammation of the pleural membrane, are either the formation of "lymph" on the pleural surface (dry pleurisy), or the exudation of serous fluid (pleurisy with effusion), or the production of pus (empyema).

**Ætiology.**—In a large number of cases the onset of a dry pleurisy, or pleurisy with effusion, occurs insidiously in apparently healthy persons, and is often attributed to exposure to cold. Among such cases a large proportion, perhaps 50 per cent., are undoubtedly tuberculous in origin. Many have a history of tubercle, or they afterwards die of phthisis or other tuberculous lesions. In many cases also the fluid inoculated into animals produces tuberculosis. Such cases of pleurisy are chronic in their course.

The pleura is subject to what is usually a more acute form of inflammation often leading to empyema as the result of many other infections, especially pneumococcal infections, and those of scarlatina, measles, rheumatic fever, septicæmia, and influenza; pleurisy is also a frequent complication of Bright's disease. In other cases the infection is more directly local, as, for instance, when the pleura is injured by fractured ribs, or when there is an extension to the pleural surface of (1) lesions in the lung, like those of pneumonia, pyæmic abscesses, growth, tubercle, or hæmorrhagic infarcts; (2) lesions of the parietes, such as abscesses, in the axilla, breast, neck, or abdominal cavity.

Pleurisy, pericarditis, and peritonitis may occur together from the same infection, which is in acute cases rheumatic, septic, or pneumococcal, in chronic cases often tuberculous (*see* Polyorrhomenitis).

The micro-organisms found in different forms of pleurisy are the following: pneumococcus, streptococcus, staphylococcus, *Bacillus tuberculosis* and *B typhosus;* more rarely Friedländer's bacillus, *B. coli communis*, *B. diphtheriæ*, *B. influenzæ*, and *Micrococcus tetragenus*. They are often combined, as, for instance, pneumococcus or tubercle bacillus with streptococci or staphylococci; the last are not commonly found alone. In the sero-fibrinous effusions of tuberculous pleurisy, tubercle bacilli are often absent; they are more often present in tuberculous purulent effusions. In purulent effusions of children, pneumococci are mostly found (80 per cent.), and in those of adults streptococci are more common (75 per cent.).

**Morbid Anatomy.**—*Dry Pleurisy.*—The first stage consists of dilatation of the vessels of the pleura, quickly followed by exudation of fluid and migration of some leucocytes. The fluid coagulates and the resulting fibrin mixed with the leucocytes, often called loosely "lymph," is deposited on the free surface. The membrane is at first minutely injected, and very soon the naturally shining surface is rendered dull by the fibrin, which can be detached as an extremely delicate membrane. If the exuded material is more abundant, it forms thick layers, firm or pasty, generally rough on the surface, or villous, or reticular.

*Pleurisy with Effusion.*—In this case an exudation of serous or sero-fibrinous fluid soon follows the production of "lymph" in a dry pleurisy, or if the process is more chronic an exudation of serous fluid occurs without the previous formation of "lymph." The fluid may accumulate to the extent of 2 or 3 pints or more in the pleural cavity. It has a yellow or greenish-yellow colour, a specific gravity of 1,005 to 1,030, often 1,015 to 1,018, and it becomes almost solid on boiling from the albumin it contains. Not infrequently there are a few flakes of fibrin, or a quantity is deposited from the liquid a short time after its removal. The liquid is quite clear, or it is opalescent or turbid from the presence of cor-

puscles. In acute cases eosinophil or polymorphonuclear cells predominate, in the more chronic cases the cells are relatively few and they are lymphocytes. In acute cases the cells may be in sufficient quantity to form a thick layer at the bottom of the fluid after its removal, and there is every gradation between this and the formation of thick pus. Sometimes the liquid is more or less tinged with blood, proceeding from new-formed vessels in the organising "lymph."

This effusion of fluid is one of the most important results of pleurisy. Confined within the cavity of the pleura, it must displace the lung from its relations to the diaphragm and the wall of the chest, and in proportion as more fluid is effused, the lung becomes collapsed. This is not due to the pressure of the fluid, but to the elasticity of the lung, which naturally favours its retraction; and, indeed, it may be found that even with a considerable quantity of fluid in the chest the pressure is still negative. But, even though this is the case, it may yet be higher than the pressure in the opposite pleura, so that the heart is displaced towards the healthy side. However, in some cases the pressure in the fluid may be greater than atmospheric, especially if it is purulent, and then, besides displacing the heart and mediastinum, it bulges the wall of the thorax outwards, distends the intercostal spaces, and forces downwards the diaphragm with the subjacent liver or spleen. In extreme cases the great vessels may be pressed upon and narrowed (Elliot Smith).

Pleurisy heals, like other inflammatory processes, by organisation of the fibrin covering the two surfaces, *i.e.* the new formation of blood vessels and of fibrous tissue. The effusion, if present, is absorbed in the course of days or weeks, and the lung and the chest wall finally come into contact either by expansion of the former, or by a gradual sinking in of the latter, or by a combination of these processes. In mild cases there may be no evidence in later years that any inflammation has ever occurred. On the other hand, the pleura over the lung may show a patch of thickening and be white in colour, while the surface is smooth and shiny. More often the parietal and visceral pleura have been in contact during organisation, so that they become united by fibrous tissue, which is known as an *adhesion*. Adhesions commonly occur between the lobes of the lungs. The vast majority of elderly people who die show evidence of some previous pleural inflammation. In severe cases the pleura may be greatly thickened, and fibrous tissue may invade the lung, producing fibrosis (*see* p. 245).

A purulent pleurisy, or *empyema*, appears sometimes to arise out of a serous pleurisy, or *simple effusion;* but it is often found soon after the onset of the symptoms, and it is then undoubtedly primary. The early occurrence of empyema is most common in association with acute lobar pneumonia, in pyæmia and septic cases, after scarlatina, and as a result of perforation of the pleura from the lung or the abdomen. If untreated, its termination is by no means so favourable as that of a serous effusion. Occasionally, no doubt, absorption takes place—that is, the fluid is taken up, the pus corpuscles become granular and fatty, and a caseous mass remains behind, or calcareous salts may be deposited in the residue. Sometimes an empyema finds its way through the pleural sac, either perforating the lung, so that the pus is expectorated, or "pointing" in one of the intercostal spaces, often the fifth, and bursting spontaneously. In either case, air may find its way into the pleural cavity, and give rise to *pyo-pneumothorax*. Rarely an empyema opens through or behind the diaphragm into the abdomen. But, if unrecognised or untreated, it may remain a long time without perforating, with incomplete absorption, rendering the patient cachectic, and preparing the way for lardaceous degeneration of the viscera.

Both in serous and purulent effusions, the cavity is occasionally divided into separate spaces by adhesions between the lungs and the parietes. The fluid is then said to be *loculated*, and the condition is of importance when the case is treated surgically.

It must be recognised that the fluid, whether serous or purulent, does not

necessarily collect at the lower part of the chest and spread uniformly upwards, although that is what takes place in the great majority of cases. Collections of pus (or serum) may be confined to small areas of the pleural surface at the middle zone, or near the apex, or between the upper and lower lobes of the lung (*interlobar pleurisy*), or between the base of the lung and the diaphragm (*diaphragmatic pleurisy*). Rarely the liquid, more often pus than serum, has been formed between the mediastinum and the inner surface of the lung, so as to exert pressure especially upon the important structures in the centre of the chest; this has been called *mediastinal pleurisy*.

**Symptoms and Physical Signs.**—*Dry Pleurisy.*—The onset of pleurisy is characterised by severe pain, caused or aggravated by the act of breathing. The pain is commonly situated low down at the side of the chest; but it may be anywhere, depending on the position of the inflammation. It is cutting or tearing, and is intensified not only by breathing, but by coughing, sneezing, and every kind of exertion. The patient generally lies on his back or on the healthy side. Acute pleurisy may begin with a chill, and there is mostly some pyrexia, in which the temperature may reach 103°, but is more often 101° or 102°. With it are the usual accompaniments: furred tongue, loss of appetite, and malaise.

On examining the chest some impairment of movement on the affected side and deficiency of vesicular murmur at the painful spot are observed; but the characteristic physical sign is the *pleuritic rub*, or friction sound, which arises by the movement upon one another of the two pleural surfaces, roughened by exudation (*see* p. 195). The sound varies with the degree of friction. In acute cases the rub may be strictly localised and easily missed; it may even be absent, if the patient is prevented by the severe pain from making the inspiratory movement necessary to produce it. In chronic cases the friction may be so great that it can be felt by the hand placed on the chest, as well as heard with the stethoscope, and is quite painless.

*Pleurisy with Effusion and Empyema.*—When liquid is effused, the two pleural surfaces become separated, the friction sound disappears, the pain diminishes, and symptoms and physical signs occur which are the direct result of the presence of liquid and the compression or displacement of the various organs which it effects. The chief symptom is shortness of breath, especially on exertion, and this dyspnœa is in proportion to the amount of liquid effused. It is often scarcely observed when the patient is still, but becomes manifest when he moves about, or even when he talks. He lies on his back, or on the affected side, to allow the greatest freedom to the healthy side. He may be entirely free from cough, or may have slight cough without expectoration. Fever commonly persists as before in acute cases. The pupils are occasionally unequal in pleuritic effusion, that on the affected side being larger.

As the fluid gravitates to the most dependent part of the chest, small quantities are usually detected at the base behind, where there is absolute dulness, while vesicular murmur, vocal resonance, and tactile vocal fremitus are much enfeebled, or entirely absent. With a considerable quantity of fluid the following physical signs are observed: On inspection the affected side of the chest is motionless, and may be obviously larger than the other; the intercostal spaces, instead of being slightly depressed below the level of the ribs, are filled up (or "obliterated"). The heart is displaced: with effusion on the right side, its impulse may be perceived beneath or outside the left nipple; with effusion on the left side, an impulse is often felt in the intercostal spaces to the right of the sternum, generally the third, fourth, and fifth, even as far as the right nipple, and in rare instances beyond it. The dulness in such cases is observed in front, in the axilla, and behind, and is continuous with dulness on the opposite side corresponding to the displaced heart. At the same time on the opposite side of the chest the resonance is not unaffected, for a triangular area of dulness is

found of which the apex is close to the spine at about the level of the angle of the scapula, and the base extends from the spine along the lower border of the lung for from 2 to 3 inches (*Grocco's paravertebral triangle*). The dulness diminishes in this area when the patient lies on the side of the effusion. The cause of Grocco's triangle is only very rarely displacement of the lower posterior part of the mediastinum over to the unaffected side. The usual explanation is as follows: Normally the expansion of the central part of the lung is only possible because the tension of the posterior mediastinum is increased, which prevents it from being sucked over. This increased tension is due to action of the opposite diaphragm. When this is deficient, as in pleural effusion, the mediastinum gives at each inspiration, so that the central part of the lung on the unaffected side receives less air and the percussion note is impaired.

The liver on the right side, or the spleen on the left, may be pushed down; and the descent of the diaphragm on the left side leads to dulness at the upper part of that space between the left lobe of the liver and the spleen, which normally yields gastric resonance (*Traube's space*). When the liquid is sufficient only to reach above two-thirds the height of the chest, there may be heard the peculiar modification of the percussion note under the clavicle and above the level of dulness, which is known as *Skodaic resonance*, which is due to relaxation of the lung (*see* p. 191).

Over the dull area there is diminution or absence of breath sounds, of vocal resonance, and of tactile vibration. At the upper level of the fluid where the lung is relaxed bronchial breathing or compensatory breathing may be heard; bronchophony or *ægophony* may be present (*see* p. 195). On the opposite side the breath sounds are exaggerated, but over Grocco's triangle they are diminished. In the case of young children the dulness on percussion is not absolute, and bronchial breathing may be heard all over the dull area, and this may lead to the belief that the lung is solid. The displacement of the heart may help in such a case.

In extreme cases where the whole of one chest is dull—back, front, and side, and from apex to base—and there is great displacement of the viscera, the disturbance of the respiratory functions may at length be fatal. The patient becomes more and more livid, rhonchi and mucous râles are heard in the hitherto healthy lung, and asphyxia ensues. Sometimes there is sudden syncope, which may reasonably be explained by pressure on the heart and great vessels.

Very rarely the pulsation of the heart (or perhaps the aorta) is communicated to a pleural effusion, either as a shock or wave transmitted to a large serous collection or as a more localised, perhaps visible, pulsation in an empyema, pointing through the chest wall. This is described as *pulsating empyema* or *pulsating pleurisy*.

The symptoms due to pleurisy affecting the different situations referred to above (*see* p. 273) must depend on the particular localisation of each. In the early stage of *interlobar pleurisy* there may be pain, cough, and oppression of breathing, but no distinctive sign. When liquid has collected to the extent of 6 or 7 ounces or more, percussion may give a dull note in the middle zone of the chest, with resonance above and below; and this may be accompanied by râles. Hæmoptysis is a frequent symptom in *interlobar pleurisy* (Dieulafoy), and if the fluid is purulent, as it often is, it may open into a bronchus, be coughed up and so heal itself, though occasionally a discharging cavity may remain for months or years.

In *diaphragmatic pleurisy* the effusion is not generally abundant; it is often preceded by severe pain with tenderness on pressing over the insertion of the diaphragm into the tenth rib in front, or on compression of the phrenic nerve in the neck. Dulness, friction sound, and ægophony are absent unless the trouble reaches the main pleural cavity, and thus a small collection of liquid, encysted here, may easily be overlooked.

Similarly a *mediastinal pleurisy* may show few distinctive signs until the collec-

## PLATE V.

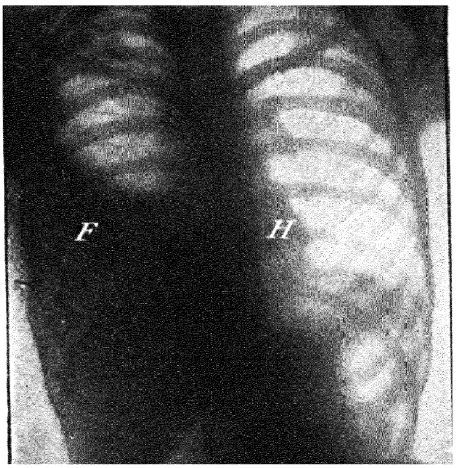
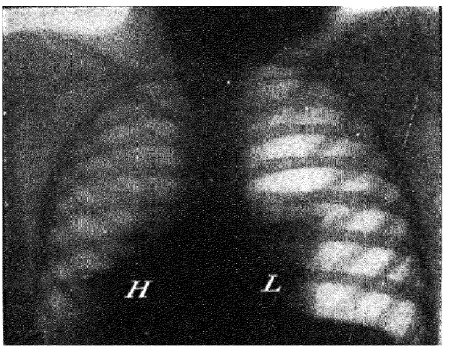



FIG. 1.—Radiogram of Chest in case of Pleural Effusion, showing dark area at base of right lung, with upper margin sloping upward from spine to axilla. Seen from the front. *F* Fluid. *H* Heart.




. 2.—Radiogram of Chest in case of Pneumothorax; the left lung is collapsed, and is seen as a small dark patch (*L*) in the lower part of the left chest, close to the spine. The heart (*H*) is displaced into the right chest. The abnormal clearness of the left chest is well shown. Seen from the front. (Taken by Dr. A. C. Jordan)

[*To face p.* 275.

tion of liquid is sufficient to press upon the important structures in the middle line of the chest. These signs are dyspnœa, attacks of oppression, wheezing and stridor, dysphagia, a brassy cough, hoarseness of voice, and distension of the veins on the surface of the chest. At the same time they may be tenderness on pressure of the dorsal vertebræ, and there may be paravertebral dulness with weak breath sounds, ægophony and râles. Over the pericardium pleurisy gives rise to so-called *pleuro-pericardial friction* (*see* p. 195). Mediastinal empyemas are liable to be discharged through the bronchial tubes. The diagnosis will be helped by X-rays, which will show whether the obstructing mass is pulsatile or not.

**Progress to Recovery.**—When recovery takes place spontaneously the liquid disappears, sometimes gradually, at others with remarkable quickness. The upper parts of the area of dulness become resonant, the vesicular murmur returns, and often with it the friction sound is again heard, louder, longer, and over a much more extensive surface than was the case previous to the effusion. In addition the rub is more distinctly palpable, but much less painful. While the liquid is being absorbed, and the lung is beginning to expand, the chest often falls in—a change which is first observed behind, where the naturally round chest becomes flat as compared with the other side. In extreme cases the chest is flattened in front and behind, these two surfaces meeting in a rounded angle in the axillary line; at the same time, the dorsal spine is curved, with the concavity towards the affected side, the shoulder is depressed, and the lower angle of the scapula projects from the ribs. Frequently the physical signs at the base remain abnormal for some time, suggesting the continuance of liquid; but this is undoubtedly due to incomplete expansion of the extreme base, with or without a layer of new fibrous tissue (thickened pleura), the result of the inflammation.

In some cases a chronic pleural effusion results, the liquid neither increasing so as to cause death nor becoming absorbed.

**Diagnosis.**—In dry pleurisy the pain has to be distinguished from other pains in the chest, the most common of which is *pleurodynia*, or muscular rheumatism. This is increased by movement, but is unaccompanied by fever or by rub. Intercostal *neuralgia* is distinguished by its relation to the nerves, and by the tender points characteristic of neuralgia. Affections of the liver, spleen, or colon may give rise to pains aggravated by breathing, for these structures are compressed by the descent of the diaphragm during inspiration.

In the stage of effusion we have to consider, first, whether there is liquid in the pleural cavity, and, secondly, what is the nature of the liquid, whether serum or pus. In acute cases, pleurisy and *pneumonia* are most likely to be confounded together, because in the early stages of pneumonia the breath sounds are often absent. Pleurisy is generally characterised by the absence of tactile vibration and the more absolute percussion dulness; and the larger effusions cause displacements of the heart and of the diaphragm, which are conclusive. Grocco's triangle is not observed in pneumonic consolidation. Generally, also, pleurisy is wanting in the very high fever, pungent heat of skin, and flushed face of pneumonia. The two may occur together, and then the bronchial breathing characteristic of pneumonia may be modified or absent, and the negative signs indicative of the lung being compressed by fluid will predominate. But should the sputum be rusty, pneumonia is certainly present. With the X-rays a very dark shadow, whose upper concave margin rises sharply from the spine towards the axilla, is produced by pleural effusion; and if the effusion is large, the lines of the diaphragm and the lower ribs are not seen. The lung above shows a faint darkening from condensation, and a displacement of the heart is generally obvious (*see* Plate V., Fig. 1). In pneumonia the lung is opaque, but there is no curved upper margin. The early signs of pleurisy are sometimes so little marked, and the effusion of liquid is so insidious, that the local features of the illness may be overlooked, and the patient may be thought to be suffering from *typhoid* or other form of fever.

In chronic cases, fluid may be simulated by most *consolidations* of the lung tissue, whether from deposit in its substance or from compression; such are some cases of tuberculous consolidation, the induration which results from heart disease, growth in the lung, compression from the front by pericardial effusion, and from below by subphrenic abscess and by growth or hydatid of the liver. The physical signs common to these conditions are dulness, loss of breath sounds, of vocal resonance, and of tactile vibration—that is, absence of all evidence of healthy lung—simply because these growths or collections of liquids compress or push up the lung in the same way as a pleural effusion does, and do away with that spongy tissue in communication with the trachea and larynx upon which the normal physical signs depend. Most of these changes affect the base of the lung, and no help can be gained from the absence of the signs (enlargement of chest or displacement of heart) which occur in the more abundant effusions. A large growth infiltrating the lung is especially deceptive, because it may form a tumour, occupying and enlarging one side of the chest, and pushing the mediastinum and heart to the other side. Another cause of dulness at the base, frequently mistaken for pleural effusion, is obstruction of one bronchus; but in this case the dulness is not usually so absolute as with pleural effusion. X-ray examination is of particular value in distinguishing subdiaphragmatic tumours, abscesses or cysts from a pleural effusion, because the diaphragm constitutes the upper surface of the former, and is convex, though it may be raised and immobile with respiration. The upper surface of a pleural effusion is concave.

In all but acute cases the pitfalls are so numerous in diagnosis that an early appeal should be made to exploration with a suitable needle and syringe. This has the additional advantage of determining, as it alone can, the nature of the fluid, and of affording material for microscopic and bacteriological examination.

With empyema the patient often has a sallow appearance or even marked anæmia; the temperature has often a hectic type, ranging from 98° or 99° in the morning to 102°, 103°, or 104° in the evening, and with this rigors or profuse sweatings may occur. But the chest may be full of pus when the temperature is quite normal. There is pronounced leucocytosis in empyema. The sudden occurrence of purulent expectoration in the course of pleurisy is an important indication of an empyema which has ruptured into a bronchus; and in cases of long duration the ends of the fingers become thickened, or "clubbed." Œdema of the chest wall occurs more frequently in empyema than in serous effusions, but in neither case is it an early sign. The chest wall overlying an empyema may be tender on pressure. In young children, a combination of cough, dyspnœa, vomiting and wasting suggests empyema.

Serum may be present in the pleural cavity from other causes than pleurisy—namely, local and general dropsy. The physical signs are the same, but *hydrothorax*, as the condition is called, generally follows upon disease of the heart, or Bright's disease, or pressure upon vessels by growth in the chest; and there is an absence of the febrile accompaniments of pleurisy.

Microscopical examination may help to a knowledge of the origin of the effusion. The liquid is centrifuged, and the sediment of cell elements is stained and examined microscopically. In passive effusions (hydrothorax) large endothelial cells predominate, but lymphocytes may be present; in infective forms of pleurisy due to streptococcus or pneumococcus the polymorphonuclear and large mononuclear leucocytes are found in excess; while in tuberculous pleurisies lymphocytes often predominate, but polymorphonuclear cells are often present as well. Tuberculosis is the commonest cause of a blood-stained effusion, but the latter also occurs with growth.

If organisms cannot be directly cultivated from the pleuritic serum, and tubercle bacilli cannot be found, its tuberculous origin may be demonstrated by the *inoculation* of a guinea-pig.

As indicated in the description of the symptoms of the localised pleurisies

(*see* p. 274), their diagnosis may be very difficult. A mediastinal pleurisy is liable to be confounded with mediastinal growths, whether malignant or lymphadenomatous; but the history of the former is usually short, and the onset rapid. Help may in all cases be obtained from the X-rays.

**Prognosis.**—Most cases of pleurisy without effusion, or with a sero-fibrinous effusion, get well either under medicinal treatment or after removing the liquid; but their subsequent history is often unfavourable (*see* p. 272). Empyema is very fatal in children under two years old; only 25 per cent. recover (**27**). This is because it is usually only one manifestation of a general pneumococcal infection, which simultaneously causes pneumonia pericarditis, etc. In older children empyema usually follows pneumonia and there are no other complications, and so the prognosis is good, better even than in adults, and more promising the earlier the pus is evacuated. This is probably because in children the majority of cases are pneumococcal, while in adults more are streptococcal (*see* p. 271). If a pneumococcal empyema is complicated by pericarditis, which is not infrequently the case, the prognosis is bad; but cases have recovered.

**Treatment.**—If there is any chance of the pleurisy being due to tubercle, the *régime* adopted should include fresh air, nourishing food and exercise, controlled by temperature observations, so long as there is no pain (*see* Treatment of Pulmonary Tuberculosis).

Pain may be alleviated by the application of linseed meal poultices, antiphlogistin or thermogen wool, and the administration of opium or morphia internally, or morphia subcutaneously. The use of blisters, of leeches, or of cupping over the painful spot, also generally gives relief. The affected side may be strapped, and by this means the respiratory movements are restrained, the pain is allayed, and inflammatory action is probably in some measure checked. The strapping should be applied in broad strips from the spine to the sternum, alternate strips passing obliquely upwards and obliquely downwards, till the whole side is covered. The patient should be kept at rest. If effusion takes place, anodynes will be less needful, and salines, such as the acetate and citrate of potassium, or of ammonium, may be given for their effect upon the excretions of the skin and kidney, the increase of which will favour absorption of the effused fluid. After a time iodide of potassium, squill, or other diuretics may be added, and it is possible that absorption is increased by counter-irritation, such as may be obtained by painting the tincture or solution of iodine over the affected side.

The most usual practice at present in cases of tuberculous effusions is to remove the fluid only when it is present in excessive amounts. It is believed that moderate quantities may be actually beneficial by compressing the lung and diminishing movement, on the same principle that artificial pneumothorax is beneficial. If the heart is pushed over, or there is absolute dulness over most of one side of the chest, some of the liquid should be removed by a trocar and cannula. The best rule is to allow it to escape until it is under atmospheric pressure, *i.e.* until it no longer runs out of the cannula of its own accord. To save time an aspirator is often used, or the liquid is drained away by syphonage through a flexible tube into a vessel placed on the floor; but it must be remembered that it is easy by such means to remove more liquid than is desirable. A recent method is to replace the fluid by an artificial pneumothorax (*see* p. 262). Great help in expanding the lung may be obtained by simple breathing exercises. The patient should take deep breaths at frequent intervals, and should also blow out against resistance.

If an exploration by the needle shows that the liquid is purulent (empyema), the surgeon should make a free incision under an anæsthetic, local or general, and resect a piece of rib, and allow the pus to drain away. Children under two do not stand rib resection very well. A cannula may be introduced between the ribs by means of a trocar, and tied in position by tapes round the body. A rubber tube which just fits the cannula is introduced into the empyema cavity

and the contents evacuated into a thick-walled conical flask, permanent suction being kept up by means of a filter-pump or other contrivance (**28**). In cases of neglected empyema of long duration, in spite of free drainage, the cavity continues to secrete pus, and the wound does not close. If this goes on too long, the supervention of lardaceous disease is to be feared. There are two possible methods of procedure: (1) The cavity may be washed out with eusol until the contents are practically sterile as tested bacteriologically. Carrel's method of continuous drainage may be used. The wound may then be allowed to close, so that the patient has permanently a sterile pneumothorax. It is best not to wash out an ordinary acute empyema cavity, as deaths have been reported from *pleural reflex syncope*. This is probably due to vagal inhibition produced by irritation of the inflamed pleura. This danger is not apparently present in chronic cases. (2) A fairly extensive surgical operation may be carried out involving the resection of enough ribs to allow the hand to enter the chest. The thick visceral pleura is peeled off (decortication), and the lung rapidly expands and fills up the cavity.

The problem often arises as to whether an operation should be carried out in cases of acute pleural effusion due to the streptococcus, for instance where the fluid is somewhat turbid and contains large numbers of polymorphonuclear cells when examined microscopically. The answer is that rib resection should only be used when actual pus is present, but the turbid fluid may be removed by aspiration if present in large amount. American observations at Camp Lee, Va., showed the advantage of this procedure in empyemas due to the hæmolytic streptococcus. In early cases pneumonia is often present with the pleurisy; further, if resection of a rib is carried out at too early a stage, the operation wound may become infected leading to a septicæmia. The effusion, even though turbid, often clears up without operation. On the other hand, pus may develop in two or three weeks, and then rib resection should be carried out. During the surgical treatment of an empyema the patient should be supported in every way by good food, fresh, bracing air, and by tonic medicines, such as quinine, iron, and cod-liver oil.

## HYDROTHORAX

The term is applied to the collection of fluid in the pleural cavity, not as a result of inflammation, but in consequence of heart disease or Bright's disease, cirrhosis of the liver, or interference with the circulation in the chest by growth. It is, indeed, dropsy of the pleural cavity; and the liquid contains less albumin and less fibrinogen than are found in pleurisy. Its physical signs are similar to those of pleuritic effusion, but the rub is, of course, absent. Arising, as it often does, from a general or central cause, it is much more often bilateral than pleurisy is; but occasionally a very large one-sided effusion may be merely dropsy. The recognition of hydrothorax, however, generally depends on the history and the previous existence of the diseases which cause it. When the liquid has been removed, the kind of cellular elements it contains may help the diagnosis (*see* p. 276). If acetic acid is added to an inflammatory effusion, a white turbidity is produced. This does not occur with a passive effusion.

Its **Treatment** is mostly of secondary importance, being involved in that of the lesion which causes it. As the liquid is almost certain to recur if removed, paracentesis or aspiration should only be performed when a very large effusion, whether on one side alone or divided between the two, is seriously impeding respiration.

## HÆMOTHORAX

By this term is meant the effusion of blood in quantity into the pleural cavity; it is not used for the merely blood-stained serous effusions so common in pleurisy.

Hæmothorax commonly results from wounds, injuries or from rupture of a thoracic aneurysm. In the case of wounds the greatest danger to life is from the blood becoming infected from the outside. It occurs sometimes in tuberculosis of the pleura, or from rupture of a pulmonary vessel into a phthisical cavity and later extravasation into the pleura. Exceptionally it occurs from bursting of an emphysematous bulla (Newton Pitt); or from degenerated vessels in association with cirrhosis of the liver, granular kidney, or dilated heart; or from malignant disease. And sometimes it appears to be primary, and the origin is never explained.

The **Physical Signs** are those of liquid in the pleural cavity. In hæmothorax following wounds the diaphragm on the same side is high and immobile: the lung is much compressed, and above the fluid it is greatly relaxed, so that Skodaic resonance is especially marked.

The **Diagnosis** will depend, in the case of aneurysm, on the previous history, and on syncope and pallor indicating rapid loss of blood. A hæmothorax may only be discovered on exploration.

**Treatment.**—If the liquid be aspirated, it is very likely to return; and probably it is better to leave the blood to be absorbed, unless it is causing distress by direct pressure. In the case of wounds a moderate hæmothorax may be aspirated a week after the wound, and it is often an advantage to replace it by oxygen. A septic hæmothorax requires free drainage.

## CHYLOTHORAX

Rarely an effusion into the pleural cavity is found to be white and milky, like the fluids sometimes present in the peritoneal cavity. In some cases it is a true *chylothorax*, in others a *chyliform effusion*, in which the milky appearance is not due to the fatty elements of chyle, but to granules of a lecithin compound (*see* Chylous Ascites). The causes are the same as in the case of the peritoneum. Cases of chylo-hæmothorax have been recorded as a result of injury to the thoracic duct in the chest.

## PNEUMOTHORAX

**Pathology.**—The presence of air in the pleural cavity constitutes a *pneumothorax*. If serum is present at the same time, it is a *hydro-pneumothorax;* if pus accompanies the air, a *pyo-pneumothorax;* if blood, a *hæmo-pneumothorax*.

Air may gain an entrance to the pleural cavity—(*a*) through an opening in the chest wall, (*b*) through a breach in the surface of the lung, or (*c*) occasionally from the rupture of some other air-containing viscus in the neighbourhood. (*a*) A pneumothorax may be brought about by any wound in the side which passes through the whole thickness of the chest wall; it is commonly produced artificially when a piece of rib is resected for empyema (pyo-pneumothorax), or in the treatment of phthisis by inducing artificial pneumothorax. (*b*) It is produced by a fractured rib puncturing both layers of the pleura, so as to let out air from the lung into the pleural cavity, while the skin remains intact. In nine out of ten cases of pneumothorax occurring spontaneously from rupture of the surface of the lung, the result is due to phthisis when a vomica ulcerates through into the pleural cavity; and less commonly an empyema makes its way through the pleura into the lung, and air passes into the pleural sac, so as to form a pyo-pneumothorax. In acute pneumonia the pleura has been known to rupture, so that air has escaped and a pneumothorax has been formed; and a pyæmic abscess or gangrene of the lung may lead to a similar result, or an emphysematous bulla may burst. Very occasionally a pneumothorax occurs spontaneously

from rupture of the lung in quite a healthy person, possibly as the result of some sudden strain. (*c*) Air may also enter the pleura in consequence of a spinal or mediastinal abscess burrowing into the pleura; and ulcer or carcinoma of the stomach, or carcinoma of the œsophagus, may let in air from the alimentary canal.

A pneumothorax may be described as open, closed, or valvular, according to the condition of the opening which created it.

*Open Pneumothorax.*—When air enters the chest from an external wound, and the wound remains patent, the lung collapses by its own elasticity; and not only the lung of the wounded side, but also the opposite lung, may contract somewhat and draw with it the mediastinum, so that slight lateral displacement of the viscera takes place, just as it does in liquid effusion. The same thing may happen if the pneumothorax results from rupture of a cavity in phthisis, supposing the aperture to remain patent, so as to keep the pleural sac in communication with the bronchial tubes. In both these cases the mean pressure of the air in the pneumothorax is equal to that of the atmosphere.

*Closed Pneumothorax.*—When the aperture is small it may be quickly closed by lymph; further extravasation is prevented, and the air may then be completely absorbed. This happens in cases of laceration of the pleura by fractured rib, and sometimes in pneumothorax from disease of the lung. In closed pneumothorax there is a slowly progressive absorption of the enclosed air; the pressure is negative, and the displacement of organs is, *cæteris paribus*, less than in open pneumothorax.

*Valvular Pneumothorax.*—A third possibility is that a shred of pleural membrane or lymph hangs over the aperture, so as to form a valve. The air is then drawn into the pleural sac by inspiration, but is unable to escape during expiration; the mean pressure becomes positive—that is, it exceeds the pressure of the atmosphere, and the displacement of viscera and distension of the chest may be extreme; thus the heart may be pushed far over to the opposite side, and the liver or spleen may be driven down by the flattening or inversion of the diaphragm. A valvular opening may, like others, become closed by adhesions.

The amount of collapse of the lung, and displacement of the viscera, is influenced in different cases by the previous condition of the lung. If the lung is extensively diseased in phthisis, or in great part adherent, the collapse will be less than if the lung is for the most part healthy.

**Physical Signs.**— Resonance depends on the presence of a cavity and of elastic walls capable of vibrating in unison with the air waves (*see* p. 190). The elasticity of the walls depends largely on the pressure of the air inside the cavity. If the pressure is very high, as in a valvular pneumothorax, the note is dull. It may also be dull if the pressure inside is the same as the atmospheric pressure, as occurs after the resection of a rib for empyema. Under favourable conditions of pressure, whether the latter is greater or less than the atmosphere, a tympanitic note is obtained on percussion, and this is accompanied by the *bruit d'airain*, or *bell sound* (*see* p. 196). and in very favourable circumstances by *metallic tinkling* (*see* p. 195). The dependence of the *bruit d'airain* on the elasticity of the walls of the cavity. which itself is determined by the pressure of the contained air, can be demonstrated by the student to himself as follows: He closes his mouth, blows out his cheeks, lays one coin on the cheek and strikes it with another. A musical sound is obtained if the pressure exerted by the cheeks on the enclosed air is correctly adjusted. Another characteristic sound is due to the dropping of fluid from the upper part of the chest into the liquid below, the noise being reverberated with almost musical quality. The respiratory murmur is often entirely inaudible, or faint amphoric breathing is present. When this kind of breathing is loud or well marked, it is probably due to an opening leading from a bronchus into the pneumothorax; but a fainter sound may occur even when adhesions

## PLATE VI.

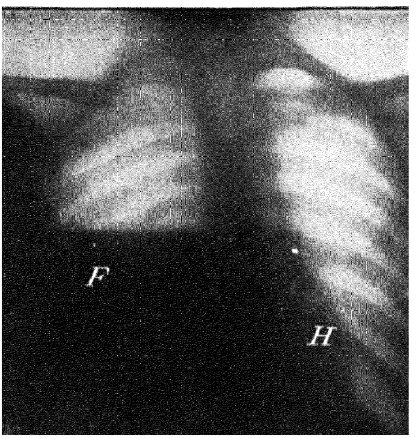



FIG. 1.—Radiogram of Chest in case of Pyo-pneumothorax. The fluid (*F*) at the left base is separated from the air by a sharp horizontal line. On shaking the patient this line is seen to break into waves. The heart (*H*) is displaced into the right chest. Seen from behind.

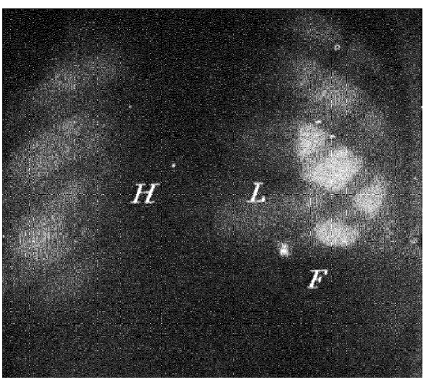



FIG. 2.—Radiogram of Chest in case of Pyo-pneumothorax. The fluid (*F*) at the left base is separated from the air (clear) by a sharp horizontal line. The collapsed lung (*L*) is seen as a vertical shadow on the inner side. The heart (*H*) is displaced into the right chest. Seen from the front. (Taken by Dr. A. C. Jordan.)

[*To face p.* 281.

have shut off the lung from the pleural cavity. Vocal resonance and tactile vibration are generally much diminished, but bronchophony or pectoriloquy may be present, when a bronchus opens into the cavity.

If there is also liquid effusion, it gravitates to the lowest part of the chest under all circumstances. If the patient is recumbent, the posterior part of the chest is dull, and the anterior part is tympanitic; if the patient now sits up, the lower part of the chest, back and front, becomes dull, while the upper part, back and front, is resonant. If *Hippocratic succussion* be employed, a splashing sound will be obtained (*see* p. 195).

The **Symptoms** of pneumothorax are very variable, depending largely upon the amount of antecedent disease. If it supervenes upon a lung extensively diseased, it may add but little to the distress already present; if it occurs in a lung for the most part, or entirely, sound, the symptoms will be pronounced; lastly, if in a case of phthisis with extensive disease on one side pneumothorax occurs on the other side, the result may be quickly fatal. The symptoms in the severe cases are sudden pain with a sense of something giving way internally, then distress of breathing, with more or less collapse, small pulse, lividity, and sweating. The breathing is rapid; the chest is distended on the affected side, and the intercostal spaces are depressed on inspiration.

These troubles may be aggravated until death takes place within a few hours or two or three days, or the first severe symptoms may subside, and comparative ease may follow, but generally with rapid breathing and orthopnœa.

**Diagnosis.**—*Emphysema* may be for a moment confounded with pneumothorax, but it is always bilateral unless compensatory on one side to disease on the other. A very large *cavity* in phthisis may sometimes simulate a localised pneumothorax in its hyper-resonance, feeble vesicular murmur, and tinkling sounds; but *bruit d'airain* must be rare in vomica, and the flattening of the chest over a cavity will generally serve to distinguish it from a pneumothorax. On the other hand, in some cases pneumothorax may be overlooked from the absence of any special symptoms at the time of its occurrence. Obstruction of a bronchus will cause the disappearance of the vesicular murmur; and in the early stages of compression by an aneurysm there may be at the same time so much distension of the lung as to yield hyper-resonance, and thus give rise to a mistaken diagnosis of pneumothorax (*see* p. 230). A *diaphragmatic hernia*, that is, the escape of the stomach or the colon into the thorax through an aperture in the diaphragm, may closely resemble pneumothorax in its physical signs, and it may arise in the same way, from a contusion of the chest. Pneumothorax may also be simulated by an unusually high position of the stomach in the chest in consequence of contraction of the left lung, and by abscess beneath the diaphragm containing air (*subphrenic pneumothorax*). The Röntgen rays show the transparency due to air in the pleural cavity, the collapsed lung, the depressed diaphragm on the same side, and the displaced heart (*see* Plate V., Fig. 2, and Plate VI.). The presence of fluid in a pneumothorax produces a very characteristic X-ray appearance when the patient is examined in the vertical position, because it is opaque and its upper surface is a horizontal line, while above it there is transparency owing to the air. Further, on inclining the body the surface of the fluid still remains horizontal.

**Prognosis.**—The occurrence of spontaneous pneumothorax in phthisis is, on the whole, a favourable event, and cases have improved subsequently (*see* Artificial Pneumothorax). At the same time it is usually only in the last stages of phthisis that it occurs, so that it is often an indication that death is near. In other cases the prognosis is, on the whole, good with suitable treatment, though it depends on the attendant circumstances.

**Treatment.**—This is, in the main, palliative. For the intense pain and distress accompanying the rupture opium or a subcutaneous injection of morphia ($\frac{1}{3}$ to $\frac{1}{2}$ grain) should be administered, and hot poultices and fomentations should

be frequently applied. Stimulants, as wine, brandy, or ether, may also be required. In cases of extreme distension it may be desirable to perform paracentesis, a trocar and cannula being inserted between the ribs over the resonant area and the air allowed to escape till atmospheric pressure is reached; the relief is, as a rule, only temporary, and the paracentesis may have to be repeated. Aspiration is generally undesirable, because it may keep open the aperture in the lung, and may draw septic matter from the lung into the pleura. If the communication with the lung becomes closed, the air will probably be absorbed; but to facilitate its absorption it may be replaced by oxygen. In hydro-pneumothorax the serum may be left alone, or, if present in large amounts, it may be removed by paracentesis; but here again it should be replaced by oxygen. If a pyo-pneumothorax is producing constitutional symptoms, *i.e.* temperature, rapid pulse, etc., it should be treated like an empyema with rib resection and drainage. Otherwise the liquid may be dealt with like serum.

## DIAPHRAGMATIC HERNIA

This condition, which is of rare occurrence, may be mentioned here, because the contents of the thorax are necessarily modified by the invading viscus, and physical signs are produced which may closely resemble those of pneumothorax. In diaphragmatic hernia one or more of the abdominal contents, generally a portion of the stomach, or of the omentum, or of the colon, passes upwards into the thorax through an aperture in the diaphragm. Mostly the aperture is the result of injury, such as a forcible sudden compression of the chest, or it is a congenital defect, or the enlargement of a natural opening. The lesion is more frequent on the left side, and the stomach is commonly the viscus, which passes into the thorax.

**Symptoms.**—When the diaphragm is ruptured by injury, the early symptoms, such as pain, dyspnœa and collapse, are partly due to the direct effects, and partly to the sudden disturbance of the contents of the thorax with compression of the lung on the same side. Pleurisy and peritonitis may also supervene with the symptoms referable to them. But in many cases the first effect of the injury passes off, and the symptoms are partly pulmonary and partly gastric or gastro-intestinal. The pulmonary symptom is mainly shortness of breath, but it is remarkable how little disturbance may be felt on the side of the chest in some cases. On the side of the abdomen are generally observed indigestion, pains, flatulence, and perhaps vomiting. These symptoms may come on periodically, being due to distension or kinking of the viscera in their abnormal situation. In a recent case severe symptoms were noticed at periodic intervals, when for some reason the stomach was forced up through the diaphragm compressing the lung.

The physical signs noted in the chest are hyper-resonance in the lower part occupied by the herniated viscus, with loss of breath sounds, gurgling sounds heard on succussion or spontaneously, metallic echo and the *bruit d'airain*. The heart may be displaced if the visceral transfer is considerable.

*Subphrenic pneumothorax* may also present somewhat similar physical signs. It is more common on the right side, and the liver is depressed by it into the abdomen. The hyper-resonance resulting from it is not likely to rise so high in the chest as in either of the other two cases, and the history will probably assist in the diagnosis.

**Diagnosis.**—The diagnosis of diaphragmatic hernia can only be made with any certainty by X-ray examination after an opaque meal. In the case of the stomach the shadow due to the meal will be seen in the chest, if the patient is examined in the horizontal position (Symonds).

**Treatment.**—Some patients are not very seriously inconvenienced after

the first troubles have passed. Surgical treatment involves opening the chest, removing portions of ribs, returning the viscus to the abdomen, and sewing up the diaphragm.

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# DISEASES OF THE ORGANS OF CIRCULATION

In the working of the normal heart, two kinds of structures are concerned: the muscular contractile walls of its cavities, which drive the blood, and the valves, which control the direction of its flow. Before describing the methods of investigation in diseases of the organs of circulation, some reference may be made to the structure and mode of action of the heart's muscle. The power of rhythmical contraction is inherent in the heart muscle. The researches of the last few

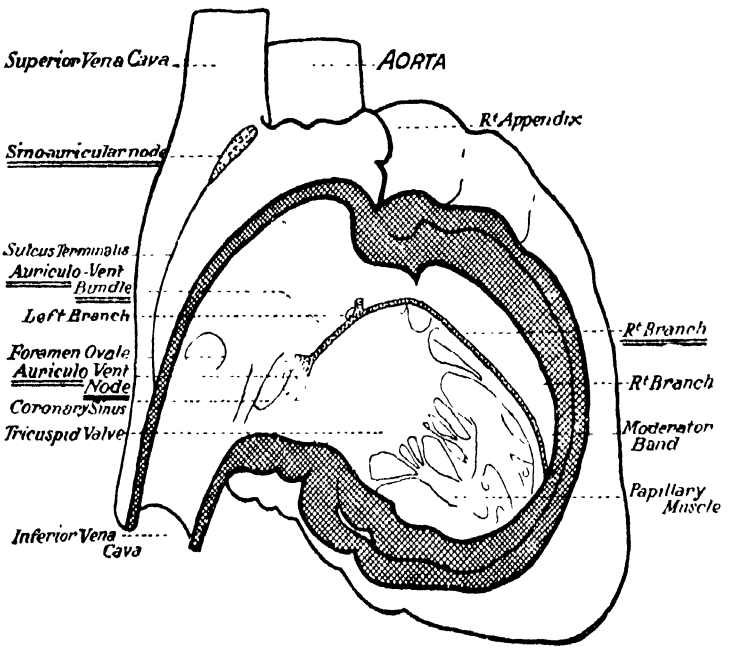



Fig. 21.—Diagram of Interior of Heart, showing position of nodes and auriculo-ventricular bundle. (*After Starling.*)

years have shown us the points in the myocardium at which stimuli to contraction commonly arise, the paths of conduction of stimuli from auricle to ventricle, the normal rate of such conduction, and the fact that contraction, though usually initiated in the auricle, and passing to the ventricle, may, in certain circumstances, be started in the ventricle. As is well known, after each ventricular systole there is a period of rest from contraction, which is broken by the systole of the auricle, and this is immediately followed by the systole of the ventricle. Three structures are involved in this process: one a small elongated mass of nerve tissue and muscular fibre and cells situated near the attachment of the superior vena cava to the auricle, called the *sino-auricular node;* another, a small mass situated in the septum of the auricles, near the opening of the coronary sinus, called the

*auriculo-ventricular node*: the third, a special band of muscular fibres known as the *auriculo-ventricular bundle* (*see* Fig. 21). This band arises in the auriculo-ventricular node and measures about 2·5 mm. in breadth: it passes from the auricular septum into the ventricular septum, lying at first below the *pars membranacea septi*, and there divides into two portions, which lie one on each side of the septum ventriculorum; the right branch passes into the moderator band. Each branch is distributed extensively in the wall of its own ventricle, terminating in so-called Purkinje's fibres, which lie beneath the endocardium in nearly all parts of the ventricles. The auriculo-ventricular node and the auriculo-ventricular bundle are sometimes called *junctional tissues*. In the normal action of the heart, the stimulus starts in the sino-auricular node, and is transmitted to the auricles. The wave of contraction which is slightly preceded by an electrical wave, called the excitation wave, spreads out radially from the S.A. node, travelling with equal velocity in all directions. The A.V. node then becomes stimulated. The excitation from the A.V. node spreads down the A.V. bundle along its various ramifications, and is spread out in the Purkinje network over the endocardium of the ventricles, and from here it spreads at right angles into the muscle of the ventricle.

Such a complicated mechanism presents many opportunities for processes of disease to attack it. The results will be considered later under the heading Abnormalities of Cardiac Action.

The heart muscle is supplied by two sets of nerve fibres: (1) from the vagus; (2) from the sympathetic. Their function is to modify the beat. Very little is known about the sympathetic apart from the fact that its stimulation accelerates the heart and strengthens the beat. Branches from the vagus end both in the S.A. and A.V. nodes. Stimulation causes the seat of origin of the excitation wave (pacemaker) to move from the upper to the lower end of the S.A. node. Other effects are described under Bradycardia.

## EXAMINATION OF THE HEART

Like the lungs, the heart is accessible to examination by the eye, the hand, and the ear. It comes into close proximity with the chest wall between the anterior margins of the lungs, over an area corresponding to the lower half of the sternum on the left of the middle line, and the inner portions of the fourth and fifth left costal cartilages and the spaces below them. The *impulse* of the heart can be determined by inspection and palpation; the *præcordial area*, or the area of chest wall over the heart, can be made out by percussion, and the *heart sounds* can be studied by auscultation.

### INSPECTION

In healthy persons, not very fat, the heart can be seen to beat over an area of about ½ inch in diameter in the fifth intercostal space, from ½ inch to 1 inch within a line drawn vertically down from the nipple, or from 2½ to 3 inches from the middle line in an average-sized adult; this beat is called the *impulse*, or *apex beat*.

In disease, various changes take place in the position and the character of the impulse. It may be displaced outwards and downwards. In extreme cases it may be felt in the axilla. In addition to the impulse in the usual position, there may be one below the ensiform cartilage (lower part of the right ventricle). The right auricle, when dilated, can sometimes be seen beating to the right of the sternum. Sometimes no impulse can be seen at all, either from feebleness of beat, or because the heart is overlaid by lung. In character the beat may be unusually forcible, or heaving, or quick, or irregular.

Inspection also shows bulging of the chest wall in some cases of great enlargement of the heart.

Movements in the epigastrium are often produced by the heart's contractions. A slight *systolic retraction* is not uncommon in healthy persons; a more marked retraction occurs with hypertrophy of the heart. A visible *systolic* impulse may be produced in the epigastrium by a hypertrophied right ventricle, by the aorta, whether aneurysmal or conducted by tumour, or by a pulsating liver.

## PALPATION

Examination with the hand confirms much that can be seen with the eye as to the position and character of the impulse; the position of the latter can usually be more closely defined by palpation than by inspection. Examination by X-rays shows that the left border of the heart usually corresponds with the point of maximum vibration at the impulse, and not with the outer and lower part of the area over which the vibrations can be felt. Occasionally X-ray examination shows that the outline of the heart does not extend as far to the left as the determination of the impulse would lead the observer to expect. In some cases of aortic aneurysm the hand placed over the base of the heart can appreciate a shock described as the *diastolic shock*, or *diastolic rebound*, which can be readily felt because the aneurysm compresses the lung and comes into closer contact with the thoracic parietes.

In some cases of valvular disease, over a limited area in which a murmur can be heard with the stethoscope, a *thrill* (*frémissement cataire*) can be felt by the hand. It is usually accompanied by a murmur, because the vibrations which cause the sound are of sufficient intensity to be felt also. Occasionally the murmur is absent, because the vibrations are so coarse (*i.e.* their rate is so slow) that they can only be felt, not heard. It is most common in mitral stenosis, and accompanies a large proportion of pre-systolic (or late diastolic) murmurs, and some mid-diastolic murmurs. Thrills with other murmurs are much less common, and the valvular lesions which they accompany may be arranged in the following order of frequency: pulmonary stenosis (congenital), tricuspid regurgitation, aortic stenosis, aortic regurgitation, mitral regurgitation, tricuspid stenosis. Aneurysms, imperfections in the septum ventriculorum, and lymph in pericarditis also give rise to palpable vibrations.

## PERCUSSION

While the greater part of the chest is resonant to percussion, from the presence of the lung, there is a small area over the surface of the heart which is not resonant. This *præcordial dulness* does not correspond to the whole anterior surface of the heart, but to what is exposed between the vertical anterior edge of the right lung and the oblique anterior edge of the left; and not all of this, because the sternum is normally resonant even up to its left border. Its limits are as follows: Above, the upper border of the fourth costal cartilage; below, the upper border of the sixth cartilage; internally, the left border of the sternum; and externally, a vertical line from $\frac{1}{2}$ inch to 1 inch within the nipple. Around this dulness above and to the right and to the left, is an area of less dulness, the outer limit of which corresponds to the outline of the heart, and therefore maps out its actual size; this reaches above the upper border of the third rib, or of the third space, to the left it reaches nearly the nipple line, and to the right the right border of the sternum; but the dulness is least marked over this bone. The former central area of dulness is often called *superficial* or *absolute dulness;* the latter surrounding band is called *deep* or *relative dulness*. It is the latter that is of greatest importance, as it corresponds with the actual borders of the heart. The relative dulness is best obtained by means of moderately light percussion. The room must be quiet. X-ray examination shows that the border of the heart usually corresponds to the point where the note first undergoes a considerable change in resonance. The lower limit of the heart's dulness cannot be discriminated from

that of the liver, and the outline is assumed to lie between the impulse and the lowest point of the right border of the dulness.

The position of the heart varies slightly with the position of the patient. The area of its projection on the chest wall extends rather lower, but it is less wide in the vertical than in the horizontal position.

When the lungs are distended, as in emphysema, it is usually quite impossible to tell the size of the heart by percussion, and in addition the impulse is often too weak to feel. An important cause of the enlargement of the area of relative dulness is distension of the pericardial sac with liquid. Exceptionally, the area may be resonant from the presence of air in this sac. The area of præcordial dulness is shifted upwards, downwards, or to either side by anything which displaces the heart in these directions.

## AUSCULTATION

With the stethoscope we hear over the cardiac region the well-known sounds of the heart, resembling the syllables "lubb dup"; the *first*, or *systolic*, duller and longer, and the *second*, or *diastolic*, sharper and shorter. The first sound is due partly to muscular contraction, and partly to sudden stretching of the auriculo-ventricular valves after closure; the second is due to sudden stretching of the semi-lunar valves after they close. In the case of both valves, the flaps normally float together immediately after the blood has stopped streaming through the orifice (**1**). The first sound is heard best near the apex of the heart, and the second is heard best at the base.

**Modifications of the Sounds.**—The heart sounds may be accentuated or diminished in loudness, or increased in number, or their time relations may be altered.

*Accentuation* arises from several causes, amongst others from retraction of the lung, so as to bring the heart closer to the chest wall; and from increased tension in the aortic or pulmonary arterial system, whereby the valves are caused to close with unusual force. This last condition affects, of course, the second sound, and it may be determined whether the aortic or pulmonary system is at fault by examining successively on either side of the sternum in the second intercostal space. On the right side, the aortic second sound can be heard more or less apart from the pulmonary; on the left side, the pulmonary apart from the aortic. Accentuation of the first sound results from excessive action of the heart, and is common also in mitral stenosis.

*Diminution* of the sounds results from feeble action of the heart, from its being unusually covered by lung, as in emphysema, or from its being surrounded by pericardial effusion. Any one heart sound is likely to be diminished if the mechanism of the valve, whose vibration causes, or contributes to it, is interfered with; and as this disordered mechanism may lead to an abnormal sound or *murmur* (*see* below), it is common to find that when a murmur of regurgitation exists at any one of the four orifices, the sound which ought to be heard at that moment is diminished or lost. But there is no necessary loss in the case of an obstructive murmur; for the normal heart sound with which it corresponds in time is not produced at the same orifice as the murmur. Thus, during an aortic obstructive and systolic murmur, the first sound is efficiently produced by the auriculo-ventricular valves and the cardiac muscle.

Sometimes more than two sounds are heard, the extra sound occurring either near the first or near the second sound. The condition is known as *reduplication* of the first or second sound.

Reduplication of the *second* sound is commonly heard at the base of the heart, especially in the pulmonary area, and it is occasionally conducted to the apex. It occurs in cases of valvular disease where there is congestion of the lungs and increased pressure in the pulmonary circuit. It is due to the fact that the pul-

monary and aortic valves do not close quite synchronously. It is probable that reduplication of the *first* sound, heard at the apex, may sometimes be due to a synchronous closure of the A.V. valves. In *canter-rhythm* there is very well marked reduplication of the first or second sound at the apex, so that there is a triple rhythm of two types, "te-lubb dup" and "lubb dup-te." There are two causes for canter-rhythm: (1) The extra sound is due to the same cause as often produces an early or late diastolic murmur in this position; *i.e.* it is an indication of mitral stenosis; (2) it is due to heart block (*q.v.*), and may be the sound actually due to contraction of the auricle. Under ordinary circumstances this is not heard, because it is so close to the ventricle that the sound of the latter swamps it. In heart block the auricle contracts some time before the ventricle, and the sound of its contraction may be heard early or late in diastole, constituting a reduplicated second or first sound respectively (Lewis).

The more quickly the heart beats, the shorter is the pause between the second sound and the following first sound. The two intervals may become equal in the rapid action which accompanies some forms of cardiac exhaustion. The heart beats are feeble, the first sound cannot be distinguished from the second, and there is a close resemblance to the sounds of the fœtal heart. The condition is called *fœtal rhythm*, or *embryocardia*.

In healthy people the first sound at the apex has twice the intensity of the second sound at the base over the aortic area. In cases of myocardial degeneration the first sound as heard at the apex is diminished in intensity until it becomes the same as or less than the intensity of the aortic second sound at the base. A special stethosocope has been devised for comparing the two, but usually the ear is quite capable of making out the difference by means of an ordinary stethoscope.

**Murmurs.**—These are adventitious sounds, which accompany or replace those which are physiological. Originally described as *bruits de souffle*, they are now commonly called *murmurs* or *bruits*, and it will be convenient to divide those that are heard over the præcordium into two classes: (*a*) those which are due to disease at the valvular orifices, and (*b*) those which are brought about by other means.

**Murmurs due to Disease at the Valvular Orifices.**—Any narrowing or *obstruction* of a cardiac orifice, such as is produced by vegetations on the valves, or by union of the valves together, will produce vibrations in the currents of blood forced through them, which will be audible as a murmur. On the other hand, if the valve is *incompetent* and does not perfectly close the orifice, some blood will flow back, or *regurgitate*, into the cavity whence it came, and a murmur will also be heard. It has been found experimentally that no sound is produced with liquid running along a pipe of uniform bore or from a wider into a narrower pipe, but that there is a sound when the fluid runs from a smaller pipe into one of larger bore, or through a narrow chink into a wider space beyond. The explanation is that the stream travels at a higher velocity in the narrower tube, and so becomes projected forward into the slowly moving fluid of the larger tube in the form of a "fluid vein," and the friction of this against the surrounding fluid causes it to break up and form eddies. The resulting vibrations are audible as sound. A waterfall provides an every-day illustration of the principle, but it is widely applicable to the human body. The murmurs produced by the constriction and incompetence of valves have already been mentioned, though it must be pointed out that an additional cause of the murmur may be the vibrations of the edges of the valves or of the vegetations in the blood stream. The principle of the fluid vein also explains the murmurs resulting from the passage of blood through a perforation in the septum ventriculorum. Normally, no sound will be heard when blood passes from the large into the smaller arteries; but there will be a sound if pressure is exerted on the artery; in fact, the diastolic pressure may be determined by such means. Similarly, a murmur may be heard if the large

veins are pressed on, and in arterial aneurysm, and arterio-venous aneurysm. Finally, the same principle is involved when sounds are produced by moving currents of air during respiration. Sounds may be produced at the mouth, as in whistling; at the external or posterior nares; at the glottis; or between the respiratory bronchioles and the air sacs (see Fig. 19, p. 193).

Cardiac murmurs differ from one another—(1) in time; (2) in their relation to the orifices of the heart; (3) in the character of the sound.

*The Time of Murmurs.*—Murmurs which are heard with the first sound, or between the first and the second sounds, occur during the contraction of the ventricles, and are called *systolic;* those which are heard with the second sound, or between it and the succeeding first sound, occur during the dilatation of the ventricle, and are called *diastolic.* Of these last, some begin at the very commencement of diastole and end before the next first sound; others commence a little later, but still end before the first sound; and others, again, begin after the second sound, and run up to, and finish in, the next first sound. These may be

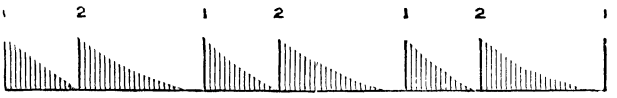



FIG. 22.—Aortic Disease. Systolic and Diastolic Murmurs

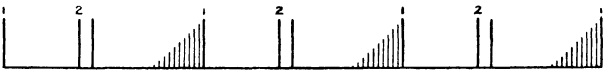



FIG. 23.—Mitral Disease. Auriculo-systolic Murmur, with Reduplicated Second Sound.

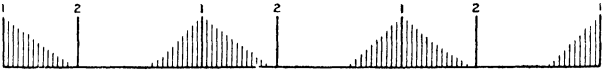



FIG. 24.—Mitral Disease. Auriculo-systolic and Systolic Murmurs.

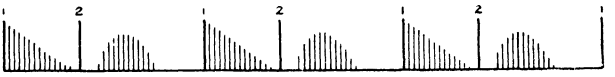



FIG. 25 —Mitral Disease. Systolic and Mid-diastolic Murmurs.

In the above diagrams Nos. 1 and 2 represent the first and second sounds respectively, and the shaded lines between them show the periods of time occupied by different murmurs.

called respectively *early, mid,* and *late* diastolic. The last is more commonly known as *pre-systolic,* and when, as in most cases, it is due to blood being forced through a narrow opening by the systole of the left auricle, it may be called *auriculo-systolic.* In determining the rhythm of a particular murmur, its position should be noted in reference to the beat of the heart, or to the beat of the carotid artery by the side of the thyroid cartilage. Either of these represents the systole of the ventricle with sufficient accuracy, but the radial pulse is one-tenth of a second later than the carotid. If the normal heart sounds are represented by "lub-dup," a systolic murmur may be represented by "luff-dup," and a mitral diastolic murmur by "lub-d-drup."

*Relation to the Valvular Orifices.*—It must now be pointed out that at certain orifices the obstructive murmurs are systolic, and the regurgitant are diastolic, while at the other orifices the regurgitant murmurs are systolic, and the obstructive are diastolic.

Thus, in obstruction at the aortic orifice the contraction of the ventricle causes a murmur by forcing blood past the obstruction; hence it is a *systolic* murmur (Fig. 22, between 1 and 2).

Regurgitation at the aortic orifice, caused by incompetence of the valves, gives rise to a murmur during the relaxation of the ventricle, because when the ventricle ceases to contract, the aorta recoils upon the column of blood within it, and forces it against and partly through the now incompetent sigmoid valves; hence there is a *diastolic* murmur (Fig. 22, between 2 and 1).

Regurgitation at the mitral orifice causes a murmur during the contraction of the ventricle, because it is during systole that these valves are called into play; hence with their failure a *systolic* murmur results (Figs. 23 and 24, between 1 and 2).

Obstruction at the mitral valve causes a murmur during the relaxation of the ventricle, because only during this relaxation can a current of blood pass from the auricle into the ventricle. Hence, such a murmur must be *diastolic* in the sense that it takes place during ventricular relaxation. The murmur will tend to occur when the rush of blood from auricle to ventricle is at a maximum, *i.e.* when the difference of pressure is greatest. The knowledge of the alterations of pressure in auricle and ventricle during the cardiac cycle indicates two places during diastole where the pressure difference will be greatest (*see* Fig. 26). The first is shortly after the beginning of diastole, *i.e.* after the second sound, when the

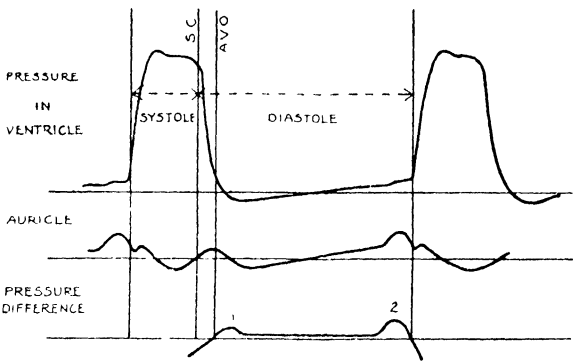



FIG. 26.—Lewis' Explanation of the Murmurs of Mitral Stenosis. S.C. Closure of semilunar valves, marking time of second sound and beginning of diastole. A.V.O. Opening of A.V. valves. 2 represents the position of the auriculo-systolic murmur. 1 represents the mid-diastolic murmur. The latter is really rather a misnomer as the murmur occurs before mid-diastole; but it begins after the beginning of diastole, so it is not early diastolic; it is really *delayed* early diastolic.

ventricle is fully relaxed, but empty, and the auricle is engorged with blood which has been collecting since the last auricular systole. The second place is during auricular systole itself, when the intra-auricular pressure is raised considerably. Consequently the two commonest murmurs in mitral stenosis are the mid-diastolic, which starts soon after the second sound (Fig. 25), and the pre-systolic or auriculo-systolic, which ends in the first sound (Figs. 23 and 24). Reference has already been made to the fact that a reduplicated second sound at the apex is often due to mitral stenosis (Fig. 23). It is produced in the same way as the mid-diastolic murmur. Occasionally an early diastolic murmur is heard in mitral stenosis, *i.e.* a murmur beginning at the second sound. This occurs particularly if the heart is beating quickly. It is not common.

The mid-diastolic murmur may be soft and blowing in character and quite short, or it may be rumbling and continue through most of diastole, and this suggests a high degree of stenosis. The auriculo-systolic murmur gets gradually louder, and ends in an accentuated first sound. It may be represented by "r-r-rup."

It is possible to have both mid-diastolic and auriculo-systolic murmurs pre-

sent at the same time, or an auriculo-systolic murmur may be accompanied by a reduplicated second sound (Fig. 23).

What is here stated of the aortic and mitral valves may be said, *mutatis mutandis*, of the pulmonary and tricuspid valves.

The relation of the murmurs to the rhythm of the heart and the flow of blood through it may be tabulated as follows :

| Orifice. | Lesion. | Murmur. | |
|---|---|---|---|
| Aortic or Pulmonary. | Obstruction. | Systolic. | |
| | Regurgitation. | Diastolic. | |
| Mitral or Tricuspid. | Obstruction. | Diastolic | Early.<br>Mid.<br>Late (Pre-systolic or auriculo-systolic). |
| | Regurgitation. | Systolic. | |

Of these the pulmonary regurgitant and tricuspid obstructive murmurs are very rare ; and murmurs due to pulmonary obstruction are less frequent than the remaining five, although a systolic murmur over the region of the pulmonary artery is quite common in association with changes in the quality or quantity of the blood, and is known as a hæmic murmur.

Obviously, the eight possible lesions above indicated (obstruction and regurgitation at each of the four orifices) cannot be distinguished solely by the relation of their murmurs to the sounds of the heart. But we find some help towards discrimination in the different points of the præcordial area at which the several murmurs are best heard ; and these are determined not so much by the actual position of the valve below the surface as by the direction of the current of blood which is flowing past the orifice at the time, and in which, indeed, the sound vibrations are largely or entirely produced. Indeed, three of the orifices (aortic, mitral, and tricuspid) lie so close together that if the murmurs were heard only at the orifice concerned, it would be very difficult to distinguish the different kinds. But the flow of blood in the aorta from mid-sternum towards the right clavicle, in the pulmonary artery from the sternum upwards towards the left, and in the heart from auricle to ventricle, conveys each murmur along a special path ; and the reflux of blood through the aortic valves into the ventricle, and through the mitral valves into the auricle, acts in a similar way in the case of regurgitant murmurs. The term *area* (mitral area, aortic area) is often applied to the part of the præcordia or adjacent chest wall where a particular murmur is commonly heard, and in auscultating the heart for valvular disease these areas must be successively examined.

*Aortic obstructive* murmurs are heard with greatest intensity at the junction of the second right costal cartilage with the sternum, and at the extremity of the second right intercostal space (aortic area) ; they can be traced upwards towards the inner half of the right clavicle, and into the vessels of the neck, and they are sometimes heard in the right supraspinous fossa.

*Aortic regurgitant* murmurs are heard over the aortic area ; they are traceable down the sternum or along the left-hand side of it, towards the apex of the heart, *i.e.* along the line of the regurgitating stream of blood. They are usually loudest to the left of the sternum, and sometimes this is the only place where they can be heard.

*Mitral obstructive* murmurs are heard most loudly at the point of impulse of the heart against the chest (mitral area) ; though sometimes audible more or less imperfectly between this point and the sternum, they are always best heard at the apex, and are often strictly limited to an area of an inch or an inch and a half in diameter. The stethoscope should always be placed over the actual heart beat, as found by examination, and not only over the spot where the impulse should be normally found. They are heard best if light pressure is used.

*Mitral regurgitant* murmurs are mostly heard with greatest intensity at the

apex of the heart, but they are commonly widely diffused, slightly over the præcordial region, towards the sternum and the base of the heart, and more loudly, as a rule, outwards to the left. In the axilla they often lose in loudness, but are again heard at the angle of the left scapula.

*Pulmonary obstructive* murmurs are heard with great intensity in the second left intercostal space at its inner end (pulmonary area), and can be traced outwards in that space, and upwards towards the left clavicle.

*Pulmonary regurgitant* murmurs are heard at the junction of the third left costal cartilage with the sternum, and thence downwards over the right ventricle, along the left border of the sternum.

*Tricuspid obstructive* murmurs are sometimes heard, with a pre-systolic or mid-diastolic rhythm (like mitral obstructive murmurs) at the left side of the sternum, over its junction with the fourth costal cartilage.

*Tricuspid regurgitant* murmurs are heard at the lower half of the sternum, over an area corresponding pretty closely to the part of the heart left exposed between the two lungs; but they are often limited to the base of the ensiform cartilage (tricuspid area).

From the above it will be easily understood that a murmur may be heard over a large extent, and may encroach on the areas of healthy valves: and that, if murmurs are produced at two orifices at the same time, much care may be required to distinguish them.

*The Character of the Murmur.*—The quality of the sound is most often blowing; it is sometimes rushing, sawing, or rasping. Sometimes murmurs have a distinctly musical quality. Half-detached fragments of valve playing in the blood current, perforations in valves, and loose chordæ tendineæ sometimes cause such murmurs. In some cases a murmur, though not strictly musical, has a different pitch at one point from that which it has an inch away.

Murmurs vary with the position of the patient, probably from the effect of gravity upon the velocity of the blood currents. Thus in the recumbent position there is an increase in the loudness of hæmic pulmonary systolic murmurs constantly, and often of aortic systolic, and mitral and tricuspid systolic murmurs; the hæmic murmur is sometimes heard only in the recumbent position. Conversely the erect position intensifies mitral obstructive murmurs, and pulmonary and aortic regurgitant murmurs. There is no hard and fast rule; *e.g.* a crescendo pre-systolic murmur may be only audible when the patient is lying down.

The murmurs due to congenital defects in the heart and aortic aneurysm are described later.

**Murmurs not Dependent upon Actual Disease of either of the Four Valvular Orifices.**—The above descriptions apply to the sounds which result from obstructions and leakages at the four valvular orifices. But abnormal sounds may be heard over the præcordial area, which arise in other ways. Some of these have been called at different times *functional murmurs*, to contrast them with those due to structural disease of the heart or its valves.

Those which we shall consider here are so-called *hæmic* murmurs, the early murmurs of cardiac disease and weakness following rheumatic fever and infectious disorders, murmurs due to dilatation of the cavities, anomalous præcordial murmurs of uncertain origin, exocardial murmurs, and the murmurs of aneurysms, and of congenital malformations.

*Hæmic Murmurs.*—In higher degrees of anæmia such as are seen in chlorosis, in pernicious anæmia, and after great losses of blood, murmurs are heard over the cardiac area.

The most common is a systolic murmur, often harsh in quality, heard loudest in the second left intercostal space, and traceable outwards along that space and towards the left clavicle—that is to say, in the area of the pulmonary artery. This murmur is often loudest in the recumbent position, and diminishes or even disappears when the patient stands up. In some cases a murmur is heard at

the apex, and in more severe anæmia, systolic murmurs may be heard at all the orifices, or even over the whole cardiac area, as well as behind.

The origin of the pulmonary hæmic murmur is obscure, and there may be several factors in its causation. It is probably largely due to the fact that the viscosity of the blood is diminished owing to the deficiency in red cells. Consequently eddy currents are more likely to be set up, even though the relation of the pulmonary opening to the size of the right ventricle and the artery remains unchanged. It is possible that a dilatation of the conus arteriosus of the pulmonary artery may also be responsible.

*The Early Murmurs of Rheumatic Fever.*—The appearance of a soft systolic murmur in the mitral area is very common in rheumatic fever, but may also occur in any other acute infection. It indicates acute infection of the heart, and is certainly due to mitral regurgitation. There may be minute pin-point vegetations on the valves, but it is difficult to see why these should give rise to incompetence. The more probable explanation is, that, owing to myocarditis, there is dilatation of the mitral ring leading to incompetence. These murmurs may disappear as the patient gets better.

Another common murmur, also heard at the apex, in rheumatic fever in children is mid-diastolic in time. This is certainly not in all cases due to mitral stenosis, as post-mortem examination has on more than one occasion disclosed a healthy valve. It is probably due to "relative stenosis," *i.e.* a normal valve opening into a dilated ventricle, and so producing a fluid vein and eddies. It is similar to Flint's well-known murmur in aortic regurgitation. The presence of this murmur in children usually means a severe grade of infection, and it often leads on in later years to a fully developed mitral stenosis.

*Exocardial Murmurs.*—These are sounds of blowing character which are caused not by changes in the interior of the heart, but by sound vibrations produced outside the heart. Some of these are due to the alteration in size of the heart at each beat, which causes movements of the air in the neighbouring part of the lung, and so gives rise to a series of short respiratory sounds with a cardiac rhythm (*cardio-pulmonary*). The most common of these is a short, high-pitched, systolic murmur, often limited to the apex, which is heard in nervous or excited persons when they are under medical examination. Such a murmur is sometimes heard at the left scapula behind, as well as in front. Its exocardial origin is by some thought to be proved, if it disappears on firm pressure with the stethoscope. But pericardial friction sounds, which are certainly produced outside the heart, and may be mistaken for internal bruits, are often increased by pressure. A systolic apex murmur, audible only during inspiration, or as long as the lung is kept expanded, is probably often cardio-pulmonary.

Other exocardial murmurs are produced by displacement of the heart, as when it is compressed by pleural effusion, or by deformities of the thorax; and others by morbid conditions of the lung and pleura immediately adjacent to the heart, and mostly on the left side. Very extraordinary murmurs are sometimes heard when a large pulmonary cavity is in close contact with the heart, the air being driven suddenly out of the cavity with each cardiac impulse.

*The Significance of Murmurs.*—It is a remarkable thing that, considering the large changes in calibre of the passage through which the blood goes—veins, auricle, ventricle, artery—no murmurs are usually produced in the heart. It is easy to imagine that a small alteration of no pathological importance might make all the difference, and cause a murmur.

As the result of experience gained in the War, especially by Lewis and his co-workers, views on the importance of murmurs have changed considerably. Where diastolic murmurs are present, indicating aortic regurgitation or mitral stenosis, the practice is not to place the man in a high category for service. However, the presence of a systolic murmur should be entirely disregarded, since it has been found that the proportion of men who eventually turn out to be unfit

is the same among those who have a systolic murmur as among those who have no murmur at all. No doubt the reason of this is that systolic murmurs heard in apparently healthy men may sometimes be exocardial, but where actual mitral regurgitation is present the lesion may be so well compensated that there is no detectable difference in the heart's efficiency, in the absence of other symptoms.

**Friction Sounds.**—Rubs or friction sounds are exocardial sounds arising from the contact of inflamed and roughened pericardial surfaces during the heart's movements.

They are generally rough and grating, and hence readily distinguished from the blowing murmurs above described; but they sometimes resemble them very closely. The pericardial rub either consists of two sounds for each beat of the heart; or it may be a triple sound of a shuffling character which is very distinctive. It is not generally localised to one or other valvular orifice, but commences at almost any part of the præcordial area, and may spread over the whole of it. As already stated, a pericardial rub is sometimes rendered louder by pressure of the stethoscope.

## ESTIMATION OF THE CAPACITY OF THE HEART FOR EFFORT

The most important of all examinations of the heart is the estimation of its reserve power, by which is meant finding out its response to exercise. In its simplest form this consists in asking the patient to take some exercise, such as walking quickly, running along the level or upstairs, or doing some simple exercises with dumb-bells, and observing whether he is abnormally breathless or exhausted after it, whether he becomes pale, whether his expression becomes anxious, and whether he develops anginal pain. The history of the patient is also of great importance in this connection, as, for instance, that from a boy he was never able to join in games at school, or that since an attack of rheumatic fever or influenza or diphtheria he has never been able to run; or that he never does run for a train, because he gets too short of breath; or that he never runs upstairs, and so on.

The response to exercise bears little relation to the loudness of the murmurs. This fact was observed when recruits were being examined for the army. Many men were found with loud præcordial murmurs who had always believed themselves healthy, with hearts which responded to exercise perfectly normally. On the other hand, in cases of aortic regurgitation the most severe cases often have the softest murmurs. Estimating the size of the heart and its shape and the extent to which the muscle is hypertrophied will give a better measure of the extent of the lesion which has to be compensated. Palpation and percussion may be employed, but the most certain information can be obtained by the X-rays. The measurements must be taken orthodiagraphically.

It is, of course, impossible to measure directly the rate of flow of blood through the heart in man; and such indirect measurements as have been made are at present of no practical importance in clinical medicine.

Another method that has been adopted is to compare the lung ventilation which results from muscular work with the ventilation while resting, but even this involves too complicated a procedure for ordinary clinical work. On the other hand, it is quite an easy matter to measure the pulse rate under these conditions. Dr. G. H. Hunt, who has standardised this method, making a large number of control observations on healthy people, believes that it is of value in estimating the cardiac efficiency in disease. The number of heart beats during the first two minutes immediately following the cessation of a given amount of work is measured and compared with the pulse rate at rest by taking the ratio. Thus, supposing the pulse rate at rest was 70 and after exercise

160 beats were counted, the ratio would be 2·29. The exercise consists in going up and down steps, each of them 6½ inches high. In exercise A the patient goes up and down a flight of ten steps fifteen times in five minutes; in B the patient does the same exercise twenty times in five minutes. In C the patient goes up and down fourteen steps twenty times in five minutes, and in D fourteen steps twenty-five times in five minutes. The following ratios were obtained for healthy people (1) when trained; (2) when untrained :—

| | A. | B. | C. | D. |
|---|---|---|---|---|
| (1) Trained . . | 2·07 | 2·11 | 2·25 | 2·85 |
| (2) Untrained . . | 2·28 | 2·30 | 2·83 | 3·31 |

From this it will be seen that the pulse after exercise is relatively quicker in the untrained than in the trained individual. Where the cardiac efficiency is impaired in disease this ratio will be still further increased, and it is a simple matter to choose one of these exercises for testing this.

## EXAMINATION OF THE HEART BY MEANS OF X-RAYS

By this means the size of the heart can be accurately determined. The method is of special importance in cases of emphysema, where percussion is usually not trustworthy. Since the rays come out from a point by reflection from the anticathode, they are not parallel, and so the shadow on the screen is larger than the heart actually is. To correct this the heart must be measured "orthodiagraphically." The diaphragm is stopped down, and the tube is moved about so that the edges of the heart are seen and marked in the middle of the narrow field on the screen.

Another method is to place the patient 7 feet in front of the tube and take a radiogram. At this distance the divergence of the rays will not be sufficient to cause any serious error.

Although examination of the heart by X-rays is a big subject, and cannot be dealt with here fully, a few of the more important points may be mentioned.

In Fig. 27, I. represents the shape of the normal heart; AB is the border of the right auricle. Sometimes marked pulsation is noticed at A, due to the right ventricle. BC is the edge of the sternum, but sometimes the superior vena cava and the ascending aorta extend beyond it, and BC then corresponds to the edge of these; DE is the aortic arch; EF is the pulmonary artery as it bends round beneath the aortic arch; F is the left auricular appendix; FG is the border of the left ventricle.

II. represents the so-called "vertical heart," and is a variation of the normal, being present in cases where the thorax is elongated. It is often found in phthisis.

In III., which represents a case of compensated aortic regurgitation, the left ventricle is rounded and the apex displaced downwards and outwards. There is excessive pulsation at the apex. In aortic stenosis the shape is much the same.

In IV., which represents mitral regurgitation, the left ventricle is enlarged to the left, but the apex is not specially rounded or displaced downwards. This corresponds to the left ventricle being dilated, but not much hypertrophied. Excessive pulsation may be noted at A due to hypertrophy of the right ventricle

In V. (mitral stenosis) the shape is characteristic, since the left auricle is dilated and often forms a projection, but the left ventricle is smaller than usual. The left border of the heart is thus more vertical than usual. The right auricle is often dilated.

In VI. (congenital pulmonary stenosis) the right ventricle is hypertrophied and the left ventricle displaced upwards and quite small. The heart has the appearance of a French sabot. There is very often, as shown in the figure, a dilatation of the origin of the pulmonary artery, but the cause of this is obscure.

Orthodiagraphic measurements, which can be quite easily obtained with an ordinary X-ray installation, are also of value in determining whether the heart

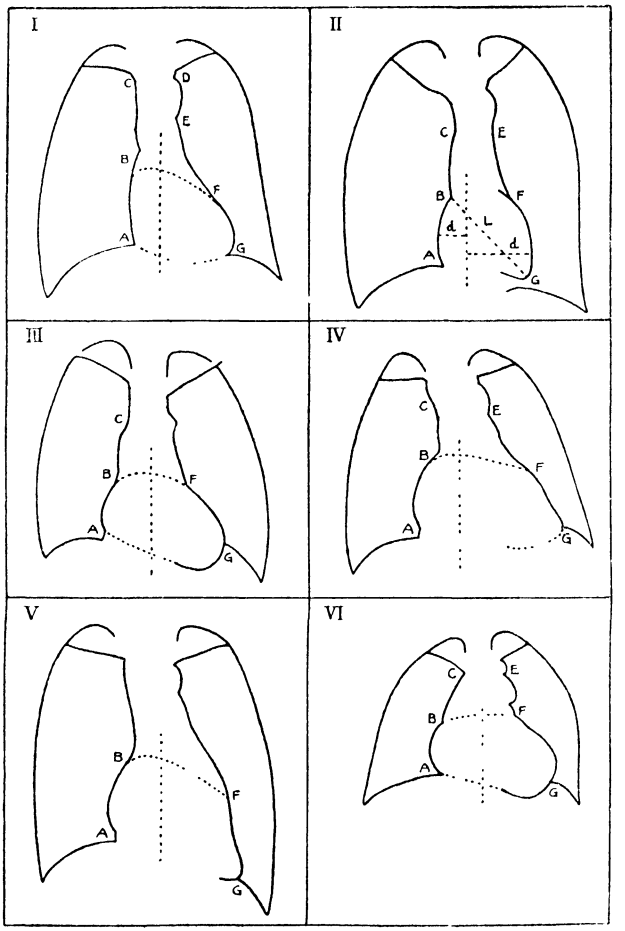



FIG. 27.—I. Orthodiagram of Normal Heart. II. Orthodiagram of Normal Vertical Heart. III. Orthodiagram of Heart in Aortic Regurgitation. IV. Orthodiagram of Heart in Mitral Regurgitation. V. Orthodiagram of Heart in Mitral Stenosis. VI. Orthodiagram of Heart in Congenital Pulmonary Stenosis. (After Vaquez and Bordet.)

is abnormally large. The two measurements, both taken during diastole, are the length (L), *i.e.* the line BG (Fig. 27, II.), and the transverse width D, made up of the two pieces *d* and *d*. Owing to the difficulty of locating B and G in many cases, the latter is usually the more satisfactory measurement. The normal values depend on the weight of the individual and possibly upon his age. In determining the weight it may be necessary, if the nutrition is very abnormal, to deduce the predicted weight from the body-length (see p. 560). The following

tables give the figures for normal individuals standing upright. The values for D may also be used to compare with the width of the heart as determined by percussion (see p. 286).

CLAYTOR AND MERRILL.

| Weight (lbs) | D ($d$ + $d$). (Cms) | | | L. (Cms) | | |
|---|---|---|---|---|---|---|
| | Min. | Mean | Max | Min. | Mean. | Max. |
| 109–117 | 10·7 | 10·9 | 11·3 | 11·8 | 12·6 | 13·5 |
| 118–126 | 11 | 11·8 | 12·5 | 12 | 13·2 | 14 |
| 127–135 | 11 | 11·9 | 13·1 | 12 | 13·4 | 14·5 |
| 136–144 | 11·5 | 12·3 | 13 | 12·5 | 13·5 | 15 |
| 145–162 | 12 | 12·4 | 13·8 | 11 | 14·6 | 15·3 |
| 163–181 | 11 | 12·9 | 13·4 | 14 | 14·7 | 15·8 |

VAQUEZ & BORDET.

| Weight (lbs.) | D ($d$ + $d$). (Cms) | | | Age | L. (Cms) | | |
|---|---|---|---|---|---|---|---|
| | Min. | Mean | Max | | Min. | Mean. | Max. |
| 110–132 | 10·7 | 11 | 13 | 20–30 | 11·2 | 12·3 | 13·5 |
| | 10·5 | 11·6 | 12·7 | 31–40 | 11 | 12 | 13 |
| | 10·3 | 10·6 | 11 | 41–50 | 11·6 | 12 | 12·5 |
| 134–154 | 10·7 | 11·6 | 12·6 | 20–30 | 11·5 | 12·5 | 13·5 |
| | 11 | 11·7 | 12·5 | 31–40 | 12 | 12·7 | 13·5 |
| | 10·5 | 11·7 | 13 | 41–50 | 12·5 | 13·1 | 13·8 |
| 156–198 | 11·3 | 12·2 | 13·2 | 20–30 | 11·6 | 12·7 | 13·8 |
| | 11·3 | 12·2 | 13·2 | 31–40 | 11·5 | 12·6 | 13·8 |
| | 12·3 | 12·9 | 13·5 | 41–50 | 13 | 13·7 | 14·5 |
| | 12·5 | 13·2 | 11 | 51–60 | 13·5 | 14·2 | 15 |

# EXAMINATION OF THE BLOOD VESSELS

## THE RADIAL PULSE

The terms *pulse* and *pulsation* refer to such variations in pressure associated with the onward movement blood, as may be felt in any vessel of the body accessible to the finger, or in any structure or organ, such as the liver, sufficiently vascular to transmit these variations. The pulse serves as an important means of ascertaining the action of the heart and the condition of the circulation. For observations upon the *arterial pulse* the radial artery at the wrist is commonly employed; but a pulse can also be felt in the ulnar artery at the wrist, in the brachial in the arm, in the carotid by the side of the thyroid cartilage, in the facial artery as it turns round the lower jaw, in the temporal artery above the ear, in the femoral artery below Poupart's ligament, in the posterior tibial

behind the inner malleolus, and in the dorsalis pedis near the base of the first metatarsal bone.

It should be remembered that the radial artery does not always lie in its normal situation, but sometimes turns over the radius to the back of the wrist, 1 or 2 inches above the joint; and this may happen on one or both sides. A minute communicating branch may sometimes be felt in its place; but, in any case, the absence of a pulse of the proper size may be very misleading unless this occasional abnormality be borne in mind. More rarely, the radial is abnormally small, and the comes nervi mediani compensates for this by its unusual size.

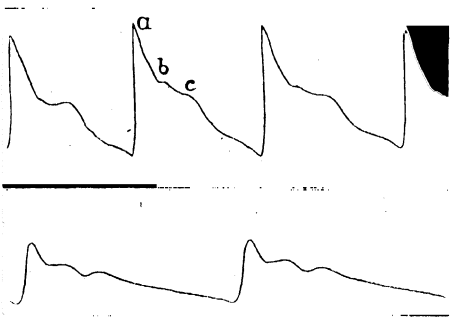



FIG. 28.—A. Normal Soft Pulse. Pressure 2 ounces. B. Hard Pulse in Gout.

The features to be noted in the pulse are the *frequency* of the beats (*pulse rate*), the uniformity of their occurrence (*rhythm* or *regularity*), the amount of the pulsation (sometimes called the *volume of the pulse*, though it must be remembered that the actual increase in diameter of the artery is very small indeed, even though it causes a considerable rise in pressure), the anatomical condition of the *arterial wall*, and the maximum systolic and mean diastolic *arterial pressure*. The difference between these two gives the *pulse pressure*.

Although these features can be recognised by the finger of the trained physician up to a certain point, minuter details can only be noted by means of instruments, of which different forms of sphygmograph and sphygmomanometer are in common use.

**The Sphygmograph.**—In this instrument a light spring presses upon the radial artery, and the movement of the artery wall is communicated to a lever; and this carries a fine point, which traces the magnified movements upon a blackened paper moving horizontally by clockwork. The pressure of the spring upon the artery required to give the true record varies with every case, and the best instruments register in ounces the amount of pressure employed.

Besides the frequency and regularity, which can be at once appreciated, there are other features of the record demanding special study. In the tracing of each beat of the arterial pulse (*see* Fig. 28) there is an upstroke, which is uninterrupted and almost vertical; and a downstroke, which is oblique, and interrupted by one or two elevations with intervening depressions.

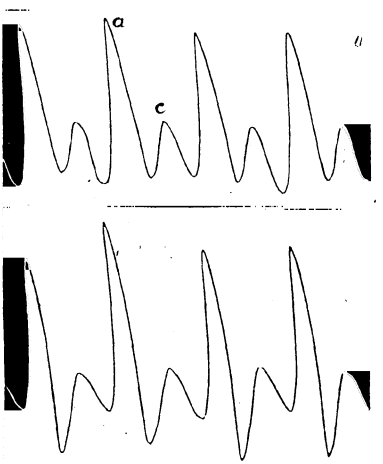



FIG. 29.—A. Dicrotic Pulse in Pyrexia. Temp. 102·2°. B. Hyperdicrotic Pulse in Pyrexia (Enteric Fever). Temp. 103°.

The upstroke represents the increase of pressure due to the contraction of the left ventricle as it drives blood into the aorta. This wave of pressure is rapidly transmitted to the peripheral arteries. The apex of this upstroke has been known as the *percussion wave*. Its height is proportionate to the force of the ventricular contraction, and the quickness or suddenness of the contraction is indicated by the vertical course of the stroke. The height is also greater when the arterial

wall is yielding, less when it is tense and resistent. Compare Figs. 28 A and 29 A, B, with Figs. 28 B and 30 A, B, C.[1]

Of the elevations in the course of the downstroke, the most constant is the *dicrotic wave* (Fig. 28 A, *c*: Fig. 29 A, *c*: Fig. 30 A, *c*). It is shown by the sphygmograph to be present in the majority of pulses. When very marked, as in Fig. 29, the wave can be felt by the finger, so that the pulse appears re-duplicated, the so-called "dicrotic" pulse. It is due to a reflected wave from the closed aortic valves and from the walls of the aorta. It is immediately preceded by a depression, the *dicrotic notch*, which corresponds to the end of the ventricular systole, and marks the closure of the aortic valves. The interval between the commencement of the percussion wave and the bottom of the dicrotic notch is thus the *systolic period*. When the dicrotic notch reaches the base line (Fig. 29 A) the pulse is called *fully dicrotic*; it sometimes falls below the base line, and is then called *hyperdicrotic* (Fig. 29 B). In this case the percussion wave of the next beat appears to come before the dicrotic wave has completely passed, and, indeed, the occurrence may be due to the increased rapidity of the beats. Dicrotism is best marked in a soft pulse, *i.e.* low-tension pulse, when the arterial wall is yielding; it is a common result of vasomotor paralysis, as seen in highly febrile conditions (Fig. 29), and can be at once produced by the administration of amyl nitrite. It is diminished or abolished by conditions leading to a hard pulse, *i.e.* a high-tension pulse, such as Bright's disease, and by aortic regurgitation, in which case the reflection of a wave takes place imperfectly.

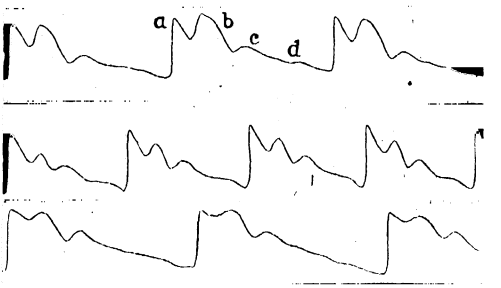



FIG. 30.—A. Acute Bright's Disease. Pressure 4 ounces. B. Acute Bright's Disease, five weeks' duration. Pressure 7 ounces. C. Chronic Bright's Disease. Pressure 6 ounces.

Between the percussion wave and the dicrotic wave—that is, preceding the aortic notch, and therefore corresponding to the period of systole of the ventricle—there is often a wave which has been attributed to the outward flow of the current of blood following the percussion wave. It is called the *tidal* or *predicrotic wave* (Fig. 28 A, *b*; Fig. 30 A, *b*, B, C). It is best seen in hard pulses (Fig. 30)—that is, in conditions of high arterial tension, when it may be supposed that the undulations of the blood would be unusually well transmitted. Occasionally the tidal wave is so well marked that the pulse appears reduplicated, the so-called *pulsus bisferiens*, to be distinguished from the dicrotic pulse, where the reduplication is due to the exaggerated dicrotic wave. On the other hand, in very soft pulses the tidal wave is lost in the percussion wave (Fig. 29 A, B). A pulse in which the tidal wave rises higher than the percussion wave has been called *anacrotic*, because the percussion wave forms an elevation in the ascending limb between the base and the highest point.

One or two slight undulations are occasionally seen after the dicrotic wave (Fig. 30 A, *d*). They occur in tracings of pulses of high tension only.

**Pulse Rate and Rhythm.**—The frequency of the pulse and its regularity are so entirely dependent upon the action of the heart, and so closely related to the rate or regularity of the left ventricle, that it is better to consider their variations when dealing with the abnormalities of cardiac action (*see* p. 303). It is sufficient to state here that normally the heart beats, that is, the ventricle

[1] The tracings were taken with a Marey's sphygmograph; a long and quick upstroke is curved backward, because the needle is at the end of a long lever, which works on a fulcrum, with an axis transverse to the line of movement of the paper.

contracts, about seventy times in the minute, with variations between fifty and eighty; that the beats recur at regular intervals; that the radial pulse wave is felt an appreciable time later than the impulse of the heart; that in certain conditions the beats of the pulse may be less numerous than the contractions of the ventricle, if from any cause some of the contractions of the ventricle are so feeble that no blood is expelled through the aortic valves.

**The Amount of the Pulsation (Volume of Pulse).**—This is determined partly by the actual size of the artery, and partly by the quantity of blood sent into the artery at each beat of the heart, and the rapidity with which the heart delivers the blood into the aorta. If much blood is sent in, the pulse is full or large; if little blood, the pulse is small.

In an irregular pulse the beats generally vary in volume as well as in the time of occurrence, for a long diastolic period gives time for more blood to accumulate in the ventricle, so that, if the heart is beating satisfactorily, more blood will be expelled through the aortic valve at the next beat (*see* Figs. 38 and 39).

In the *pulsus paradoxus* there is a considerable diminution or complete absence of the pulse during inspiration. Though rare, it may occur under several conditions, such as mediastino-pericarditis, pericarditis, mediastinal tumour, great cardiac weakness, pleural effusion, or obstruction of the air passages.

**The Arterial Wall.**—If the pulse be stopped by the pressure of the finger, and emptied of blood, it should in health be scarcely perceptible as a separate structure; if it is thickened or rigid from arterio-sclerosis, it is easily felt; and if highly calcareous, the irregularities of the deposit can be felt on passing the finger up and down the length of the vessel.

**Hardness of the Pulse: Arterial Tension.**—If the finger be pressed upon the artery with increasing force, the flow of blood is at length stopped. It is best to compress the artery with the index finger of one hand, placing the index finger of the other hand below, so as to feel when the pulse is stopped. The other fingers of this hand may be pressed on the artery farther on, so as to prevent a pulse from coming up from below through arterial anastomoses. Those in which slight pressure is sufficient are called *soft* or *compressible* pulses; those in which much pressure is required are called *hard* or *incompressible* pulses. If when the pulse has been stopped by compression the finger be slowly lifted, the blood will be felt to pass under the finger with much greater force in the case of the hard than in the case of the soft pulse. A more certain estimate of the hardness of the pulse can be derived from the use of the sphygmograph above described, and of different forms of sphygmomanometer. An estimate of the mean diastolic pressure may be obtained by pressing on the artery with the finger until the maximum pulsation is felt.

**The Sphygmomanometer.**—In this instrument, of which there are several varieties (Riva-Rocci, Erlanger, Gibson, Hill and Barnard), the arterial pressure is measured by its displacing effect upon a column of mercury. In most instruments the upper arm is encircled by a broad double band or bag of india-rubber, covered with some inextensible material, into which air can be forced by an india-rubber ball and valve through a connecting rubber tube; another tube proceeding from this is connected with the manometer, and the pressure in the india-rubber bag is measured in millimetres of mercury. When the instrument is adjusted, air is pumped into the armlet and manometer until the pressure is more than sufficient to stop the pulse at the wrist. The air is then gradually allowed to escape, until the pressure is reduced to a point at which the pulse is just felt. The figure on the scale at which the mercury then stands represents the *systolic pressure*.

The mean diastolic pressure is best measured by auscultation. The stethoscope or phonendoscope is applied over the brachial artery at the bend of the elbow. If the pulse be first completely obliterated by pressure, and then the pressure gradually reduced, four phases of sound are heard in order. The first

phase is a faint throb, which is first observed at a pressure a few millimetres above the maximum systolic pressure as indicated by palpation. This changes into a loud, short murmur—the second phase. At a still lower pressure the murmur changes into a loud throb—the third phase. This suddenly changes into a soft throb—the fourth phase. It is the pressure corresponding to the change from third to fourth phase that is the mean diastolic pressure.

Kilgore has carried out an extensive series of observations on men between sixteen and thirty-six years. The average systolic pressure was 120 mm., but there was a wide range on each side of this, and in 2 per cent. of cases the pressure was found to be under 95 mm. and above 140 mm. Excitement, due to the taking of the measurements, may produce quite high results, and this, no doubt, accounts for the higher readings obtained, especially as these were not substantiated on a second examination. Measurements of diastolic pressure by the auscultatory method gave a mean value of 80 mm., but there were many readings at 70 and 90 mm., and 2 per cent. of the observations were below 60 and above 100 (see also p. 369). In the new-born infant the systolic pressure has been found to be 43 mm. on the average, rising to 78 mm. after ten days.

A hard pulse, or pulse of high pressure, is not necessarily very small in volume or very large. The artery feels like a cord, even though there are no changes in the artery wall, but, of course, the two conditions, rigidity of arterial wall and hardness of pulse, may co-exist. A pulse is hard in proportion to (1) the quantity of blood thrown into the arterial system, (2) the difficulty of egress through the arterioles, capillaries and veins (often spoken of as capillary resistance), and (3) the degree of contraction of the arterial coats upon their contents. It is soft under the opposite conditions. Thus, hardness is favoured by a strongly acting heart, a normal amount of blood, and contraction of the peripheral arterioles—as, for instance, by cold, which stimulates the muscular coat of the arteries (vasomotor stimulation). Softness of pulse is favoured by a feeble heart, by valvular imperfections interfering with the supply of the blood to the arterial system, by a free flow through the capillary area, and by dilatation of the arteries and arterioles as a result of vasomotor paralysis. The hard pulse is also said to be indicative of *high arterial tension;* the soft pulse of *low arterial tension.* (*See* Abnormal Arterial Tension.)

## AUSCULTATION OF THE ARTERIES

If the carotid or subclavian artery is auscultated without pressure by the stethoscope two sounds are usually heard—a systolic sound due to expansion of the vessel, and a diastolic sound which is the conducted aortic second sound. The first of these is sometimes absent. Over the abdominal aorta and the femoral artery a systolic sound is heard like the above. In the other arteries, as a rule, nothing is heard unless pressure is exerted by the stethoscope.

When a saccular dilatation of an artery, or aneurysm, occurs in any part of the body or limbs, a systolic murmur is frequently heard, and it is attributed to the fluid vein and eddies produced by the passage of blood from the orifice of the artery into the wider space, the sac of the aneurysm. Since aneurysms often form in connection with the base and wall of the aorta, which are in close proximity to the heart, they may cause murmurs in the præcordial area, with difficulty distinguished from those produced at the cardiac orifices.

## THE VENOUS PULSE

Some pulsation in the larger veins of the body appears to be a normal phenomenon; and an undulating or distinctly pulsatile movement can be seen in the jugular veins, both external and internal, of some persons with quite healthy circulations; but usually it is absent or inconspicuous. The relation of the

movements to those of the radial pulse is often difficult to make out on simple inspection; the external jugular may be seen just above the clavicle, and its movement differs from arterial movements in that the expansion is slow, while the collapse is more sudden, and corresponds nearly to the rise of the radial.

The internal jugular produces a large undulatory movement with slow rise and quicker fall over the side of the neck between the angle of the jaw and the sterno-mastoid muscle, and it must not be confounded with carotid pulsation.

More accurate information can be obtained by tracings from the jugular pulse by means of a tambour applied to it, the movements of which are transmitted to a needle writing either on a drum, or on the smoked paper of the sphygmograph, parallel with a radial tracing, as in the *polygraph* of Mackenzie. The best way of getting the jugular pulse is to place the shallow cup of the apparatus over the jugular bulb, *i.e.* a little above and 1 inch external to the sternal end of the clavicle, preferably to the right side. The sterno-mastoid fibres of the same side are relaxed. The tracing usually obtained shows three waves (positive waves), with, of course, intervening depressions (negative waves). The first positive wave (Fig. 31, *a*) occurs just before the systolic period of the radial tracing (*see* p. 298), and is admitted by all to be due to contraction of the right auricle. The

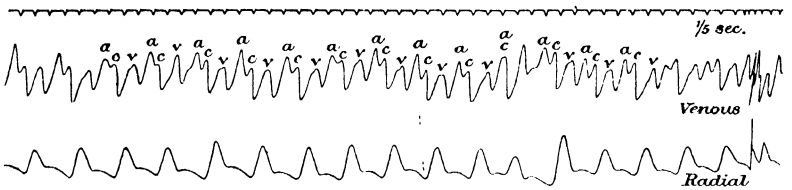



FIG. 31.—A Polygraphic Curve showing the Three Waves of the Physiological Venous Pulse in the upper tracing, which was taken from the veins of the neck. The lower tracing is from the radial artery. With each systole of the ventricle are two waves, *c* and *v*. Preceding *c* in each cycle, and pre-systolic in time, a wave, *a*, is seen, which is the result of auricular systole. (*T. Lewis.*)

second positive wave (*c*) has been shown by animal experiment to be due to the projection of the auriculo-ventricular valve (tricuspid) into the right auricle during the contraction of the right ventricle; on the other hand, in many human tracings it is doubtless chiefly due to the pulse in the carotid artery. In either case it is recognised as representing a ventricular contraction; and the interval between *a* and *c* is taken to be the measure of the time of conduction of the wave of muscular contraction from auricle to ventricle. In normal individuals, its duration is about one-fifth of a second. The third wave (*v*) is probably due to the shock, caused by the sudden stretching of the pulmonary valves after closure, being conducted backwards through ventricle, auricle and veins.

An important modification of the venous pulse is that in which the *a* wave is absent, and only the *c* and *v* waves occur (*see* Fig. 44). From the absence of the *a* wave, it is inferred that the auricle is not contracting normally, and hence that the waves recorded are due only to the ventricle. The normal jugular tracing with the three waves is thus held to represent an *auricular form of venous pulse;* while the tracing last described represents a *ventricular form of venous pulse,* or the pulse of auricular fibrillation.

Pulsation of a different kind is sometimes seen in the *peripheral veins,* especially those of the backs of the hands and feet, which is due to the transmission of the arterial wave through the capillaries to the veins. It results from great relaxation of the vascular walls with a powerful or excited action of the heart; thus it may occur in febrile conditions, in the heat of summer, or after a full meal.

### AUSCULTATION OF THE VEINS

If, in very anæmic persons, and in healthy children, the stethoscope be placed over the lower part of the jugular vein, at the point of separation of the sternal from the clavicular attachments of the sterno-mastoid, a continuous humming or rushing noise will be heard, which has been called the venous hum, or *bruit de diable*, from a French toy, called " diable," which makes a similar noise. This murmur is heard best in the erect posture, with the face turned away from the side which is being examined.

## ABNORMALITIES OF CARDIAC ACTION

The normal mechanism of the heart beat has already been considered. Two different methods are commonly employed in medicine to find out what abnormalities, if any, are present in this mechanism. Their chief value is that they indicate auricular as well as ventricular contractions.

In the first, by means of the polygraph, the jugular pulse and an arterial pulse, such as the radial, are taken simultaneously and compared. The tracings indicate the presence or absence of auricular contractions, and their relation to ventricular contractions, and the length of time intervening between the two; and if this is greater than one-fifth of a second the presence of partial heart block is suggested.

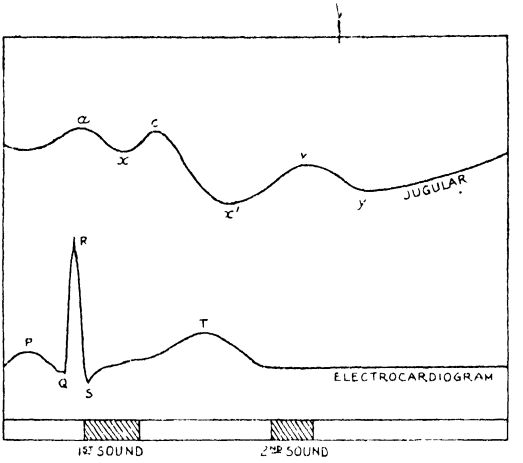



FIG. 32.—Diagram of Jugular and Electro-cardiographic tracing, and Heart Sounds corresponding with One Heart Beat.

The second method is by means of the electro-cardiograph, a sensitive galvanometer which records the electric currents generated by the heart at each beat. The instrument consists of a fine quartz fibre silvered over and suspended between the poles of a powerful electro-magnet. Each end of the fibre is connected up with the limbs of the patient in three ways, commonly called leads. In Lead I., or the transverse, the two hands are connected with the instrument, and represent a contact with the two sides of the base of the heart, that is, the auricles and the base of the ventricles. In Lead II., or the axial, the right hand and the left foot are used, and represent a connection with the top of the auricles and the apex. In Lead III., or the left lateral, the left hand and the left foot are used, and represent the left auricle and ventricle. The electric currents generated by the heart are passed to the galvanometer by these leads. The quartz fibre moves, and its excursion is magnified and projected by means of a telescope on to a photographic plate or paper. The result is an electro-cardiogram. The tracing corresponding to one heart beat is shown diagrammatically in Fig. 32. The jugular tracing is also shown, and the approximate position of the heart sounds. In the electro-cardiogram there is a wave corresponding to, and very slightly preceding, the auricular contraction usually designated by the letter P. This is followed by a depression, Q, a sharp elevation, R, a sudden depression, S, and a gradual elevation, T. The complicated figure, QRST, corresponds to and begins just in front of the ventricular contraction.

As in the case of the jugular pulse, an electro-cardiogram indicates the relations of auricular contractions to ventricular contractions, and the length of time intervening between them—the P.-R. interval; but in addition certain inferences may be drawn from the shape of the ventricular complex, which becomes modified, increasing the seriousness of the prognosis, if certain branches of the auriculo-ventricular bundle are blocked (branch-bundle block), or if a ventricular contraction arises from an abnormal position in the heart, instead of coming down the bundle in the usual way from the auriculo-ventricular node. Again, a persistent inversion of the T wave in Leads I. and II., so as to produce a depression instead of a wave, suggests myocardial degeneration (**2**). Naturally in both methods an accurate time-marker is used so as to show the correct time relations of the different parts.

The advantages of the polygraph method are the portability and simplicity of the apparatus; on the other hand, it is sometimes very difficult, if not impossible, to get a tracing at all, and although the results are usually sufficient for all practical purposes, they do not give the same detail as can be obtained by the electrical method. The disadvantage of the electro-cardiograph is the complicated nature of the apparatus; on the other hand, when working satisfactorily, results can always be obtained very easily with no trouble to the patient, who may be lying in bed in a different building if necessary.

It must not be imagined that these elaborate methods are now essential for the diagnosis of common irregularities of the heart. As the result of research by their means it is now possible to diagnose these conditions in the majority of cases simply by auscultation, palpation and with the help of a sphygmogram.

## SINUS IRREGULARITY

This condition is common in children. During inspiration the heart beats become more frequent, and during expiration and at the end of the expiration the rate is diminished again. Electro-cardiographic examination has shown that the beats are always perfectly normal, and that the alteration in the rate of the heart is due to an alteration in the rate at which the sino-auricular node sends out its impulses, this rate being influenced by vagal action. The tone of the vagus is diminished during inspiration. This respiratory irregularity has been called the "youthful irregularity" by Mackenzie, and is perfectly normal. Occasionally the same phenomenon is observed with adults during quiet respiration; it is practically always observed when the respiration is deep. Occasionally there is a similar alteration in rate, with no relation to the breathing at all. Sinus arrhythmia is abolished when the rate of the heart is increased, as in exercise. Its chief importance is that it may be confused with other types of irregularity. When recognised, it should be treated as of no importance, and no remedial measures should be tried.

## HEART BLOCK AND ADAMS-STOKES SYNDROME

**Heart Block** is due to an impairment in the conducting powers of the auriculo-ventricular node, the auriculo-ventricular bundle, or any of its branches. It occurs in acute infective diseases, particularly rheumatism, and also after treatment with digitalis. In these cases it is temporary. A permanent state of heart block occurs in hearts permanently damaged by rheumatism or syphilis, and in elderly people with myocardial degeneration.

The first thing that occurs is a slight increase in the P.-R. interval (normally 0·12—0·18 second). The *a.-c.* interval is greater than 0·2 second. This can only occasionally be recognised clinically. However, it can be recognised under certain circumstances. Under normal conditions the auricular contraction cannot be heard with a stethoscope, probably because it takes place so close to the contraction of the ventricle that the noise of the latter completely swamps it.

When, however, the space between the two becomes greater, the auricle can be heard separately. Under these conditions a double first sound may be heard when listening to the apex, or a double second sound in the same position, when the interval between the auricular and ventricular beats is greater still, *i.e.* when the auricle beats at the beginning of diastole, very soon after the second sound. Both these cases are types of canter-rhythm. Again, in young children presystolic or early diastolic murmurs may occasionally indicate heart block and not mitral stenosis. Heart block is probably present much more often than is commonly supposed at the bedside.

FIG. 33. Electrocardiogram of Partial Heart Block in a case of Subacute Cystitis. There is an interval of 0·32 sec. between the auricular wave P and the ventricular complex RST. Such a condition might lead to dropped beats (Lewis).

In a more advanced stage of heart block the P.-R. interval becomes still more prolonged, and occasionally the impulse fails to get through to the ventricle, so that ventricular beats are missed altogether. This can be recognised by auscultating the heart, when suddenly in the midst of a natural rhythm one heart beat will be completely missed altogether; naturally the pulse will show the same intermittent character. In more advanced cases we may get a condition known as 2—1 or 3—1 heart block, where only every second or third beat of the auricle is effective in exciting a ventricular contraction.

So far we have only considered heart block when the interval between the auricular and ventricular contractions is increased, or where certain auricular contractions do not excite the corresponding ventricular contractions. All these are cases of partial heart block. In complete heart block none of the auricular contractions get through to the ventricle at all. Fortunately under these conditions the ventricle starts beating on its own account, but its own proper rhythm is very slow, usually below forty a minute. An electro-cardiogram shows a perfectly regular series of auricular contractions and another regular series of ventricular contractions, but the two series are completely dissociated from one another. A regular pulse of less than forty a minute is, then, suggestive of complete heart block, though it should be pointed out that cases with

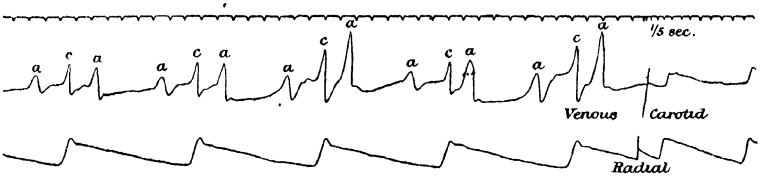



FIG. 34.—A Polygraphic Curve from a Case of 2—1 Heart Block. The upper tracing is from the veins of the neck, the lower tracing from the radial artery. Each radial beat is accompanied by a systolic elevation, *c*. At regular intervals in the curve additional waves due to auricular systole are present, marked *a*. The auricle is contracting twice as frequently as the ventricle. (*After Lewis.*)

a pulse rate of thirty-five to forty a minute are met with, showing a normal rhythm. But there is another sign, which was first described by Galabin, in 1875, and was published in the Guy's Hospital Reports of that year. It was the first suggestion that in man there could be complete dissociation of auricular and ventricular rhythms. In his case the pulse rate was between twenty and thirty per minute, and he

actually heard the auricular contractions by auscultation. "We have here," he says, "a heart the auricle of which sometimes contracted twice in the interval between two ventricular pulsations, and sometimes singly in the midst of a long pause, instead of just before the systole of the ventricle." There is another sign also depending on the fact that the auricular beat can be heard, and that it keeps on occurring at different points in the cycle. Thus, at one time there may be an apparently reduplicated second sound, at another time a reduplicated first sound, and at another time the first sound is greatly increased in intensity, *i.e.* when the auricle and ventricle contract simultaneously. In the last case it is possible to see simultaneously a sudden large wave in the veins of the neck. This is due to the fact that the auricular contractions cannot drive the blood forward into the ventricle, as this is contracting. Hence the blood from the auricle tends to be driven backwards.

**Adams-Stokes Syndrome.**—It was observed by R. Adams in 1827, and by W. Stokes in 1846, that patients with abnormally slow pulse might be subject

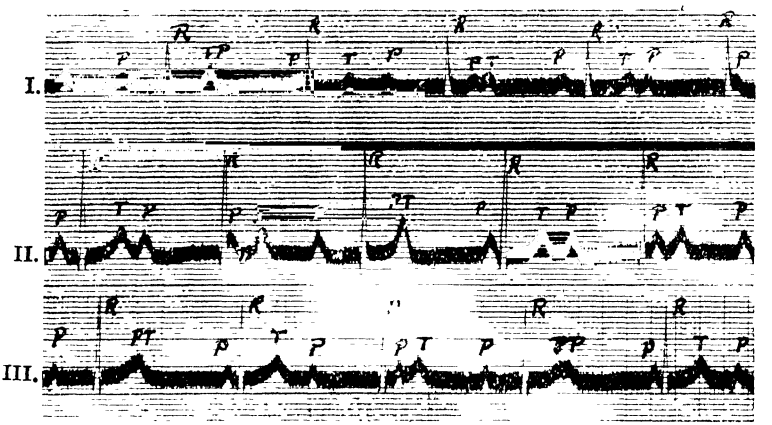



FIG. 35.—An Electro-cardiogram of Complete Heart Block. The rhythms of auricle and ventricle are dissociated, and the ventricle is no longer responding to the auricle. The ventricular systole produces the displacements R and T; the auricular systoles produce the movements P, which are distributed uniformly through the tracing, and have no constant relations to the ventricular movements; but they are nearly twice as frequent. (*After Russell Wells.*)

to attacks of syncope, unconsciousness, or convulsions. Stokes, indeed, observed that the veins in the neck were beating faster than the radial pulse; and in two of these earlier cases there was fatty degeneration of the heart, in which the A.V. bundle, then unknown, must have been involved. Spens had seen a similar case in 1792 (Lea).

The condition is due to cerebral anæmia, produced by sudden failure of the circulation from heart block. Although heart block is common, this syndrome is uncommon. It occurs in elderly people suffering from the more severe grades of heart block, or from complete heart block. The ventricle suddenly beats very slowly, or stops altogether for a time. The symptoms depend on the length of time that the circulation is inadequate. An elderly man was observed in whom there was complete cessation of the heart for forty seconds every two minutes, so that there was an opportunity of repeatedly observing the exact progression of the symptoms (3). Immediately on cessation of the heart there was pallor with slight lividity of the face; after two or three seconds he stopped talking and fell back with a groan. Consciousness was lost in from five to seven seconds. Contractions of various muscles and involuntary movements of the limbs were

then noticed. The breathing gradually became deeper and more convulsive, the accessory muscles of respiration being brought into play. In about twenty seconds he was breathing at a rate of forty-four a minute, and very deeply. It resembled the panting that is only noticed after the severest forms of muscular exertion. He was livid and pale. The corneal and light reflexes were abolished. When the heart began again, the face flushed bright red after the first two or three beats. There was profuse sweating, and lachrymal secretion, and the conjunctivæ were congested. He then rapidly regained his normal appearance.

**Prognosis.**—The significance of heart block depends on various conditions. In temporary cases associated with infectious conditions, such as rheumatism, it means that the myocardium is definitely affected, though recovery may occur. Hence rest and care are necessary. In permanent cases the heart block will not be usually fatal itself, but it is important in so far as it gives an indication as to the healthiness of the myocardium as a whole. If there is a lesion in the auriculo-ventricular bundle, there may be lesions spread throughout the muscle, and evidence of myocardial degeneration must be looked for, and this may be the cause of heart failure. Where there are fits there is an added seriousness to the prognosis, as these fits may in themselves be fatal.

**Treatment.**—In cases of heart block, treatment should be directed to the condition of the heart generally; there is no reason to withhold digitalis for fear of its increasing the grade of heart block, if it is considered that digitalis is otherwise indicated, for the relief of œdema, etc. Some people with heart block have led active lives, but in most cases exertion should be avoided. When there are fits, adrenalin in doses of 5, 10 or 15 minims has been successful, by increasing the ventricular rate and increasing the conduction (**4**). Atropine, which is usually given, is rarely successful. In syphilitic cases it is most important to use mercury and iodides.

**Sino-auricular Block.**—This is a rare condition. It sometimes arises during the course of an acute specific fever; in other cases there are signs of myocardial degeneration. The pulse is slow and irregular, because, owing to the auricle failing to respond to excitation from the sino-auricular node, a heart beat is completely missed out. This may happen several times in succession, the heart failing to beat for several seconds at a time. It can be differentiated from heart block by polygraphic or electro-cardiographic tracings, since the auricle, as well as the ventricle, is infrequent. In a case recently observed, the condition cleared up during exercise, but reappeared as the pulse slowed down (**5**).

## PREMATURE BEATS; EXTRA SYSTOLES

This type is one of the most common of all cardiac irregularities. It is commoner in men than women. Probably most people who reach middle life or advanced years have extra systoles at one time or another. In ordinary practice they are commonest among those who exhibit evidence of cardiac disease, but they often appear in other people as well. They are always abolished when the heart quickens, as occurs in exercise, and they often reappear as the heart slows down again.

The formation of premature beats is due to over-excitability of certain parts of the heart, which initiate extra heart beats before the proper time; after this premature beat has occurred, there is a compensatory pause until the heart takes up its normal rhythm again. The patient experiences corresponding sensations. The premature beat itself may or may not be felt in the chest. The long pause is noticed and often gives rise to a sense of uneasiness; this is ended by a sudden bump or shock in the chest when the heart renews its normal rhythm. These sensations are one of the cardiac sensations that are known as palpitations.

Research has shown that these beats arise either in the auricle or in the ventricle due to over-excitability of these parts. They may arise in a part of the normal

path of conduction of the heart beat, when they are indistinguishable by the electro-cardiograph from the normal beat except that they occur earlier than they should, and so the rhythm of the heart is disturbed. These beats are known as "homogenetic," or "homotopic." On the other hand, they may arise from a part of the heart outside the normal path of conduction, when they are called "heterogenetic," or "heterotopic." In this case the shape of the curve on the

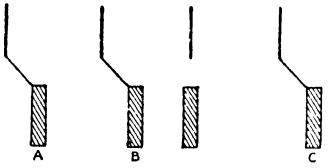



FIG. 36.—Diagram showing Ventricular Extra Systole. The auricular rate is undisturbed by the occurrence, so that the original rhythm of the heart is resumed afterwards. This is shown by the interval BC being exactly twice AB. (*After Lewis.*)

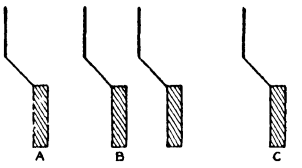



FIG. 37.—Diagram showing Auricular Extra Systole. Since the auricle takes part in the irregularity its rhythm is disturbed. The next beat comes in rather sooner than in Fig. 36. This is shown by the interval BC being rather less than twice AB. (*After Lewis.*)

electro-cardiogram is abnormal. It is, of course, impossible to recognise these distinctions by auscultation. A radial tracing may help to determine if they are auricular or ventricular in origin, because in the latter case the interval between the two beats on each side of the extra systole will be equal to twice the normal interval, while in the former case it will be less (*see* Figs. 36 and 37). In certain cases these beats occur frequently. If we get a series consisting of alternate normal and premature beats the condition is known as "pulsus bigeminus," to be distinguished from "pulsus alternans." A normal beat may be regularly followed by two extra systoles, when a triple rhythm will be observed (pulsus trigeminus).

Premature beats may produce two effects on the pulse. The corresponding pulse wave may be small and followed by a compensatory pause, or there may be a complete intermission of the pulse for a beat (Figs. 38 and 39). Premature

FIG. 38. Tracing of Radial Pulse showing Premature Beat at *x*, probably ventricular.

FIG. 39.—Tracing from Radial Pulse with Intermission, the Premature Beat not reaching the wrist. It is probably ventricular.

beats can be readily recognised by the stethoscope. Variations occur according as the extra beat of the heart is a strong one or a weak one, *i.e.* sufficient to raise the aortic valves and expel some blood or not. In the former case a group of four sounds coming close together in the middle of a series of normal heart sounds will be heard. The four sounds may be represented by the expressions "lub," "dup," "tum," "ti." The "lub-dup" represents the two sounds of the normal beat; the third sound of the group, "tum," represents the sound of the extra ventricular contraction, and the "ti" the closing of the aortic valves. When

the extra systole is not strong enough to raise the aortic valves the fourth sound of the group, "ti," is absent. A group of three sounds will then be heard close together represented by the expressions "lub," "dup," "tum."

In the majority of cases extra systoles are of no prognostic significance. However, the more serious condition of paroxysmal tachycardia is to some extent related, and the possibility of this condition supervening should be borne in mind. However, there is not much evidence that this often occurs. When extra systoles occur in people with heart disease, the nature of the lesions should be taken into account in estimating the prognosis, and the presence of the extra systoles need not make the prognosis worse. When they occur in people who are quite healthy, the patient should be reassured as to their unimportance.

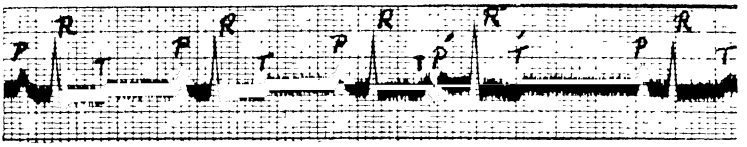



FIG. 40.—Electro-cardiogram of Heterotopic Auricular Extra Systole. The waves P', R', T', represent the extra systole, which follows quickly on the preceding beat, and is itself followed by a longer interval than occurs between the normal beats at the beginning of the tracing. The distance between the two R's on each side of R' is less than double the normal interval between the beats. P' is different from P in shape. This shows that the extra systole is heterotopic. (*After Russell Wells.*)

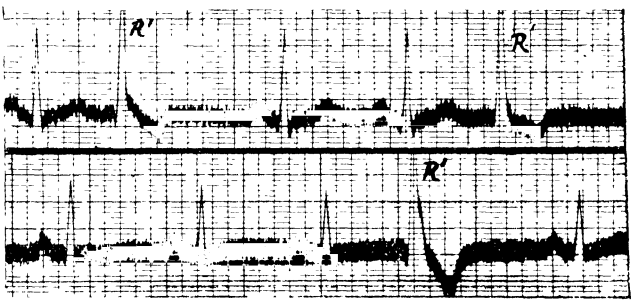



FIG. 41.—Electro-cardiogram of Heterotopic Ventricular Extra Systole. The abnormal stimulus is supposed to arise near the base or in the right ventricle. The ventricular spike R' has an abnormal form, and is not preceded by an auricular wave. The distance between the normal waves on each side of R' is equal to two normal intervals. (*After Russell Wells.*)

It has been commonly taught that in no case should any treatment be specially instituted, and the patients should not be advised to refrain from outdoor games, or, in fact, any exercise that they would otherwise be able to perform. This is going rather too far when the extra systoles are associated with valvular disease. A patient with mild aortic and mitral disease of rheumatic origin, known to the writer, was indulging in too much hard muscular work and frequently experienced extra systoles, although the exercise never made him short of breath. Eventually angina pectoris supervened. On returning to a more restful condition of living he noticed that the extra systoles practically disappeared. Their presence in the first place might have been taken to indicate that the exercise was too much. On the other hand, the writer has met with healthy students who have developed extra systoles when for some reason they have stopped their regular exercise. On beginning exercise again the extra systoles have disappeared.

## FREQUENT ACTION OF THE HEART

(*Tachycardia*)

The heart beats, and with it the pulse, more frequently than usual in various circumstances. Exertion will quicken the heart to more than double the normal frequency, but with the cessation of effort the pulse returns in two or three minutes to its normal rate. Under nervous influences the rate is also increased: the quickened action of the heart from emotional causes is familiar enough; and a quickened action arises also from paralysis of the vagus, as is seen sometimes in multiple neuritis In rare cases individuals have the power voluntarily to increase or diminish the rate of the heart. A common type of quickening of the heart beat in which an emotional factor often plays a part, is one of the forms of "palpitation" that are so often complained of. A very common cause of increased pulse frequency in disease is *febrile reaction:* and the change is, in part, attributable to the toxins which cause the fever, though it must be remembered that direct heat alone, as experienced in a hot bath, or in heated air, will quicken the heart. Tachycardia is an important feature in Graves' disease, due, it is supposed, to an excess or modification of the internal secretion of the thyroid gland: and it results also from atropine and some other poisons. In all these instances electro-cardiographic examination has shown that the increased frequency is due to excitation of the normal pacemaker of the heart The record of the heart beat itself is normal.

Another cause is structural disease of the heart, whether myocardial or valvular, inefficiency in each single beat requiring an increased number of the beats in a given time to produce an adequate circulation. Apart from any of these causes, there occur from time to time cases of paroxysmal increase in the frequency of the heart's beat, which will now be considered.

## SIMPLE PAROXYSMAL TACHYCARDIA

By simple paroxysmal tachycardia is meant a condition where the normal mechanism of the heart suddenly gives place to a series of rapid and regular beats, varying between 110 and 200 a minute. These beats are due to a new centre, arising in the heart, which initiates beats on its own account, the function of the normal pacemaker being temporarily abolished. The beats are thus heterotopic in origin, and they may be regarded as a series of extra systoles. The new centre

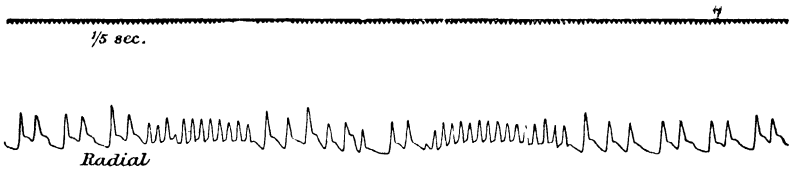



FIG. 42.—An Arterial Tracing from a Case of Paroxysmal Tachycardia. Two short paroxysms are shown, each lasting approximately five seconds. Between the paroxysms the pulse is irregular. (*After Lewis.*)

may be (1) in the auricle, *i.e.* near the sino-auricular node, in the general musculature of the auricle, in the auricular-ventricular node, or in the main bundle beyond the node, or (2) in the right or left ventricle, where they probably arise in the Purkinje network. These varieties can be differentiated by the electrocardiograph. The auricular type of simple paroxysmal tachycardia is the commoner of the two. When originating in the auricle, the beats spread normally to the ventricle, but when they start in the ventricle, they spread back to the auricle in a direction the reverse of the normal.

The condition occurs at any age. It is particularly associated with myocardial degeneration, and very often, but not always, post-mortem examination discloses a fibroid heart with coronary sclerosis.

The characteristic of the condition is its sudden onset. The attack may last any time, from a few seconds to several weeks. The heart is quick and regular, and is uninfluenced by posture. This helps to differentiate the condition from other types of tachycardia. The heart sounds are tic-tac in character, and any murmurs that may have been present disappear. At the height of the paroxysm the pulse felt at the wrist is often rather irregular. Its ending is as abrupt as its beginning, the pulse at first becoming abnormally slow and possibly showing extra systoles before regaining its normal rhythm.

**Symptoms.**—No symptoms may be caused if the attacks are of short duration, and particularly if the patient becomes accustomed to them. There is usually distress if they last some time. Fluttering in the chest is complained of, and beating in the neck. There is lassitude, exhaustion, coldness of the extremities and sweating. Symptoms of indigestion come on—flatulence, salivation, nausea and vomiting. There may be anginal symptoms—a feeling of tightness in the chest and substernal pain. Later there may be signs of cardiac failure, due to embarrassment of the heart owing to its rapid action, with dilatation of the heart and engorgement of the veins at the root of the neck, enlarged and tender liver, generalised œdema, etc. As soon as the attack stops all these symptoms disappear at once, though there may be a varying degree of exhaustion remaining if the attack has been severe.

**Diagnosis.**—This depends on a careful examination of the patient, with an inquiry as to whether other attacks of a similar nature have previously occurred. These cases have sometimes been diagnosed as perforated gastric ulcer, and an operation performed. The diagnosis of "acute dilatation of the heart" has also been made.

**Prognosis.**—The main points in prognosis are (1) the extent to which the heart muscle is permanently damaged, if at all, because attacks may be solely due to nervous action, the heart muscle being healthy; (2) the severity of the attacks, and especially the signs of cardiac failure. It is not uncommon for young people to have one or two isolated attacks from some toxic cause or other, which never recur. When the heart muscle is sound, particularly in young people who get periodical attacks, life may not be curtailed at all, and there is quite a chance of getting rid of the attacks. Death has occurred in a paroxysm, though the greater number of them pass off. If called to a patient during an attack, it will almost certainly be correct to assure him or his friends that he will recover from this particular paroxysm.

**Treatment.**—Careful questioning will sometimes elicit a definite cause for the attacks, such as strong coffee, excess of smoking, alcohol, emotion, infection, such as an acute coryza, sudden strain. All these should be guarded against. Lewis states that a broad abdominal binder put on before rising and worn during the day-time has kept off the attacks. Sometimes a full course of digitalis may be successful. There are many ways in which the attacks themselves have been stopped, such as assuming a particular attitude, *i.e.* bending down with the head between the knees, crawling on the hands and knees, or resting in the knee-elbow position, or lying supine. The induction of vomiting, the relief of flatulence, applying ice to the præcordium or a tight binder round the abdomen, pressing on the vagi in the neck, intravenous injection of digitalin or strophanthin, respiratory effort by making rapidly deep inspirations and expirations, have all been successful in arresting attacks. Sometimes patients find out something for themselves which invariably stops the attack at will. In three cases parathyroid ($\frac{1}{10}$ gr. three times a day) was successful (**6**).

During long attacks it is essential to make the patient comfortable and to induce sleep by opiates if necessary. Signs of oncoming cardiac failure will

require appropriate treatment. Venesection may be useful. A history of attacks does not preclude the use of a general anæsthetic, if an operation is necessary.

## AURICULAR FLUTTER

This name has been given to a form of tachycardia, in which the auricle contracts very rapidly—from 200 to 330 in different cases—but the ventricle usually beats only with one-half or one-quarter of this frequency, owing to some degree of auriculo-ventricular heart block becoming established. Thus it appears that only every second or every fourth auricular contraction is conducted to the ventricle. Since in auricular flutter the auricle does not beat faster than 330 in the minute, this form of tachycardia may be excluded from consideration by a radial pulse rate of more than 160; on the other hand, if the ventricular rate is only one-fourth of the auricular rate, it is clear that the pulse, being only seventy or eighty per minute, would give rise to no suspicion of the existence of a tachycardia. The disease, in such a case, being a tachycardia of the auricles only, could

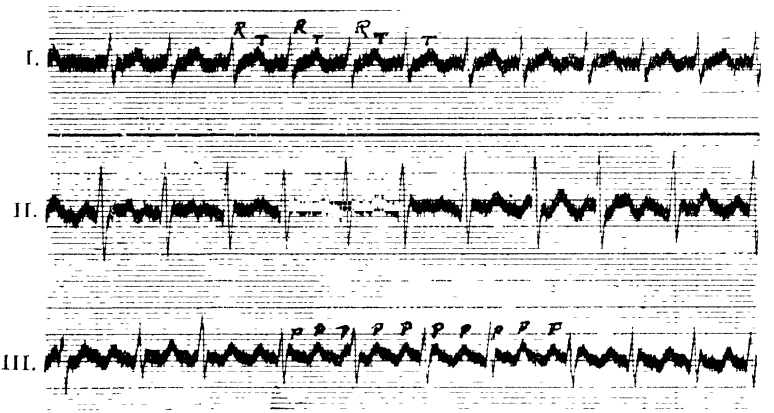



FIG. 43.—Electro-cardiogram of Auricular Flutter. In Lead I. the ventricular waves, R and T, are well marked, and are calculated to occur at the rate of 140 in the minute; no auricular waves, P, can be readily distinguished. In Lead III. the auricular waves, P, are well shown, and are calculated to be at the rate of 312, a little more than double the ventricular waves. (*After Russell Wells.*)

be detected only by a venous pulse tracing, or by the electro-cardiogram, in which latter the auricular beats (P) will be two, or four, for every ventricular beat (R). But this uniformity is not always maintained, and irregular pulses may occur. The tachycardia begins and ends abruptly, and is uninfluenced by position and exercise in the same way as the more usual forms of paroxysmal tachycardia. But the condition is much more likely to continue for long periods, and is less often temporary (*see* Fig. 43).

**Pathology.**—If a ring of muscle is cut out of an auricle, and this is stimulated at one point, two waves of contraction start from this point and pass round in opposite directions with the same velocity, meeting at the opposite side of the ring. Since at the time they meet the muscle is contracting, it is also *refractory*, so that the waves cannot cross one another, and they die out (Mines). The normal heart beat in the auricle may be considered as a succession of beats of this kind which pass round the auricle and die away when the waves meet each other. Suppose there was a sudden temporary block in the original ring, so that the wave could only travel round in one direction, it would continue round and round in the same direction, since there is no wave in the opposite direction to stop it by making

the muscle refractory; it would go on travelling round indefinitely. Such a state of affairs occurs in auricular flutter, the wave travelling round and round the auricular muscle, the P-wave on the electro-cardiogram corresponding to one complete circuit (Lewis). As the wave of excitation passes round it produces offshoots which pass all over the auricular muscle, producing contraction and exciting the ventricles. However, some degree of A.V. block always becomes established, so that the ventricles only respond to every two, three or four auricular beats.

**Treatment.**—Digitalis is found to have a beneficial effect in the cases in which the pulse rate is from 130 to 170, and the auricular beat has twice that rate. The drug appears to increase the resistance to the transmission of impulses along the A.V. bundle, and thus the ventricular rate is diminished and the pulse is slowed. In many cases the digitalis upsets the regular though rapid contractions of the auricle, and induces auricular fibrillation (*see* p. 314). If the digitalis is then stopped, the heart may take on its normal action, instead of going back to the state of auricular flutter. Alternatively, quinidine may be used either to stop the flutter in the first place, or to stop the fibrillation which has been induced by digitalis treatment (see p. 316).

## PULSUS ALTERNANS

In this form of abnormal heart beat there is a regular alternation of small and large beats, but, unlike the pulsus bigeminus, or coupled beats, in which the interval following the smaller beat is longer than that following the larger beat, the intervals throughout are almost exactly uniform. If the difference between a small and a large pulse beat is not very marked, it may be unrecognised by the finger, and the sphygmograph may be necessary to demonstrate the condition. If the difference is pronounced, that is, if the alternate weak beats are very small, they may be missed by the finger, and the pulse may be thought to be abnormally slow, that is, slower by one-half than it actually is. The condition may also be recognised by taking the blood pressure with a sphygmomanometer. At a certain pressure the weaker beats are eliminated, and the pulse at the wrist apparently drops to half its original rate.

This abnormality is certainly due to defective contractility or exhaustion of the myocardium; it is increased or made manifest by exercise; it may be temporary and disappear. But if it is continuous it points to a persistent cause for the defect of contractile power, as, for instance, myocardial degeneration. Experience shows that pronounced cases rarely last more than two years, and not infrequently death is sudden. The prognosis is worse if alternation is observed when the pulse is slow. It is not so bad if the condition only appears when the heart is beating rapidly.

**Treatment.**—The heart must be rested in order to conserve its power. Digitalis has been found of value in those cases where the pulse is quick, especially if œdema is present. Its administration slows the pulse and abolishes the alternation.

## AURICULAR FIBRILLATION

The importance of this condition is indicated by the fact that more than half the cases admitted into a general hospital for heart failure suffer from auricular fibrillation. Cases of auricular fibrillation can be divided into two groups, rheumatic and non-rheumatic; the rheumatic group is about twice as large as the non-rheumatic, and of the rheumatic group about two-thirds are cases of mitral stenosis. Roughly speaking, of all cases of auricular fibrillation met with one-half will be cases of mitral stenosis.

**Pathology.**—Auricular fibrillation in the human subject was first described in 1909, independently by Rothberger and Winterburg, and by Lewis in this

country. The clinical condition had been known from the earliest times, and the pulse had been called the pulsus irregularis perpetuus. Mackenzie observed that in jugular tracings the *a* wave was absent, and he first of all regarded this as due to paralysis of the auricle, but he soon discarded this view, and considered that the auricle and ventricle were beating simultaneously. He called this "nodal rhythm," because he thought the stimulus started in the auriculo-ventricular

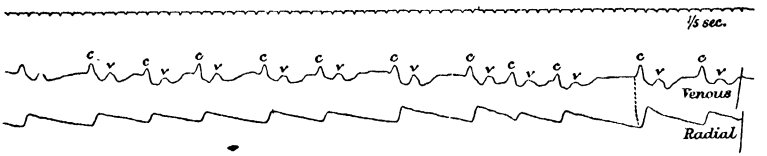



FIG. 44.—A Polygraphic Record from a Patient with Complete Irregularity of the Heart. Each systole of the ventricle is accompanied by *c* and *v* waves in the upper or venous curve. There is a total absence of the usual pre-systolic wave *a*. The absence of *a* and the presence of the irregularity are attributed to fibrillation of the auricle. (*After Lewis.*)

node. It is now known that nodal rhythm is something quite different. The real nature of the clinical condition was recognised by comparing electro-cardiograms and jugular pulse tracings of patients with tracings from dogs in which the auricles were fibrillating, owing to stimulation with the faradic current. Recent experiments by Lewis and his co-workers have thrown additional light on the pathology of the condition. It has already been explained that auricular flutter is due to a wave of contraction passing continuously round and round the auricle. Fibrillation is also due to a circulating wave; but in this condition the wave travels much faster, *i.e.* up to 600 revolutions per minute, the circumference

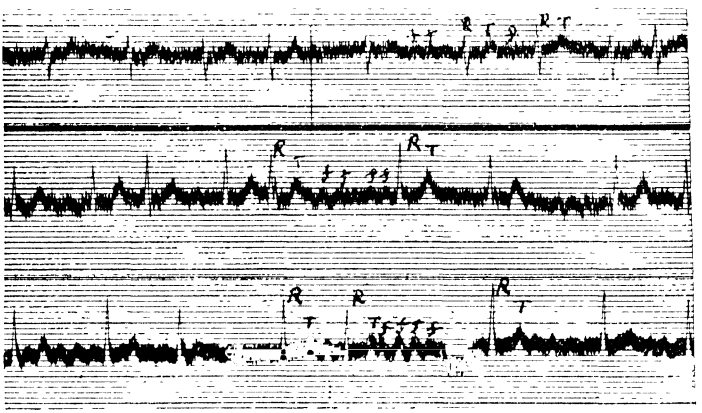


FIG. 45.—Electro-cardiogram of Auricular Fibrillation. The ventricular waves, R and T, are very irregularly placed; and there are no auricular waves, P, but their place is taken by numerous small waves, *f*, scattered through the curve. (*After Russell Wells.*)

of the circle is smaller, the path is more irregular, and the offshoots over the auricular wall are also much more irregular. The reason for this is that, when the wave is travelling so quickly, the auricular muscle over which the wave is travelling is in a partly refractory state, so that only certain fibres are capable of conducting the impulse, and these change each time the main circulating wave comes round again, producing extreme irregularity in the path of the contractions. The A.V. node, like the rest of the muscle, experiences these irregular

excitations, so that the ventricle also beats irregularly; but some degree of A.V block is always established, and so the ventricle does not respond to each impulse but in spite of this in an untreated case the ventricular rate is rapid and irregular.

**Diagnosis.**—The recognition of auricular fibrillation is in most cases comparatively easy. The apex beat and the pulse both show a complete absence of any dominant rhythm. The beats are completely irregular both in strength and in rate. If a radial tracing shows the irregularity well, it will be noticed that at the end of one of the longer pauses a comparatively weak beat is obtained instead of the strong beat that might be expected. *Vice versâ*, after a short pause a strong beat may be observed (*see* Fig. 44).

When the rate of the heart is quick, on comparing the pulse with the heart beat as heard by auscultation, it is found that many heart beats entirely fail to get through to the wrist. In cases of mitral stenosis there is never a true auriculo-systolic murmur, because the auricle has ceased to beat. The murmur, if heard at all, will always be early or mid-diastolic. Occasionally it becomes pre-systolic, but that is only because the heart sometimes contracts before the diastolic murmur is complete. A pre-systolic murmur is never heard coming after the end of the mid-diastolic murmur when the pause is sufficiently long for the latter murmur to be completed.

A heart beating irregularly, and at a greater rate than 120 a minute, is almost bound to be a case of auricular fibrillation. When the heart is beating slowly, say under 100, and not very irregularly, recognition is more difficult. However, there is this further valuable sign: The patient is asked to take some mild form of exercise, such as bending up and down or sitting up and lying down in bed once or twice. If auricular fibrillation is present, the exercise will make the heart more irregular. In other conditions, such as extra systoles or heart block, the pulse becomes more regular with exercise.

**Prognosis.**—When auricular fibrillation has once developed, it usually persists for the rest of the patient's life. It is a serious condition. Not many patients live longer than ten years. The immediate prognosis depends largely on being able to keep the pulse rate below 90, which is a favourable sign, and this is the great aim in treatment. Quinidine has recently been successfully used as a cure in about 50 per cent. of cases.

**Treatment.**—The great reputation digitalis possesses in heart disease rests chiefly on its success in treating this condition.

Digitalis was first introduced into medicine by William Withering, a physician of Birmingham, in 1775. He obtained a family recipe for the cure of dropsy which had long been kept secret by an old woman of Shropshire, who had effected cures when the more regular practitioners had failed. The medicine contained twenty or more different herbs, but Withering picked out the foxglove as being the active one. His book describes 163 cases, but he pays more attention to the disappearance of dropsy and the corresponding diuresis than to the effect on the heart, though this is specifically mentioned in a few cases. However, he makes the following note: "It has a power over the motion of the heart to a degree yet unobserved in any other medicine."

If the pulse is over 100, the patient should be put to bed and treated with 10 to 15 minims of tincture of digitalis three or four times a day, or the fresh infusion may be used. If there is no slowing of the pulse, the dose is increased until definite symptoms of digitalis poisoning begin to appear. These are nausea, vomiting, diarrhœa, and headache. The dose must now be diminished sufficiently to avoid these symptoms, and it may be continued until the desired effect is obtained. If unsuccessful, digitalin or digitoxin, gr. $\frac{1}{250}$ by the mouth, or, if necessary, strophanthus, may be tried.

In acute or very serious cases, strophanthin, gr. $\frac{1}{200}$, may be given intravenously in normal saline, and the dose repeated. It may also be given subcutaneously or intramuscularly. Intravenous strophanthin is not very safe,

because the amount of the drug it is necessary to give to obtain the most beneficial therapeutic effect is close to the toxic dose. A safer method of treating acute cases of auricular fibrillation is that of Eggleston, who has shown that doses of digitalis much larger than those usually employed may be administered by mouth to patients with auricular fibrillation and cardiac failure, when it is important to produce a rapid and complete effect. It is essential to use a properly standardised preparation of digitalis. The unit for digitalis therapy in America is the "cat unit," *i.e.* the minimum lethal dose of digitalis for a cat of given weight (expressed in milligrammes of powdered leaves), when administered intravenously. The "cat unit" of most good preparations of digitalis in this country represents 100 milligrammes of the powdered leaves. Under these circumstances the total amount of digitalis that may be administered to a patient is given by the following formulæ:

Number of cubic centimetres of tr. digitalis = 0·15 × body weight in pounds.

Number of grammes of powdered leaves = 0·015 × body weight in pounds.

The normal body weight of the patient must be used, and in calculating this from the actual weight of the patient allowance must be made for any œdema that may be present. The weight predicted from the body-length might be used (see p. 560); the latter should be measured between the vertex and ischial tuberosities while the patient lies in bed. It is perhaps safer only to employ three-quarters of the calculated amount. This for an adult is usually found by calculation to be about 3 drachms of the tincture. It is best to give this in three or four doses at six-hourly intervals, beginning with half the total amount, then one-quarter, then one-sixth, then one-twelfth. This method of treatment is found to be safer than using strophanthin intravenously, because if the patient has any idiosyncrasy to digitalis, the drug will be vomited up, whereas it is impossible rapidly to get rid of an excess of a drug that has been administered intravenously.

The aim of digitalis treatment is to keep the pulse between 80 and 90 while the patient is lying in bed. When this is attained, the patient may get up, and the dose is then gradually diminished, when it will usually be found that the pulse remains slow. The minimum dose necessary for this is determined, and the patient should continue taking this during the remainder of his life. If, in spite of digitalis treatment, the pulse rate still remains high, nothing more can be done with this kind of drug. The other indications are to avoid excessive exertion, to avoid infection, and pregnancy, and constipation, and to keep as healthy as possible. General anæsthetics should only be given if the patient's life can be considerably prolonged by an operation, *i.e.* when one is practically unavoidable.

The action of digitalis in slowing the heart is due to the production of heart block, in degree, sufficient to stop the numerous impulses from the auricles which are overwhelming the ventricle. This heart block is probably due to direct action on the cardiac muscle, presumably somewhere in the auriculo-ventricular bundle or at the A.V. node. In healthy hearts digitalis acts by producing vagal inhibition. When the digitalis is pushed to too great an extent, apart from the other symptoms of poisoning, "digitalis coupling" may occur. The pulse is slow, and the heart beats alternately strongly and weakly. When this occurs the digitalis should be omitted. It is a sign of danger.

Quinidine was introduced by Frey in 1918 for the treatment of auricular fibrillation. It acts on the auricular muscle in two ways: (1) It lengthens the refractory period. It has been pointed out that in auricular fibrillation there is a rapid circus movement round the auricle, and further the wave stretches nearly round, so that there is only a very small gap of excitable muscle between the head of the advancing wave and its receding tail. When the refractory period increases, the muscle in the small gap becomes inexcitable, and the move-

ment stops. (2) It lessens the conduction of the auricle, so that the wave travels slower. This action is opposed to the previous one, and tends to the perpetuation of the wave because the gap of excitable muscle is increased. The effect of quinidine depends on which of these actions is the stronger. If the first is, the auricular fibrillation will cease, and the heart will take up its normal rhythm again. This occurs in about half the cases; in the other half the drug is of no use, and this may be because the second action prevails.

Before beginning treatment with quinidine, it is best to give a preliminary course of digitalis treatment with the patient at rest in bed, so as to reduce the ventricular rate to between 70 and 80, and improve his condition. If no improvement takes place, the case is unsuitable for quinidine, since the patient's original condition must have been due rather to defects in the heart itself than to the abnormal rhythm. Cases of auricular fibrillation, with a slow ventricular rate independently of digitalis, are also not very suitable for quinidine treatment. A history of recent embolism is also a contra-indication because a thrombus, if present in the auricle, may become loose and cause embolism when the auricle begins to beat normally again.

If quinidine treatment has been decided on, the digitalis is stopped, and on the next day a preliminary dose of 0·2 gramme of quinidine sulphate is given by mouth to see whether there is any idiosyncrasy to the drug. It is then given in doses of 0·4 gramme in gelatine capsules, at first once a day, then twice a day, and then three or four times a day. A careful watch is kept on the rate and regularity of the pulse, and the action of the drug may be further elucidated by means of electro-cardiograms. It is best to use direct leads from the chest instead of from the limbs (Drury and Iliescu). The ventricular rate increases, to begin with, while the auricular rate falls. Occasionally quinidine causes urticaria, headache, dizziness, disturbances of vision, nausea, vomiting and pyrexia. The drug must certainly be stopped if there are signs of heart failure, or if the patient complains of præcordial discomfort or palpitations. When the auricular fibrillation has been abolished it is as well to continue with small doses of quinidine (0·8 gramme per diem) for some weeks; the amount can be gradually reduced and the drug eventually omitted. A relapse, which is not uncommon, should be treated along the same lines.

In general, patients feel more comfortable when the heart's rhythm is normal, and for this reason there is an advantage in quinidine over digitalis, which does not abolish fibrillation. Quinidine may be given both in permanent and in paroxysmal auricular fibrillation. Thyro-toxic fibrillation cases do well with quinidine if the hyper-thyroidism has been efficiently treated. Quinidine and digitalis should not be given simultaneously.

## INFREQUENT ACTION

### (*Bradycardia*)

Although seventy in the minute is often regarded as the normal frequency of the radial pulse and heart, a pulse of sixty per minute is quite common. A pulse of fifty-three or fifty is normal in some individuals, and the rate in these slow-pulsed persons may fall to forty-eight in the cold hours of midnight or early morning. A slow pulse rate is observed in starvation or under-nutrition, and is, no doubt, associated with the lessened metabolism that occurs.

In examining for this condition it is, of course, essential to observe the heart as well as the radial pulse, as there are several conditions in which the beat of the heart may not get through to the wrist, such as pulsus alternans or pulsus bigeminus. Bradycardia might be suspected if the pulse only were felt, whereas the heart would be beating at a normal rate.

Many conditions of bradycardia are due to vagal stimulation, which may be produced reflexly. The slow pulse of cerebral compression, jaundice, conva-

lescence after influenza and other acute infective diseases is due to this. The same is the case with the slowing of the whole heart that causes the fainting attack so commonly due to emotion or prolonged standing, and possibly the fainting attacks in aortic disease.

The alternate slowing and quickening of the heart during respiration (sinus arrhythmia) is also due to vagal action, and the rather uncommon "phasic irregularity," where the whole heart slows periodically, independently of respiration and without apparent reason.

Sino-auricular block is another uncommon condition producing bradycardia, but sino-auricular block itself may be due to vagal stimulation. A fairly common cause of prolonged bradycardia is to be found in auriculo-ventricular block, especially where the block is complete.

### VENTRICULAR FIBRILLATION

By means of the electro-cardiograph the condition of fibrillation has been recognised as occurring in the ventricle as well as in the auricle.

It is observed as a rule immediately before death, though not necessarily as the cause of death; but in animals, and on rare occasions in man, it has ceased in time for recovery to take place. It appears to be the cause of death in lightning stroke, and is believed by some to be the explanation of some of the fatal cases of chloroform anæsthesia.

# DISEASES OF THE HEART

The diseases of the heart must be considered in relation to three separate structures: the *myocardium*, or muscular substance of the organ; the *endocardium*, with the valves which are so closely related to it; and the *pericardium*, or membrane surrounding the heart.

## HYPERTROPHY

Hypertrophy is increase in size of the walls of the heart. It may be associated with increase of the heart's cavities (dilatation), or it may occur alone. Hypertrophy of the heart is the natural response of the body to an increased amount of work thrown upon the heart, and can take place so long as nutrition is well maintained by a proper supply of blood.

Accurate information can be obtained from the use of X-rays as to the size and outline of the heart, but without discrimination between hypertrophy and dilatation. By such means it has been found that the size of the heart is larger in athletes than in sedentary workers, and in a group of undergraduates it has been found that the size of the heart increased when they became accustomed to regular strenuous exercise. This increase in size was presumably due to hypertrophy.

Probably the most accurate information as to the relative hypertrophy of right and left ventricle can be obtained by the electro-cardiograph. Thus in the normal heart (*see* Fig. 32) the first ventricular wave, R, in the second lead is higher than that in the third lead. This is due to the fact that the heart is tilted to one side and its axis is not coincident with that of the body. An enlargement of the heart to the left, as in hypertrophy of the left ventricle, will shift the electrical axis of the heart still more, and as a result there will be a greater difference between the R-waves in the second and third leads; the wave, R, will be tall in the former, and short or even inverted in the latter. The converse

effect is seen if there is hypertrophy or enlargement of the right side; that is, the wave in the second or axial lead is small, and that in the third lead is large.

Lewis and Cotton have proved the accuracy of this method of estimating hypertrophy by carefully dissecting the heart after death, so as to separate the right from the left ventricle, and comparing the weights of the two. In normal cases the ratio of left to right ventricle was 1·8 to 1. In marked mitral stenosis it was 1·25 to 1, showing preponderance of the right ventricle. In aortic disease there was left-sided preponderance in eight cases, while in seven cases the hypertrophy of the right side was as marked as the left. In the hypertrophy associated with interstitial nephritis there was usually slight left preponderance, the ratio being 2·07 to 1 on the average.

Other causes of left-sided hypertrophy are—(1) disease of the aortic valves with narrowing of the orifice; (2) very rarely, congenital narrowing of the aorta; (3) increased arterial tension; (4) continued rapid action of the heart, such as occurs in exophthalmic goitre; (5) pericardial adhesions, which interfere with the movements of the heart and so throw extra work upon it.

Hypertrophy of the right ventricle arises, in the majority of cases, from obstruction to the pulmonary circulation, (1) at the pulmonary orifice, from congenital malformations of the valves, congenital constriction of the orifice, acquired disease of the pulmonary valves, or pressure on the base of the pulmonary artery by aortic aneurysm; (2) in the lungs, from emphysema, chronic bronchitis, bronchiectasis, and occasionally chronic phthisis; and (3) from primary disease on the left side of the heart, whereby the left auricle and pulmonary circulation become engorged, with rise of blood pressure in the lungs.

**Anatomical Changes.**—In hypertrophy of the left ventricle, the heart is enlarged downwards: and the ventricular wall may be double its normal thickness. It is in aortic regurgitation that the highest degree of hypertrophy is reached. In such cases there is always at the same time dilatation of the ventricle, and proportionate changes in the other cavities, so that the weight of the heart may be anything between 600 and 1,300 grammes. Such examples are known as the *cor bovinum*. In hypertrophy of the right ventricle its apex encroaches on the apex of the heart, and in marked cases the left ventricle is hardly visible when looking at the heart from the front. The right ventricle also extends further to the right than usual.

**Physical Signs.**—Lewis has found that not all the ordinarily accepted physical signs of right- and left-sided hypertrophy are very reliable. They are as follows: In the case of left-sided hypertrophy, in pronounced cases the impulse is heaving and diffused over a wide area, so as to be manifest to the eye or the hand over a space of 2 or 3 square inches by a movement of elevation communicated to the ribs as well as to the intercostal space; indeed, the chest may be permanently bulged by the enlarged heart. The position of the impulse is altered, so as to be lower and farther out than the normal. The præcordial dulness tends to be increased in proportion to the enlargement. It may reach externally 1 inch or more beyond the left nipple. It must, however, be allowed that in so much enlargement dilatation has a considerable share, and that in pure uncomplicated hypertrophy the increase of the præcordial dulness may be very slight. Emphysema of the lungs may completely conceal an enlarged heart. A permanently raised blood pressure always means a hypertrophied left ventricle.

The signs of hypertrophy of the right ventricle are analogous to those of left-sided hypertrophy. A systolic impulse may be seen in the epigastrium due often to the impact of the right ventricle against the liver rather than to direct contact of the ventricle with the abdominal walls at this point; the apex beat may also be carried somewhat to the left.

The auscultatory signs vary, as the conditions which cause the hypertrophy

themselves may modify the heart sounds; for instance, disease of the valves and altered conditions of the arterial circulation. Thus, valvular diseases give rise to murmurs, accompanying either the first or the second sound, such as have already been described (*see* p. 288); and high blood pressure itself modifies the heart sounds. The first sound is duller or more muffled than normal, because the muscular element plays a larger part in its production; at the same time the sound from the stretching auriculo-ventricular valves has to travel through a greater thickness of muscle and is partly lost in consequence. The aortic second sound is accentuated.

**Diagnosis.**—It must be recognised, first of all, that we may entirely fail to demonstrate hypertrophy of the heart by the ordinary physical methods, although it is afterwards shown at the autopsy to be enlarged. Hypertrophy may be simulated by the following conditions: (1) Over-action of the heart from *excitement*. This condition is often seen in young people apparently well, examined perhaps for an appointment or for life insurance. The action of the heart is here generally rapid, the beat is quick and sudden rather than slow and heaving; the patient is obviously nervous, and the condition is easily shown to be merely temporary. A cardio-pulmonary murmur is sometimes heard under such circumstances (*see* p. 293). (2) The heart may be uncovered from retraction of the left lung, and a greater surface being in contact with the chest wall, the impulse may be more extensive and may appear more forcible than usual. (3) *Pericardial effusion* is frequently confounded with hypertrophy and dilatation, perhaps more often with the latter (*see* Pericarditis). (4) Displacement of the heart from new growths, or pleuritic effusion. (5) Aneurysm. The use of X-rays will give much assistance in most cases. In all enlargements of the heart the relative share of hypertrophy and dilatation must, if possible, be estimated. Probably no considerable enlargement takes place without dilatation. Moderate enlargements may arise acutely from dilatation; and dilatation may be inferred to be greater, the more feeble the impulse, so long as emphysema of the lung can be excluded.

*Hypertrophy of the Auricles.* This rarely occurs without dilatation, but predominates in the left auricle in mitral stenosis. It arises from constriction or incompetence of the auriculo-ventricular valves, or from hypertrophy and dilatation of either ventricle acting back upon the corresponding auricle.

## DILATATION

**Ætiology.** The term *dilatation* is applied to an increase in the size of the cavities of the heart during diastole. It is possible that in certain pathological conditions the ventricles fail to contract completely on their contents, so as to leave some residual blood, but nothing is known for certain about this in man, although it may occur experimentally in the "heart-lung preparation" (Starling).

There are two kinds of dilatation. (1) There is the compensatory dilatation of certain types of valvular disease, due to the fact that the particular chamber of the heart has to accommodate an extra quantity of blood in order to keep up the circulation. This is compatible with a perfectly healthy myocardium. (2) There is the dilatation, rapid or gradual, that spells the onset of cardiac failure and may be associated with valvular disease, Bright's disease and arterio-sclerosis It may be due to yielding of the ventricular walls, which is promoted by various degenerative changes in the myocardium; these are the fatty, fibroid, and senile degenerations, which follow upon anæmia, chlorosis, and severe or prolonged infections, and the myocarditis and pericarditis of acute rheumatism and coronary sclerosis. The excessive indulgence in alcohol is sometimes a cause of cardiac dilatation with all its consequences. Although this is the usual explanation of

dilatation, Starling's experiments suggest that it, too, may be a compensatory mechanism, enabling the heart to beat more forcibly because the greater the relaxation of the cardiac muscle-fibre, the stronger the beat. This is called the "Law of the Heart."

It is probable that slight dilatation of the heart occurs normally during muscular exercise, the effect of this being to increase the output of blood at each beat. It is, however, quite certain from X-ray evidence that immediately after exercise the heart normally contracts down so as to become slightly smaller than its resting size. On the other hand, it has been found that in cases of incipient cardiac failure the heart shows dilatation after muscular exercise.

Cases of acute dilatation of the heart after a sudden strain have been described, but although it is probable that such a condition does occasionally occur in a diseased heart, the evidence of its actual occurrence is not satisfactory. No doubt some cases of dilatation that have been described were really cases of paroxysmal tachycardia. It is doubtful if acute dilatation ever affects a healthy heart, but this is still a disputed point.

**Anatomical Changes.** The effects on the size and shape of the heart vary with the cavity concerned. In general dilatation the heart becomes more globular, and is widened transversely. The dilated left ventricle increases to the left; when the right ventricle is much dilated, the triangular shape of the heart is lost, it becomes more globular, and the apex is formed partly by the right ventricle instead of being formed entirely by the left. The thickness of the walls will depend on the presence or absence of accompanying hypertrophy. In dilatation with thinning, the ventricular walls may be reduced to $\frac{1}{6}$ inch, and even less at the apex, which is commonly the thinnest part. The auriculo-ventricular orifices share in the dilatation, and incompetence of the valves often results.

The auricles may undergo very considerable dilatation, and this is often accompanied by some hypertrophy of the auricular walls.

**Symptoms.**—Dilatation of the heart accompanying gradual cardiac failure will be accompanied by the symptoms of cardiac failure. Various irregularities in the mechanism of the heart beat may also be present. There may be, in addition, symptoms due to the lesion that is causing the dilatation, whether valvular disease, fatty or fibroid degeneration, etc. The immediate effects of dilatation as seen in infectious diseases have been described (*see* p. 19). Acute dilatation may give rise to faintness, syncope, vomiting, and even insensibility and death.

**Physical Signs.**—The increased size of the heart, as indicated by enlargement of the præcordial dulness or of the shadow of the heart produced by X-rays, may be due to hypertrophy or dilatation or both of these combined. However, it is probable that any considerable enlargement of the heart observed by these methods is chiefly due to dilatation. Any enlargement to the right of the sternum is due to auricular dilatation, because the auricular wall, even if hypertrophied, is quite thin; and it is practically always *right* auricular dilatation. But in several cases of mitral disease, with enormous enlargement of the left auricle, the latter has actually formed the right border of the heart (**7**). It is often possible to feel the beat of the heart by placing the fingers in the intercostal spaces over the dilated auricle. Dilatation of the left auricle may cause dysphagia by pressure on the œsophagus. It may compress the lower part of the left lung or the left bronchus, or cause paralysis of the left vocal cord. The left auricle may be observed on the X-ray screen when the patient stands in the oblique position, facing the tube, with his right shoulder towards the observer. The rays pass through the posterior mediastinum, between the vertebral column and the left auricle.

Dilatation of the ventricles causes an increase of the præcordial dulness and of the X-ray shadow to the left of the sternum. In extreme cases the shadow

reaches the left wall of the chest, and the impulse can be felt in the axilla. Dilatation of the left ventricle is often accompanied by a systolic murmur, due to mitral regurgitation, and dilatation of the right ventricle often causes tricuspid regurgitation.

**Diagnosis.**—Dilatation has an important share in all the considerable enlargements of the heart, and it may be confounded with the several lesions mentioned under Hypertrophy (*see* p. 318). The chief physical evidence is the displacement of the impulse towards or beyond the nipple line; and if that is produced by pleural effusion on the right side, or by aneurysm, some other proof of the existence of such a lesion will probably be afforded. The triangular dulness of pericardial effusion on the right side may be like that of a dilated right auricle, and it may be very difficult to distinguish the two, but there are many points of distinction, which, besides the associated vascular conditions and the history or presence of a rub, may be of value (*see* p. 357).

## MYOCARDITIS

Myocarditis, or inflammation of the muscle of the heart, may be either acute or chronic. *Chronic myocarditis* can only be recognised in its final stage of fibroid change, and is included in the description of fibroid degeneration (*see* p. 324).

*Acute myocarditis* occurs mostly in connection with pericarditis or endocarditis as a part of rheumatic fever. In fatal cases of pericarditis, the layer of muscular tissue immediately under the pericardium is sometimes seen to be paler than normal, as if fatty; and in cases of adherent pericardium, bands of fibrous tissue may be found extending from the surface into the substance of the heart. According to Carey Coombs, rheumatic myocarditis in young persons shows the following changes: In the muscular fibre cells, fatty granules and droplets first appearing near the nucleus; in the interstitial tissue, first, nodules consisting of large fusiform cells, of which many are multinuclear, and, secondly, collections of leucocytes, chiefly polymorphonuclear. The nodules are closely related to the arteries and arterioles, and occur deeply in the muscular wall and near the root of the aorta and the mitral ring. They are more common in the left ventricle, and they terminate in cloudy swelling or fibrous transformation (cicatrix). These groups of cells are usually known as Aschoff's nodes.

A more local inflammation of the myocardium results from malignant endocarditis, where ulceration of a valve extends to its base, and then invades the muscle; or where vegetations or semi-detached fragments set up ulceration in adjacent parts of the endocardium by friction or contact, and this involves the myocardium. The effects upon the general circulation may be of the same kind as those of malignant endocarditis.

A third form is *suppurative myocarditis*, which is chiefly the result of pyæmia. Small abscesses occur in the substance of the heart, mostly in the wall of the left ventricle, and may approach so near to the pericardium as to rupture into its cavity and set up acute pericarditis. This form of myocarditis occurs especially in connection with, and secondarily to, acute necrosis of the long bones (*see* p. 51).

The **Symptoms** of myocarditis are not always pronounced (*see* also under Acute Endocarditis). It diminishes the contractile power of the heart, the first sound is fainter, and the pulse becomes feebler or irregular, or there may be tachycardia or heart block; there may be dyspnœa and tendency to collapse if the heart begins to fail. Dilatation with increased præcordial dulness may be present. A systolic murmur is often heard at the apex of the heart, and this is probably due to yielding of the mitral ring, causing regurgitation, but it may possibly be due to vegetations on the mitral valve (*see* p. 328).

The symptoms of abscess of the heart are uncertain, and the diagnosis from physical signs is equally obscure.

**Prognosis** and **Treatment.**—See under Acute Endocarditis.

## DEGENERATION OF THE MYOCARDIUM

The muscular wall of the heart is liable to the following forms of degeneration: *pigmentary, fatty,* and *fibroid.*

### PIGMENTARY DEGENERATION

*(Brown Atrophy of the Heart)*

The heart is smaller than normal, and the muscular fibre, instead of having a full red colour, is of a dull brownish red, and softer and more friable than is natural. Under the microscope the fibrillæ are seen to contain a number of minute yellow granules. It occurs in senile and cachectic conditions, being common in fatal cases of malignant disease of other organs.

### FATTY DEGENERATION

This change in the muscular fibres must be distinguished from the deposit of fat about the heart (*see* p. 326). In the latter the ordinary adipose tissue is deposited beneath the pericardium, and invades the muscular fibres because the connective tissue cells between the fibres become loaded with fat. In the former, or true fatty degeneration, the muscular fibrillæ themselves are the seat of minute fat granules, which replace the true sarcous elements and rob the muscle of so much of its contractile tissue. This true fatty degeneration occurs in different forms: the muscular wall may be uniformly affected, or the fatty changes may be limited to a small patch, or to the layer underlying the pericardium, as described under Myocarditis, or it may consist of streaks and lines on the inner surface of the heart. When the affection is general the heart is of softer consistence, more easily lacerable, of pale pink or buff colour, and often somewhat larger than normal, from yielding of the affected muscular tissue. When the fat is deposited in lines or streaks it gives a characteristic appearance, the lines of pale yellow colour being often arranged upon the darker red muscle, like the markings of a tabby cat. They are seen mostly on the musculi papillares, on the posterior wall of either ventricle, and on the septum in the right ventricle. Fatty degeneration is common in hypertrophied hearts, and may be present even when the muscle has quite a normal colour.

**Ætiology.**—The causes of fatty degeneration of the heart are general and local. It may be the result of a general tendency to degeneration, such as occurs at an advanced age, and is accompanied by obstruction of the coronary arteries due to atheroma or syphilitic changes, by which the nutrition of the heart wall is impaired. On the other hand, fatty degeneration is seen constantly in pernicious anæmia, and often in other forms of anæmia, in purpura and scurvy, and in cachectic conditions, such as phthisis and cancer; in poisoning by phosphorus, by some mineral substances (lead, antimony, arsenic), and in chronic alcoholism. In most of the acute febrile diseases the consistency of the heart is sometimes altered, as the result of a finely granular condition of the muscular fibres, which is probably not to be separated from fatty degeneration. This is the case in enteric and typhus fevers, in yellow fever, diphtheria, small-pox, and measles. Fatty degeneration also arises from myocarditis, and is seen in connection with long-standing valvular disease, with renal disease, and in hypertrophy or dilatation from other causes.

**Treatment.**—This must consist in avoidance of undue exertion, of making

efforts with the breath held, or of mental excitement: in moderate doses of stimulants, and the exhibition of tonics, such as quinine, arsenic, iron, and strychnine. Digitalis must be given with caution, and only in cases where the beat is frequent and irregular, with evidence of dilatation.

Cardiac failure in enteric fever requires the free employment of stimulants, with ammonia and digitalis in frequent doses.

### FIBROID DEGENERATION

In this form of degeneration, the muscular tissue of the heart is replaced by white fibrous or connective tissue. The change is in most instances partial, so that streaks and patches of a white, yellowish-white, or grey colour are seen deep in the muscular substance. It affects the lower third of the ventricle, the lower third of the septum, the musculi papillares, and sometimes the bases of diseased valves. Only occasionally is the ventricle almost entirely converted into fibrous tissue, but even here some traces of muscular fibre may be found on microscopical examination. A very slight degree of this must often result from rheumatic myocarditis, and in more pronounced cases the co-existence of pericardial or endocardial lesions will sometimes show its inflammatory origin (*chronic myocarditis, interstitial myocarditis*). On the other hand, fibrosis of the heart is often due to obstruction of the coronary arteries, and may be associated with fatty degeneration. Under these circumstances the blood supply is insufficient for the nutrition of active muscle. The latter atrophies, and is replaced by fatty or fibrous tissue. If the coronary obstruction takes place fairly quickly, fatty degeneration occurs. If the obstruction is more chronic, fibroid change predominates. It has been ascribed also to alcoholism, to long-continued congestion, and to syphilis (*syphilitic myocarditis*). A patch of fibrosis in the heart may also be a secondary result of infarct. The heart affected with fibroid disease is generally hypertrophied, and it may be dilated, or the subject of adherent pericardium; the affected part of the heart's wall is often thinner than normal, and it may be bulged out into a distinct aneurysm.

### SYMPTOMS OF MYOCARDIAL DEGENERATION

In early cases the patient complains of breathlessness on exertion. There may be præcordial pain, which may be noticed after exercise, or there may be definite anginal attacks. The patients may suffer from cerebral attacks, with unconsciousness and convulsions, but these attacks are probably due to some abnormality of the cardiac mechanism, such as heart block, or vagal stimulation. Appetite is often poor, and the patient may complain of indigestion. Sometimes these are the only symptoms complained of, and on inquiry the history of shortness of breath on exertion points to the heart as the primary cause of the gastric symptoms.

The physical signs attributed to myocardial degeneration are feeble cardiac impulse with faintness of the first heart sound, which may become equal to or less than the intensity of the second sound over the aortic area. This diminution of intensity gives a tic-tac character to the sounds when examined with a stethoscope, and this is further emphasised by a relative shortening of the diastolic interval, so that the sounds become more evenly spaced.

In cases of anæmia, the symptoms of cardiac insufficiency are merged in those due to the anæmia, and are discussed under this heading. In the course of severe pyrexial illnesses, like typhoid fever, the following symptoms suggest the occurrence of granular or fatty change in the myocardium. The pulse, hitherto rapid, becomes weak and irregular, the cardiac impulse is feeble, the apex beat is displaced outwards, and the first sound is so faint as to be scarcely audible. The patient becomes pale and in later stages livid.

The symptomatology of a group of cases showing myocardial changes has been

very carefully studied recently (**8**). The cases are met with very commonly. The patients are elderly, and suffer from shortness of breath, coming on at first after exertion, but later on they are more or less permanently dyspnœic. In some cases there is Cheyne-Stokes breathing, and in some other cases there are sudden attacks of extreme breathlessness, which come on particularly at night-time, and which might be described by the old-fashioned term *cardiac asthma*. There may or may not be orthopnœa present. Cyanosis is conspicuously absent, and if present at all its amount could hardly be considered enough to account for the dyspnœa. The rate of the heart is usually increased. The mechanism of the beat may be normal, but common irregularities such as those due to heart block, auricular fibrillations, extra-systoles, and alternation may be present. The heart is increased in size. Murmurs may be present. The body temperature tends to be subnormal. The advanced signs of cardiac failure, such as anasarca, congestion of the lungs and liver, may be present. There is usually some retention of urea in the blood, but the values are not extreme—at most 0·1 per cent., and usually 0·05 per cent. (normal 0·03). Albuminuria with casts may be present, and unless there are signs of venous stasis, the urine is increased in volume. Post-mortem examination shows that there is advanced coronary sclerosis of the heart, and there may be fatty degeneration. The kidneys show thickening of the vessels and some fibrosis, often of a patchy nature. The glomeruli are often congested, and some are hyaline, but there is no extensive obliteration of glomeruli, as occurs in chronic interstitial nephritis when the patient has died from uræmia. Sometimes these patients are described as cardio-renal cases, but it seems clear that although the kidneys always show some changes, and might be termed senile arterio-sclerotic kidneys (*see* p. 633), these are not the essential cause of the disease. The urea retention never reaches a fully uræmic figure of 0·2 or 0·3 per cent. Hence they are best described as cases of myocardial degeneration.

It was thought that the dyspnœa was due to increase of fixed acid in the blood (*see* p. 517), but it is clear that for some reason in these cases Barcroft's method of estimating this by the oxygen dissociation curve of the blood cannot be applied, and although in these cases the blood does part with its oxygen more readily than usual (meionexy), the reason for this is unknown (**9**). It has been recently discovered that the $CO_2$ in the arterial blood is increased in the dyspnœic cases, and the oxygen content of the arterial blood may be low. Consequently, it has been suggested that the dyspnœa, which may be extreme, is brought about by bronchial spasm (**10**). On this view, the term *asthma* can be used for this type of case (*see* p. 227). However, in pure cardiac dyspnœa, *e.g.* mitral stenosis, the $CO_2$ is low from increased respiration (**34**).

## RUPTURE OF THE HEART

Apart from injury, this is mostly a consequence of fatty degeneration or fibroid changes; in a very small proportion of cases, its cause is abscess, malignant endocarditis, aneurysm, or changes, perhaps syphilitic, in the coronary artery, leading to hæmorrhage into the myocardium. It occurs, like fatty degeneration, in old people, and not infrequently follows muscular efforts. The left ventricle has been the seat of the rupture in three-fourths of the cases on record. The patient is suddenly seized with intense cardiac pain, followed quickly by pallor, unconsciousness, a few convulsive twitchings, and death. In rare cases life has lasted some hours, or even days, with pallor, cold sweats, feeble pulse, and sighing respiration. It may then closely resemble the rupture of an aneurysm into the pericardium. Absolute rest, with the head low, maintenance of bodily warmth by external applications and of the circulation by small quantities of stimulants frequently administered, are clearly the indications for the almost hopeless treatment of such a condition.

## ANEURYSM OF THE HEART

Aneurysms of the heart may be acute or chronic.

*Acute aneurysms* arise from ulcerative endocarditis of the ventricle wall, in the manner which will be described under Malignant Endocarditis, and this is a rather frequent cause of aneurysm of the *pars membranacea septi*, or undefended space, as well as of the valves (*see* p. 331). Aneurysms of the pars membranacea are sometimes congenital. In either case, the sac opens towards the left ventricle. The condition is not recognisable during life.

*Chronic aneurysms* of the heart commonly arise in connection with fibroid degeneration. The cavity affected is weakened at one spot by this conversion of its muscular fibre into fibrous tissue, and dilates under the pressure of the blood into a sac. The left ventricle is their usual seat, and only a few cases are on record of aneurysms of the other three cavities. In two cases out of three they occupy the apex; they form rounded sacs, of which the communication from the ventricle may be of the same size as the sac itself, or very much smaller. The former variety is more frequent when they arise at the apex—that is, the aneurysm is continuous with the cavity of the ventricle; the latter, more sacculated variety, occurs more often at the side or the base of the ventricle. In size they have been compared to nuts, fowls' eggs, or small oranges; a few have been much larger. The walls are generally very thin, and sometimes infiltrated with calcareous matter; they are lined by endocardium, and mostly contain fibrinous coagulum. They have been found at all ages, from twelve upwards: and in males more often than in females. Aneurysms of the *coronary arteries* have been found on rare occasions. They are probably due to softening and consequent enlargement of the artery behind an embolus (**11**).

**Symptoms,** if present, are those of myocardial degeneration in those cases where the aneurysm is secondary to this cause. X-ray examination is a valuable means of diagnosis. In a considerable proportion of cases death has taken place either suddenly, and probably from syncope; or, as afterwards proved, from rupture of the aneurysm into the pericardium.

## FATTY OVERGROWTH

In this condition, which must be distinguished from fatty degeneration, the surface of the heart is overlaid by a large quantity of fat, so that the muscular fibres may be entirely concealed from view. The fat, which is simply an overgrowth of adipose tissue beneath the pericardium, encroaches upon the muscular wall, and, pressing upon the muscular fibres, causes them to waste to a certain extent. It occurs for the most part in persons suffering from general excess of fat, or obesity.

**Symptoms.**—In the more pronounced cases—like those of fatty degeneration proper, the result of enfeebled action of the heart—there are diminished impulse, faint sounds, and weak, small pulse; dyspnœa on exertion, and occasionally attacks of syncope. Death takes place suddenly in some instances. The symptoms may be in part due to accompanying atheroma of the arteries.

**Treatment.**—The treatment of fatty infiltration of the heart should be conducted much on the same lines as that of *obesity*, with due consideration of the fact that the heart is affected. Excess in eating and drinking should be avoided; alcoholic beverages should be forbidden. The necessary fluids are better taken shortly before or two hours after a meal than with it. The diet should consist chiefly of lean meat, chicken, fish, green vegetables, and ripe fruit, while starchy, saccharine, and fatty foods should be reduced to a minimum. Periods of fasting for a day or two at a time may be tried in order that the body may utilise its superfluous store of fat. The plan also may be

adopted of prescribing a vegetable and fruit diet, which has the advantage of being bulky, so that the stomach is filled after a meal, with the result that hunger pangs are stopped. At the same time, the calorie value of the diet is low, so that the body is obliged to draw on its reserves of fat.

Another important point is to encourage the patient to take regular exercise, with the proviso that he should immediately stop with the onset of pain or if he becomes unduly short of breath. Oertel's method consists of systematic hill climbing combined with a diet in which fluids and fat-forming elements are reduced to a minimum.

The system of treatment initiated by Schott and others at Nauheim, but supplied also at various British spas, is probably more suitable to fatty overgrowth of the heart than it is to severe valvular diseases or myocardial degeneration. It consists partly of immersion in saline baths, partly of regulated exercises of the muscles of the arms, trunk, and legs conducted slowly against resistance on the part of an attendant. The different springs of Nauheim have a temperature of from 60° to 95° F., and contain free carbon dioxide, in addition to the salines, of which the most abundant are sodium chloride and calcium chloride and bicarbonate. An important influence is attributed to the stimulating action upon the skin of the saline ingredients, and the minute particles of carbon dioxide gas. An elaborate series of movements has been devised, comprising flexion and extension at all the large joints, these movements being made by the patient, but gently opposed throughout by the attendant. No movement is repeated at the same sitting, an interval of rest occurs between any two successive movements, and indications of strain or dyspnœa or distress are the signals for stopping the exercises. The results of this combined treatment are stated to be slowing and lessened irregularity of the pulse, great diminution of the præcordial dulness, and general improvement in the comfort of the patient.

It is allowed that the waters of Nauheim may be successfully imitated by the addition to other waters of equivalent proportions of sodium chloride (1 to 3 per cent.) or calcium chloride (0·2 to 0·5 per cent.), or sufficient quantities of bicarbonate of sodium and hydrochloric acid to cause effervescence.

## NEW GROWTHS AND PARASITES

Under this head we may shortly mention *tubercle*, *cancer*, *syphilis*, *hydatids*, and *cysticerci*.

**Tubercle.**—Tubercles not infrequently form in connection with inflammation of the pericardium, when they are found as whitish-grey or yellowish granulations, mostly in the substance of the pericardial lymph or false membrane uniting the layers of the cavity, or sometimes actually under the layer of the visceral pericardium. They occur in the course of general tuberculosis, and there may be secondary advanced tuberculosis elsewhere. The diagnosis can only be made from the appearance of pericarditis under those circumstances; but it must be remembered that a pericarditis, which is not tubercular, may also arise in the course of phthisis. Isolated deposits of tubercle are exceedingly rare.

**Neoplasm.**—This attacks the heart in different forms, chiefly as lymphoma, sarcoma, and melanotic carcinoma. Frequently the heart is affected as a consequence of sarcoma or lymphoma of the mediastinal glands; the tumour then spreads along the veins, invades the auricles, and appears as nodular elevations under the pericardium. Sometimes the tumour is secondary to a similar growth in another part of the body. A primary isolated deposit in the heart is exceedingly rare. In one case, where the tumour had invaded and partially obliterated the right ventricle and pulmonary opening, there was continuous intense cyanosis, with no increase in red cells, so that polycythæmia

rubra was excluded, and there was also no evidence of sulphæmo-globinæmia, but often there are no symptoms that are distinctive of the condition. It could only be inferred, in certain cases, from the existence of intra-thoracic growth. Walshe records a case where the conversion of the anterior wall of the right auricle into cancer was unattended with any clinical evidence pointing to the heart.

**Syphilis.** The lesions of this disease occur as arteritis, as fibrous scars (*syphilitic myocarditis*), as fibroid masses, or as distinct gummas, which may be cheesy, and even softening in the centre, affecting the muscular substance of the heart in the same way as the voluntary muscles and causing some surrounding inflammation. The gummas are seated chiefly in the walls of the ventricles. They produce no characteristic symptoms apart from those considered under the heading of Myocardial Degeneration. Sometimes the A.-V. bundle is affected, leading to Adams-Stokes' syndrome.

**Parasites.**– *Hydatid cysts* occasionally develop in the substance of the heart, and project in the course of their growth either towards the pericardium, or into one of the cavities. The cysts are single, or may contain daughter-vesicles. Their effect upon the heart depends, of course, upon the size to which they grow.

The *cysticercus* of the tænia solium is also sometimes found in the walls of the heart.

## ENDOCARDITIS

Endocarditis, like so many other inflammatory processes, is probably always due to the action of micro-organisms or their toxins. As a rule the parts first affected are the valves of the left side of the heart: the lesion is often confined to them, and may completely subside, or if any traces are left they consist of structural damage to the valve of which the later consequences are solely dependent on the mechanical failure of the valve. This is a simple *acute endocarditis*. In other cases, more extensive changes take place in the valves, micro-organisms are present in great numbers, and being conveyed by the current of blood to remote parts of the body, set up fresh foci of disease—*malignant endocarditis*. The term *chronic endocarditis* is applied to the permanent deformities and changes in the valves which result from a simple acute endocarditis, as well as to a separate inflammatory process of slow development.

### ACUTE ENDOCARDITIS

**Ætiology.**—Endocarditis is, in the great majority of cases, an infection due to the virus of acute rheumatism (*see* p. 55), occurring also during the progress of chorea. Myocarditis is usually associated with it. It also occurs in scarlet fever, diphtheria, typhoid, and other infectious diseases. It may occasionally occur during the progress of Bright's disease, syphilis and other chronic maladies. It may occur after local injuries, such as the rupture of a sigmoid valve, or of the chordæ tendineæ, and as the result of unnatural friction of one part of the heart with another. The passage of currents of blood through abnormal apertures may cause the local inflammation of the endocardium. In patients dying from any infective disease it is very common to see minute vegetations on the valves, particularly the aortic valves, indicating a terminal infection of the blood stream.

It is always in limited patches, and never affects the whole interior of the heart. In rheumatic fever and other general diseases it affects the valves early and often alone.

The relation of endocarditis to the two sides of the heart is of very great importance. If endocarditis occurs during fœtal life, it is believed to attack the pulmonary or tricuspid valves; but it is extremely rare for endocarditis

acquired during life to attack the valves on the right side of the heart *only*. It occurred only once in 20,000 post-mortem examinations at Guy's Hospital. On the other hand, right-sided endocarditis associated with left-sided endocarditis is not so very uncommon (**12**). The commonest occurrence is for the valves on the *left* side to be affected alone. Hence what follows in this chapter chiefly concerns the aortic and mitral valves.

**Anatomical Changes.**—The earliest change is a very slight swelling of the subendocardial tissue near the edge of the valve, on the auricular side of a mitral cusp, or the ventricular side of an aortic cusp, so as to form a number of beadlike elevations, usually described as *vegetations;* and these occupy at first the line where the valve touches its fellow on closure, and not the free edge of the valve. The swelling results from œdema and infiltration with lymphocytes, the consequence of rheumatism. In the inflamed valves are nodules containing large multinuclear cells. Later the endothelium of the valves over the inflamed area becomes loosened, and fibrin and polymorphonuclear leucocytes are deposited from the ventricular blood. By the continued addition of deposits of fibrin, very large vegetations may be formed which project into the valvular orifice, and from which particles, loosened by softening, may be detached by the current of blood. Such a detached particle is carried by the blood to distant vessels of gradually diminishing size, and ultimately meeting with one small enough to resist its further progress, becomes impacted therein. This process, known as *embolism*, is an important element in malignant endocarditis. When subsidence of the inflammation occurs complete resolution probably sometimes takes place; there is, however, more often some formation of vessels, organisation of the infiltration, and growth of fibrous tissue, by the gradual and irregular contraction of which—a process similar to that which occurs after inflammation in other parts—the valves become shortened, deformed, and incapable of completely covering the orifice they are intended to close. In some cases the fibrous tissue acquires an almost cartilaginous hardness, or calcareous particles are deposited, and the valve segments not only fail to close the valve aperture, but, by their constant projection into the orifice, offer a definite obstruction to the passage of the blood through it.

In acute stages, streptococci, staphylococci, pneumococci, and diplococci have been found; but they are absent from the chronic lesions. It is possible that in rheumatic cases acute endocarditis is due to streptococci growing in pairs (Poynton and Paine); but this is not generally accepted at the present time.

**Symptoms.**—Endocarditis, as it occurs in rheumatic fever, has but few symptoms, and these may be due to associated myocarditis. Indeed, it mostly proceeds without any appreciable alteration of those which are due to the rheumatism, except that persistent tachycardia is a fairly common occurrence. Acute heart block also occurs, and in some cases a reduplicated first or second sound or a diastolic murmur heard at the apex may be due to the auricles contracting perceptibly before the ventricles in this condition, but all these physical signs may also be due to the rush of blood through the mitral valve as in mitral stenosis. An early indication of some cardiac change is a slight prolongation, or roughness, or some want of clearness of the first sound in the aortic or mitral area. Within twenty-four hours it may lengthen into a distinct murmur, or soft blowing sound, which accompanies, and does not abolish, the first sound. If the aortic valve is affected, the second sound may become imperfect, and a diastolic murmur may become developed, but this is much less frequent, while the systolic murmur in the mitral area is the most common of all. This physical sign is heard in somewhat less than half of all cases of rheumatic fever, and mostly within seven days of the onset. It is probably due to dilatation of the mitral valve from myocarditis, but this is usually accompanied by vegetations on the valve, and in any case it is possible that these in themselves may cause regurgitation by making the valves less flexible

than usual. Its duration is variable; it may disappear entirely in the course of the rheumatic attack; or it may become louder and harsher, more widely diffused, or definitely follow the course of the blood current, showing the existence of valvular incompetence.

**Diagnosis.**—This requires some care, as the murmurs of recent acute endocarditis and myocarditis may be confounded with the murmurs of old *valvular disease*, and with *pericardial* friction sounds. The chief point to note is that the murmur in question is generally soft in quality, systolic in time, and strictly limited to the area of the valve affected—that is, either the aortic or the mitral area. Acute simple endocarditis of the pulmonary or of the tricuspid valve is practically out of the question. A functional or hæmic murmur is generally loudest over the pulmonary artery, and often harsh in quality. The murmur of chronic valvular disease is often loud or harsh, heard over a large area, and accompanied by some alteration in the size or shape of the heart.

**Prognosis.**—In the course of an attack of rheumatic fever, or of any other illness causing acute endocarditis, there is nothing to guide one as to the outcome of the disease. In a large number of cases the murmur disappears, and the patient apparently recovers completely; in a certain proportion of these, nevertheless, valvular disease is found several years afterwards, showing that the valves were really affected all the time. In a few cases the affected valve soon becomes incompetent, or stenosis may come on gradually.

**Treatment.**—The main essential of treatment in endocarditis and myocarditis is to keep the patient at rest in bed until all active inflammation of the heart has subsided. It is difficult to be certain when this is actually the case, so that patients, particularly if they are children, may be kept in bed many weeks to be on the safe side. In any case the patient should not be allowed to get up till the pulse and temperature have fallen to normal and have remained so for some days. Another useful indication is the character of the murmurs. As long as these show any change from day to day it means that there is active inflammation present. Possible foci of infection should be looked for and dealt with (*see* p. 59).

Salicylates have been prescribed in large doses with the idea that they act specifically in rheumatic heart disease. Almost certainly this is not the case, and there is the disadvantage in this treatment that the temperature may be artificially lowered so that a valuable sign of active disease is abolished.

## MALIGNANT ENDOCARDITIS

(*Infective, Ulcerative or Bacterial Endocarditis*)

**Ætiology.**—Acute rheumatism is an antecedent of malignant endocarditis, but the proportion of cases (fifty-three out of 160—Osler) is less than that in which rheumatism is related to simple endocarditis; in some of these the symptoms have developed in the course of the rheumatic fever, and in others they have arisen in the stage of chronic valvular disease, which predisposes to the occurrence of infection. On the other hand, malignant endocarditis may occur even though the valves are healthy, especially after exposure and hard muscular work. This has been verified by war experience (**13**). Besides rheumatism, its predisposing cause may be found in acute pneumonia, in the eruptive fevers such as scarlatina, in puerperal processes, in the existence of open wounds on the surface of the body, in septicæmia and pyæmia, in malaria, and in some other conditions. Purulent discharges from the mucous membranes (urethritis, vaginitis, pyorrhœa alveolaris) may give rise to the disease, but the latter is more likely to arise when for some reason the pus is held back, and there is no free drainage. This is particularly likely to occur in the chronic tonsillitis of children, or dental abscesses in the case of adults. Various micro-organisms are found in the organs in infective endocarditis. Streptococci, similar to those found in the mouth or

large intestine, are most common; staphylococci, meningococci, pneumococci, the *Bacillus pneumoniæ* of Friedländer and the bacilli of tubercle, diphtheria, and typhoid and the gonococcus and anaerobic bacilli are found occasionally. From some centre of infection the organisms find an entrance into the blood and are thence deposited on the valves. Streptococci are often found in the blood during life in the acuter forms of the disease.

**Anatomical Changes.**—In this form of endocarditis the tissue of the inflamed valve is softened, and breaks down, so that erosions or ulcerations take place, and, as a result of this, fibrin is deposited upon the roughened surface, and accumulates into irregular masses of vegetations, which may reach the size of a hazel nut. By suitable methods the micro-organisms can be demonstrated on the surface, and more or less deeply in the substance of the vegetations and fibrinous deposits, where they form considerable masses in acute cases, while beneath the infected surface there is a zone of polymorphonuclear infiltration; when the disease is chronic the micro-organisms are far fewer and processes of repair by means of fibro-blasts are more obvious. Several important changes result from these processes in the valve. The valve itself may be perforated, or strips of tissue may be partly separated and hang loosely in the blood current, or portions may be completely detached. Sometimes a part of the valve is so weakened by the destructive process that it yields before the pressure of the blood, and a saccular dilatation, or *aneurysm*, of the valve is formed, projecting on the opposite side. Another result is the occurrence of endocarditis, or endarteritis, in adjacent parts from a strip of the valve playing backwards and forwards in the blood currents with the systole and diastole of the ventricle, and striking alternately the walls of the cavities in front and behind. In the case of mitral endocarditis, these are the left ventricle and the left auricle; in the case of aortic endocarditis, they are the aorta and the left ventricle. At the spot struck infection takes place, and causes a fresh patch of inflammation of the lining membrane.

But the most important effect of malignant endocarditis is the infection of the whole arterial system by particles detached from the valves being carried to remote parts; and it is to this process, combined with the presence of organisms in the detached fragments, that the special features of this disease are due. Embolism may take place in almost any part of the body. It is especially common in the vessels of the spleen and kidneys, but it happens also in the vessels of the brain, alimentary canal, skin, retina, and lungs, and the larger arteries supplying the limbs, such as the radial, ulnar, tibial, brachial, and others. The local results of these impactions are: (1) Obstruction of the circulation; (2) necrosis or hæmorrhage, or both, within the area of distribution of the obstructed vessel, and the formation of infarcts; and (3) suppuration in the same area from the septic influence of the micro-organisms, when the process is acute (*see* Embolism).

The effects upon the various organs, as they may be seen in different cases of malignant endocarditis, are—softening and abscess of the brain, and meningitis; retinal hæmorrhages and optic neuritis; diffused swelling, infarction, and abscess of the spleen. There are two ways in which the kidneys may be affected: there may be acute nephritis presenting typical histological features; but there may be an acute embolic nephritis giving rise to *flea-bitten kidneys*, which are often large and finely mottled or speckled with hæmorrhagic points upon a white ground. Microscopically collections of small white cells due to infective emboli may be seen in the glomerular tuft. The kidneys may show infarcts. There may also be hæmorrhages under the skin, hæmorrhagic infarctions and abscesses of the lungs, pleurisy and empyema.

Malignant endocarditis, like simple endocarditis, affects chiefly the left side of the heart; but the proportion of cases in which the right side is involved is much larger than in the simple form. In the majority of cases malignant endocarditis occurs on valves showing the effects of previous simple endocarditis.

**Symptoms.**—The symptoms and course of the disease present the greatest variety. In some cases the symptoms at first are simply the occurrence of fever with afternoon rises of temperature, or perhaps sweating, in a patient living an active life, though perhaps known to have valvular disease, more or less perfectly compensated. The temperature may be high, reaching 102° or 103°; but it is generally remittent or intermittent, and sometimes with remarkable regularity for long periods. There is often free perspiration, and there may be an occasional rigor. The pulse is rapid, ranging from ninety to 120, or even higher. If the heart be auscultated, a murmur will generally be heard at one or other orifice, mostly, however, on the left side. Still it must not be forgotten that in these cases murmurs may be entirely absent. In the cases with a previous history of rheumatism, or known cardiac disease, there may be abnormalities in the size and action of the heart. The respirations are rapid, sometimes without definite lesions of the lungs, at others with signs of bronchitis, œdema, or congestion.

In a considerable number of cases there is a close resemblance to *typhoid fever*, chiefly on account of the almost spontaneous occurrence of fever, with headache and perhaps enlarged spleen. Thus, the patient may have been perfectly well until he complains of some such symptoms as usher in other severe febrile diseases, pain in the head, or in the back and limbs, or a definite rigor or rigors. Then follow severe pyrexia, with its usual conditions—high temperature, quick pulse and respiration, dry tongue, loss of appetite, thirst and malaise. Frequently, within a few days, the patient is prostrate, apathetic, drowsy, and at night delirious; but the time of appearance of this symptom, determined presumably by the virulence of the toxin, is variable. The condition of the bowels varies, but there are often loose yellow motions, with much resemblance to those of typhoid fever; and the abdomen may be distended. The spleen is enlarged either from the general infection or from embolism; but there are no rose spots. The duration of these cases is generally from ten days to two or three weeks, that is, much less than in cases of chronic infective endocarditis

In another group of cases rigors are a prominent symptom, occurring once, twice, or more times in the day, and the resemblance to *pyæmia* from wounds is very close. Rheumatism and actual cardiac disease are less often present as antecedents, and the endocarditis more often affects the right side of the heart than in the typhoid form. It may begin, like the last, with vague or more decided pains in the limbs or back, until the first rigor occurs; and intense anæmia is characteristically present. The heart may present but little or no evidence of enlargement, and the murmur may be limited to the area of the pulmonary artery, in which case it may be thought that the murmur is simply hæmic, as a consequence of the pronounced anæmia; on the other hand, in many cases the same conditions of the cardiac apparatus may exist as in the other forms—namely, the murmurs of mitral or of aortic disease. For the rest, the local conditions are not distinctive. The spleen may be enlarged, diarrhœa with loose yellow motions often occurs, and albuminuria may be present. Sometimes the joints inflame or suppurate. The temperature rises to a great height, 105° or 106°, in the rigors, and it may fall to normal or subnormal in the intervals. Sweating occurs, and is often profuse. As the case continues emaciation becomes more marked, and with increasing delirium and apathy or coma death results. These cases are often of short duration.

In again another group the organisms invade the cerebral meninges, and the symptoms of meningitis form the prominent feature. This often occurs in association with pneumonia, and the case is a combined pneumococcal meningitis and pneumococcal endocarditis. The cerebral symptoms consist of headache, convulsions, and coma, and death may take place quickly after their onset. The meningitis is often basal, and the exudation consists of greenish lymph or pus, containing pneumococci. Marked cerebral symptoms, with a finely petechial eruption, may cause the case for a time to resemble typhus.

The symptoms of a general infection with micro-organisms from the cardiac valves may arise at any period in the history of a case of cardiac valvular disease. In chronic sufferers, confined to bed by dropsy or other results of cardiac failure, the change to a malignant type of endocarditis is indicated by remittent pyrexia, and by the occurrence of embolism in different parts of the body; but the cardiac symptoms may continue predominant, and thus this group of cases may be distinguished from those first described.

In any case of the disease, to the symptoms dependent upon septicæmic infection may be added those due to the obstruction by embolism of arteries or arterioles. Sometimes embolism of a large vessel in the brain will occur, and cause hemiplegia; if a vessel in the leg or arm is obstructed, there will be loss of the pulse at the wrist or ankle; but neither gangrene nor coldness need occur, unless a very large vessel is involved. More frequent are embolisms of the small vessels in the viscera; there is thus often enlargement and tenderness of the spleen, due in part to infarcts, and the spleen may weigh from 20 to 30 ounces. Infarcts also occur in the kidney, accompanied, it may be, with pain, and the appearance of albumin or blood in the urine. In some cases petechial hæmorrhages appear under the skin, the petechiæ being generally small, and situate on the trunk, about the groins and axillæ; exceptionally a *purpuric* condition may be present for months. Sometimes small painful erythematous swellings appear on the skin, particularly on the flexor surfaces of the fingers, last a few days and disappear again; or there is deep-seated pain, and a larger tender lump can be felt beneath the skin in the deeper tissues of the limbs or body, lasting a few days. These are known as *Osler's spots*, and are especially diagnostic in the subacute disease. These are presumably embolic events. Hæmorrhages are often seen in the retina, and there may be also hæmoptysis or epistaxis. Some inflammatory conditions are probably also referable to embolic processes, such as the form of nephritis already mentioned (*see* p. 331), though whether as the result of vascular obstruction or of the introduction of micro-organisms may be doubtful; albuminuria from nephritis or infarct is frequent. Optic neuritis is sometimes present.

There is a particular group of more or less chronic cases known as *subacute bacterial endocarditis* or *endocarditis lenta* (**13, 16, 17**). The disease arises insidiously. The patient becomes progressively pale and anæmic, suffers from lassitude, and there are often slight rises of temperature, often not above 100° F. Clubbing of the fingers is characteristic and there is enlargement of the spleen. Petechial and other embolic symptoms are found occasionally, *e.g.* there may be slight albuminuria, and centrifugalisation of the urine may disclose the presence of blood cells. Osler's spots are specially characteristic of this form of the disease.

The duration of malignant endocarditis is very variable. Some cases last six or seven months with little else than a constant pyrexia; cases of a pyæmic or severe typhoid type, and those with meningitis, are often fatal in a few weeks or days. Death occurs commonly from cardiac failure or uræmia, or embolism.

**Diagnosis.**—This rests upon the pyrexia of remittent or septic type, the existence of valvular disease, and the evidences of embolism above enumerated; marked anæmia and optic neuritis, if present, are also valuable signs. The heart should be examined in all cases of pyrexia of uncertain origin. But a murmur may be wanting throughout the whole illness; and even if present the valvular disease does not exclude the possibility of influenza, typhoid, or tuberculosis. Thus the diagnosis may often have to depend upon the occurrence of embolisms. Malignant endocarditis is frequently mistaken for *typhoid fever*. Both are acute severe febrile diseases, and in both there may be a marked absence of localising symptoms; on the other hand, there is often in malignant endocarditis swelling of the abdomen, with frequent loose yellow motions; and enlargement of the spleen may further increase the resemblance. The following are points of

difference: In malignant endocarditis there is a more uniformly remittent or intermittent course of temperature, which may last several weeks; rigors, petechiæ under the skin, much pallor of the face, optic neuritis, or retinal hæmorrhages, if present, are in favour of endocarditis.

In some of the subacute cases the spleen may be so enlarged, and the anæmia so considerable, that *splenic anæmia* is simulated; this is all the more likely if there are petechiæ or hæmorrhages from the mucous membranes and if the murmur is not unmistakably organic.

In the present day the occurrence of rigors should make one think as readily of malignant endocarditis as of the relatively infrequent *pyæmia*. In the latter, as a rule, the patient has more frequent rigors, an earthy colour of the face, and the evidence of inflammation at the base of the lung; in the former rigors are less constant, and may cease to occur long before the end of the case. But a wound may be the cause of septic endocarditis; and the diagnosis is further complicated by the fact that the endocarditis may itself be only a part of a typical pyæmia. *Malaria* may also be suggested by septic endocarditis. A continued pyrexia without obvious signs may be due to commencing *miliary tuberculosis* as well as malignant endocarditis; but after a time local signs peculiar to one or the other ought to be observed.

One should always be alive to the possibility of septic conditions arising in the course of old heart disease, whether the patient be active or bedridden. Marked anæmia, continued pyrexia, rigors, prostration out of proportion to the valvular lesion, and the evidences of embolism in kidneys, spleen, or brain, are the facts that should guide the physician.

The blood should be examined for streptococci and other organisms.

**Prognosis.**—This is exceedingly bad, and the recovery of a well-marked case of either typhoid or pyæmic form is rare. On the other hand, attacks of subacute bacterial endocarditis have subsided, but they may occur again after a varying interval.

**Treatment** can obviously be little more than palliative in the majority of cases. As in pyæmia, if there is any wound or sore, it should be rendered aseptic, and an attempt may be made to influence the disease by frequent doses of quinine (5 grains), sodium sulphocarbolate (10 to 20 grains), or sodium benzoate (20 grains). In a few cases good results have followed the subcutaneous injection of an antistreptococcal serum, or of an autogenous vaccine. But it is sometimes impossible to find any micro-organisms in the blood; and even when they are found, and a vaccine can be prepared, it is with rare exceptions useless. A more promising line of treatment when an organism has been isolated is to inoculate a relative and carry out an immuno-transfusion from the relative to the patient (*see* p. 546). The general rules for nursing and dieting in febrile states are applicable. Profuse diarrhœa may be checked, if required, by astringents. The delirium is rarely so violent as to require any special treatment. Stimulants are naturally given, as the heart's action early tends to be seriously affected.

## CHRONIC ENDOCARDITIS AND DISEASES OF THE VALVES

Little is known *clinically* of the course of chronic inflammation of the cardiac valves. What is called chronic inflammation is the thickening, shrinking, or deformity of the valve which is found after death, and which has been often preceded years before by rheumatic fever with or without accompanying evidence of acute endocarditis. An acute attack of endocarditis may cause deformity in the valves during the process of healing. The deformity may become worse as the result of later acute attacks. On the other hand, there may be from the outset

a slow smouldering infection on the valves, with few physical signs, lasting for months or years, which produces deformity in the valves.

As seen in the aortic valves, the changes consist in thickening of the base of the cusps, and to a less extent of the free edge, with shortening of the radial measurements, so that it is obvious that the cusps cannot meet to cover the orifice. Since the three cusps are discrete, chronic inflammation causes them to shrink independently of one another. There may be some thickening at the three points on the wall of the aorta where the valve segments meet, and they are commonly slightly adherent at these points, but not enough to cause stenosis of the opening. It is decidedly uncommon for the valves to be so fused together and at the same time thickened and retracted as both to present an obstruction to the flow of blood and to prevent complete closure—a great contrast to the case of the mitral valve. Exceptionally the fusion is so complete as to allow only a small opening for the passage of blood into the aorta, and yet the valve closes well.

The mitral valve is liable to similar inflammatory changes, but the results are quite different owing to its structure. It is really a misnomer to speak of the two cusps of the mitral valve. The latter really consists of a fringe or curtain surrounding the opening, which is more developed on one side to form the aortic cusp of the mitral; but the so-called marginal cusp hardly exists as a separate structure. Inflammation leads to thickening of the valve and (1) to shortening from above downwards, so that complete adaptation on closure is lost and there is regurgitation. But there is also (2) shortening of the valve circumferentially, leading to stenosis, and the longer the inflammation lasts the greater the stenosis. These two types of shortening often co-exist, producing a double lesion. Slight inflammation will not cause enough circumferential shortening to cause clinical stenosis, though there may be regurgitation. Severe inflammation may lead to regurgitation alone, stenosis alone, or more commonly to the two combined. The presence of stenosis always means a severe infection of the valve. Post-mortem examination also shows that in mitral disease the cordæ tendineæ are thickened and shortened—so shortened, indeed, that the valve curtain is continuous with the musculi papillares, and these themselves are invaded by the fibrotic change. In some cases of stenosis there is circumferential shortening without much shortening from above downwards; in other cases both types of shortening exist, so that only a narrow slit presents itself on the auricular side in the dense surface of the valve. A distinction is thus drawn between *funnel-shaped* and *buttonhole* orifices; and the former appears to be very much more frequent in children (8 to 1, Allbutt), the latter in adults (25 to 1).

The pulmonary valve sometimes presents changes suggestive of chronic endocarditis, rarely as a result of acquired disease, nearly always as a congenital lesion. It is very doubtful whether this is a truly inflammatory lesion (*see* Congenital Malformations). In the tricuspid valve lesions of the same nature as those of the mitral valve are seen, but they are much less extensive as a rule, and do not frequently cause an obstruction like that which is so common on the left side of the heart.

It will be understood that, as a result of the above structural changes, the efficiency of the valves must be seriously impaired, and the circulation of the blood through the heart must be to a greater or less extent affected. This takes place in two ways: first, the thickening of the valve, or vegetations or fibrinous masses upon it, or the union of two or more segments of a valve together, materially narrows or constricts the orifice through which the blood passes from auricle to ventricle, or from ventricle to aorta; secondly, the contractions or deformities of the valve so shorten them or diminish their area as to render them incapable of closing the orifice, and preventing reflux of blood from ventricle to auricle, or from aorta to ventricle. The one case is called *obstruction* or *stenosis*, the other *regurgitation* or *incompetence*. These two conditions may occur singly

or combined at any one of the four orifices of the heart; but they are much more frequent on the left side of the heart than the right, because endocarditis, which is the chief cause of the valvular deformities, rarely attacks the right heart.

In connection with the effect of lesions attributable to chronic endocarditis upon the circulation, it is desirable to point out that changes in the valves and neighbouring structures arising from causes other than inflammation may equally interfere with the proper action of the valves, and equally disturb the circulation and lead to all the secondary lesions which will in due course be described. Thus at the aortic orifice ruptures of the segments of the valves from strain and injury will lead forthwith to regurgitation, while secondary endocarditis and deposits of fibrin upon the injured valve will cause obstruction in addition. Atheroma and syphilitic endarteritis are fertile causes of lesions, not only in the aortic valves, but in the aortic wall adjacent, by which the functions of the valves are prejudiced, chiefly in the direction of incompetence. These operate to a less degree in the case of the mitral valve. Another very frequent cause of regurgitation through an orifice is dilatation of the valvular ring or orifice from loss of tone in the muscles of the adjacent cardiac cavity, while the valve cusps themselves are healthy; and this loss of tone may be the effect of myocarditis or other change in the myocardium weakening the muscular structure, or of an abnormally high tension of the blood in the cavity.

**Relative Frequency of Valvular Lesions.**—Disease of the mitral valve is more common than that of the aortic valve. From clinical evidence, regurgitation by itself is the most frequent event at the mitral orifice, a combination of obstruction and regurgitation next in frequency, and pure obstruction least frequent. However, the majority of cases of mitral disease of whatever kind coming to autopsy are found to have stenosis. This is due to the fact that stenosis is the natural result of prolonged inflammation of the mitral valve. At the aortic orifice, regurgitation is very much commoner than obstruction. The significance of the to-and-fro aortic murmur is dealt with on p. 340. Mitral regurgitation is often the result of, and then accompanies, aortic disease. On the right side of the heart tricuspid regurgitation is the only form that is at all frequent, and it is mostly secondary to mitral disease, or to chronic pulmonary disease, such as emphysema or bronchiectasis. It may also follow mitral disease, when this is the result of aortic disease.

**Compensation.**—The actual changes that occur in the heart depend on the nature of the valvular lesion, and will be considered in detail with the different lesions. The presence of a valvular defect entails more work on the part of the heart, and the latter responds by hypertrophy of its muscle. The hypertrophy has been divided into two types. The so-called *concentric hypertrophy* means that the muscle is hypertrophied, but there is no dilatation of the corresponding cavity. In *excentric hypertrophy* there is a preliminary dilatation and a secondary hypertrophy. The type of valve determines which of these two kinds takes place. Thus, aortic stenosis leads to concentric hypertrophy and aortic regurgitation to excentric hypertrophy of the left ventricle.

In the case of mild valvular lesions the heart will hypertrophy to such an extent that, whatever work it is called upon to perform, its response will be as effective as in a normal person. The patient will not become more than usually short of breath after the severest forms of muscular exercise, and the breathlessness will disappear as quickly as usual after the exercise is finished. Such a lesion may be said to be *fully compensated*.

If the valvular lesion is of a higher grade, the patient may be comfortable when at rest or during mild exercise, but may become more than normally short of breath when the exercise is more severe, and this breathlessness may persist for some time. In this case the heart is compensated for mild exercise, but is uncompensated for severe exercise. The term *partial* or *incomplete* compensation may be used for such a case.

In still more severe cases, where the heart is failing, the patient exhibits breathlessness and perhaps other symptoms, even when lying at rest in bed. In this case complete failure of compensation has occurred, and the various chambers of the heart become dilated to a greater extent than they were previous to the failure of compensation.

Compensatory dilatation and hypertrophy affect primarily the particular chamber of the heart with which the affected valves are in relation. However, when failure of compensation occurs the other chambers of the heart become involved. For instance, if the left ventricle dilates, the muscular ring from which the mitral valve is suspended shares in the dilatation. This causes mitral regurgitation. This leads to engorgement of the left auricle and of the lungs with rise of the pulmonary blood pressure. To overcome this resistance the right ventricle hypertrophies. If dilatation of this chamber occurs, it leads to tricuspid regurgitation with engorgement of the abdominal viscera. Valvular disease is associated with fatty and fibroid degeneration of the myocardium. This degeneration may be due to the same cause as produced the original valvular disease, *e.g.* rheumatic fever, syphilis, etc., or it may be the direct result of lack of nutrition of the heart muscle.

There is a tendency at the present day to consider the state of the myocardium as the all-important factor in determining the onset of cardiac failure in valvular disease, and it is probably correct that although valvular disease unaccompanied by myocardial changes may cause symptoms of breathlessness and exhaustion after exercise, indicating relative failure of compensation, complete failure of compensation will be hardly likely to occur if the condition of the myocardium is sound. At the same time the heart should be regarded as a whole. It is really impossible to separate the effect of valvular disease from that of myocardial changes, if both are present together.

**Effects of Failure of Compensation upon other Organs.**—The secondary remote effects of valvular disease on other organs, produced as they are by defects of the circulation of the blood, are numerous and widespread. They are most manifest in the subcutaneous tissues, which become œdematous, and in the lungs, liver, kidneys, spleen, and gastro-intestinal mucous membranes. The most frequent changes are *venous congestion* and *œdema*.

*Anasarca.*—A general œdema of the subcutaneous tissues is called anasarca. In heart disease it first becomes apparent in the feet and ankles when the patient is walking about, and gradually extends up the legs. But even when it is very extensive it is generally confined to the lower extremities and lower half of the trunk, leaving the face, chest, arms, and hands of their normal size. Bolton's recent work has shown that the œdema fluid, which is primarily due to lack of nutrition of the capillary walls, owing to stagnation of the blood stream, is actually originated in the neighbourhood of the heart, and is increased by a general plethora in the circulation with raised capillary pressure. On the other hand, there is increased absorption of œdema fluid in the legs. We must imagine that a regular circulation of œdema fluid occurs—exudation from the visceral peritoneum near the heart, filtration downwards through the cellular tissues under gravity, and absorption into the circulation in the lower limbs. It is purely due to gravity that it first becomes visible round the ankles, since there is no exudation actually occurring there.

*Lungs.*—The delay in the circulation which results from impaired action of the valves on the left side of the heart first affects the lungs: the blood in the pulmonary veins flows with difficulty into the left auricle, and more or less stagnation of the blood in the capillaries takes place. In early stages there is simply undue fulness of the venous radicles in the lung; there is often, in addition, a transudation of serum into the air vesicles and minute bronchial tubes, so that on section of the lung a quantity of yellowish or almost colourless frothy liquid flows from it; and in advanced cases the most affected parts of the lung become

solid, tough, airless, dull brown in colour, from the presence of hæmatogenous pigment, and uniformly smooth. This condition has been called *heart-lung*, or *brown induration*. Both induration and ordinary œdema especially affect the bases of the lower lobes. As a result of local interference with the circulation, some transudation of fluid into the pleural cavity (hydrothorax) often occurs, and there is more or less proneness to inflammatory lesions of the lung, either in the form of bronchitis, pneumonia, or pleurisy. None of these effects is necessarily bilateral: pleural effusions are often on one side only.

*Liver.*—The hepatic veins open into the inferior vena cava so close to the right auricle that the influence of cardiac disease upon the circulation of the liver can be readily understood. The organ enlarges considerably, and becomes darker in colour, and in advanced conditions acquires a peculiar appearance of red, yellow, and white mottling, to which the name of *nutmeg liver* has been applied. On section the centre of each lobule is seen to be occupied by the enlarged hepatic vein rootlet transversely divided, and the adjacent central zone of the lobule is dark red or purple; outside this is a zone of yellow colour from the retention of bile within it; while the external zone of the lobule is of white or grey colour which the microscope shows to consist of cells in a state of advanced fatty degeneration.

*Kidneys.*—These are, as a rule, only congested, becoming in consequence larger and dark-coloured; but from long-continued congestion a certain amount of fibrous tissue may develop, and by its irregular distribution and contraction it may produce a granular condition of the surface.

*Other Abdominal Organs.*—The *spleen* becomes hard and darker than normal, and, though varying in size, is often small. The congestion of the *stomach* and *intestine*, like that of the spleen, is, of course, secondary to that of the liver, since the veins derived from these organs empty themselves into the portal vein. The mucous membrane becomes congested, and after death considerable distension of the vessels, and sometimes hæmorrhages into the substance of the mucous membrane, may be seen. *Ascites*, or dropsy of the peritoneal cavity, is another result of the obstruction to the circulation in the radicles of the portal vein.

**Complications.**—Every result of simple venous stagnation must be considered a part of heart disease, such as brown induration of the lung and albuminuria. But pleurisy, pneumonia, embolism, and malignant endocarditis may be regarded rather as complications. With a failing circulation fibrin may be deposited upon the valves and in recesses of the dilated cardiac cavities. Hence detached particles may be carried into the cerebral vessels, causing hemiplegia; into the splenic or renal vessels, producing the characteristic infarcts, or more rarely into the vessels of the limbs, abolishing the pulse or leading to gangrene. Malignant endocarditis is frequently grafted upon chronic disease.

## AORTIC DISEASE

There are two main causes of aortic valve disease. In the first place, there is acute rheumatism. Here disease of the aortic valves is usually associated with disease of the mitral valves; it is quite uncommon for the aortic valves to be affected alone. In the second place, there is syphilis. This disease usually produces an aortitis primarily, and the aortic valves become involved in the same process. It is quite common in syphilitic cases for disease of the aortic valves to be unaccompanied by mitral disease. When the valves have been infected in either of these ways, the lesion may be aggravated by frequent and continued strains upon the circulation, especially from the excessive use of the arms, such as arises in blacksmiths, sawyers, and others with laborious occupations. Sudden rupture of the valves also sometimes takes place.

## AORTIC STENOSIS

In Cohnheim's classical experiments on animals a ligature was passed round the aorta and gradually tightened. The lesion was compensated for by the greater power of the muscular contractions of the ventricle, *i.e.* the output of the heart per minute, and the arterial and venous pressures remained the same, but the intra-ventricular pressure was much increased, the heart rate was slowed, while the actual duration of the contraction of the ventricle was increased, in the endeavour of the heart to force its content of blood into the aorta against this artificially increased resistance. It was only when the constriction reached a certain limit that the heart suddenly failed.

Exactly the same conditions are present in aortic stenosis caused by disease, except that here, since the lesion has been produced gradually, the heart has had time to adapt itself. The increased work that falls on the heart causes a primary hypertrophy of the left ventricle. With a stationary lesion this is sufficient by itself to empty the ventricle completely through the narrow opening at each systole. Only when compensation begins to fail is there a dilatation of the ventricle, which spreads to the other chambers of the heart.

In pure *aortic stenosis*, which is an exceptional condition for reasons already explained (*see* p. 335), the murmur is systolic, audible in the second right intercostal space near the sternum, traceable up towards the right clavicle, and audible in the carotid arteries. If stenosis is considerable a thrill may be present, felt at the same spot, and also systolic. The pulse is often characteristic; the obstacle interposed in the current of blood prevents the full effect of the ventricular contraction upon the column of blood in the systemic arteries, and the pulse can be felt to have lost its suddenness and to rise quite slowly. The pulse tracing is then *anacrotic*, that is, the percussion wave is lower than the succeeding tidal wave, and appears as an elevation on the ascending limb. In the extreme variety of this form this wave is rounded off, or entirely absent, and the tracing resembles that shown in Fig. 47, A (p. 375). The pulse is infrequent and sluggish when palpated (*pulsus tardus*). When the aortic stenosis is fully compensated the patient may have no symptoms. In other cases pain in the chest and a feeling of oppression may be noticed. When compensation begins to fail, shortness of breath and signs of venous congestion will be observed.

## AORTIC REGURGITATION

If one of the aortic valves is experimentally damaged in an animal, compensation takes place at once, just as in aortic stenosis. The output of the heart remains unaffected, and the venous pressure remains unaltered. However, the systolic pressure is much increased while the diastolic pressure is much diminished, and the mean between these two pressures remains about the same as before.

It has been found that the normal human heart at rest supplies the arterial system with about 3 litres of blood a minute (or roughly 40 c.c. per beat). This is the amount required for the proper nutrition of the heart itself, the brain and other organs. During exercise as much as 12 litres a minute is passed through the heart. Suppose that during each diastole half of the blood delivered passes back into the left ventricle owing to incompetence of the aortic valves. Then, since the rate of the heart remains the same in compensated lesions, the heart must deliver 80 c.c. through the aortic valves at each beat in order to maintain the supply of 3 litres of blood a minute. This means that the left ventricle must have permanently a capacity of 80 c.c. instead of 40 c.c. Hence in aortic regurgitation there is a primary dilatation of the left ventricle. However, there will also be a secondary hypertrophy of the muscle wall, because the work of the left ventricle will be increased, since it now has to deliver 80 c.c. instead of 40 c.c. of blood through the aortic valves against the pressure in the arteries.

During exercise the heart not only beats quicker, but the volume of blood expelled at each beat is larger. Suppose the output at each beat is doubled, then in regurgitant cases it will be necessary for the heart to expel 160 c.c. at each beat instead of 80 c.c. in the normal case. This temporary dilatation in the pathological condition will probably be much beyond the capacity of the heart, so that it will be unable to supply the requisite amount of blood. This illustrates the loss of reserve power on the part of the heart in lesions which may be perfectly compensated at rest.

**Physical Signs.**—Cases of aortic regurgitation are characterised by the murmur already described (*see* pp. 290, 291). Sometimes the murmur is only audible by means of a wooden stethoscope, or by direct application of the ear to the chest wall. Occasionally it is quite inaudible. An aortic diastolic murmur is very commonly accompanied by a systolic murmur in the aortic area traceable up into the neck, forming a so-called to-and-fro murmur. This does not mean an associated stenosis, unless there are other signs of it, *e.g.* a thrill, small anacrotic or bisferiens pulse, hypertrophied left ventricle, because stiffened or roughened valves may lead to a fluid vein and eddy currents during systole, and so to a murmur, without any narrowing of the sectional area of the orifice. Further, aortic stenosis is incompatible with much regurgitation, because the latter would mean that the aperture was big, which is itself inconsistent with stenosis. A typical murmur of aortic regurgitation is also not uncommonly associated with a diastolic or pre-systolic murmur heard at the apex although the mitral valve is perfectly healthy. This murmur may be accompanied by a thrill. This association was described by Flint in 1862, and the murmur is named after him (*see* also p. 293). In aortic regurgitation the heart is dilated and hypertrophied, and the impulse is carried downwards and slightly outwards.

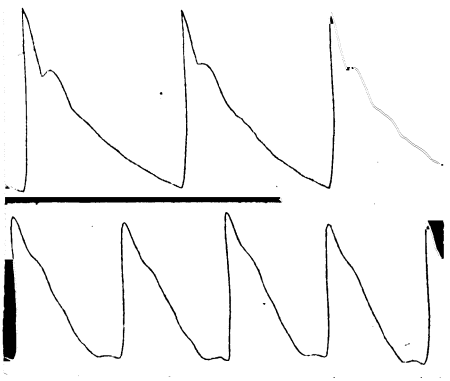


FIG. 46.—A. Pulse of Aortic Regurgitation. Pressure, 3 ounces. B. Pulse of Aortic Regurgitation. Pressure, 4½ ounces.

The systolic pressure as tested by the sphygmomanometer is often raised. The diastolic pressure is, on the other hand, very low. It is, in fact, quite common to hear a loud systolic murmur over the brachial artery with no pressure in the armlet, indicating that the diastolic pressure falls to zero. Whenever in the examination of a patient the diastolic pressure is found to be below 50 mm., this fact is very suggestive of aortic regurgitation. It is also found in aortic regurgitation that the systolic pressure in the femoral artery is higher than in the brachial. The sudden rise of the pulse wave and its equally sudden subsidence yield a peculiar sensation to the finger, which is expressed by the various names given to this form of pulse, such as *kicking*, *splashing*, *water-hammer*, and *shotty*. It is also known as the *pulsus celer*. All over the body the sudden and extensive movements of expansion and contraction in the arteries produce marked effects. The vessels of the neck throb visibly and often painfully, the digital arteries can be felt with unusual distinctness, and the pulsation of the retinal arteries can be easily seen with the ophthalmoscope. The explanation is as follows: The arterial diastolic pressure is low, and against this pressure the hypertrophied heart expels an abnormally large volume of blood at a rapid rate, causing a rapid abnormally high systolic pressure. The pressure rapidly falls because the blood escapes into the periphery more quickly than the heart delivers the last portion

of its contents into the aorta. The sudden rapid fall is not due to the escape of blood back into the heart through the leaky valve (though the low diastolic pressure is due to this), because it occurs *before* the dicrotic notch (*see* Fig. 46, A). A similar phenomenon consists of rhythmical jerks of the head, synchronous with the pulse (*signe de Musset*). This is not peculiar to aortic regurgitation; it occurs in aortic aneurysm, and in large pleural effusions.

Aortic incompetence may also cause *capillary pulsation*. This may be seen under the nails, in the cheeks, or in the area of dilated capillaries produced by drawing a sharp point over the surface of the forehead; or by pressing a microscope glass slide upon the mucous membrane of the everted lower lip. In either case the vascular area under observation becomes alternately darker and paler with each beat of the heart.

**Symptoms.**—The symptoms of aortic regurgitation are strikingly different from those of mitral disease. This is due to the fact that the arterial supply from the heart may become deficient without any concomitant congestion of the lungs. The former gives rise to symptoms referable to cerebral anæmia, and there are sudden attacks of giddiness and faintness. Sleeplessness and nose bleeding are other common features. Patients are often *anæmic*, with pale face and lips and mucous membranes. There is often no shortness of breath. But the latter comes on if the whole heart begins to fail, and then all the symptoms and signs of venous congestion become obvious. Substernal pain is common in aortic regurgitation, and typical attacks of angina pectoris may occur. Patients occasionally die from a sudden attack of syncope, and it is possible that this may be due to vagal inhibition of the heart.

### MITRAL REGURGITATION

Since at each systole blood passes back into the left auricle, compensation for this lesion consists in a primary dilatation of the left ventricle and left auricle for the following reason. During systole the auricle receives blood that has leaked back through the mitral valve, but it also receives its normal quota from the lungs, so that it must dilate. All this blood passes into the ventricle, which must also dilate to receive it. The extra work involved in filling up the auricle and ventricle falls to a large extent on the right ventricle, which, in consequence, undergoes secondary hypertrophy, so as to hold more blood, and enable the full quota to be delivered to the aorta at each beat. Hypertrophy of the left ventricle will not be so obvious as in aortic regurgitation, because no work will be done in respect of the blood that regurgitates through into the auricle, since in the latter the pressure is low. The physical signs are the displacement of the impulse downwards and outwards, the systolic murmur audible at the apex, in the axilla, and often at one or both bases behind, and the accentuation of the second sound over the pulmonary artery. In early stages the impulse is more or less heaving and regular, and the pulse soft. In later stages the heart becomes irregular, whether from frequent premature beats or from auricular fibrillation, with a more and more feeble impulse, while the pulse is correspondingly small, feeble, irregular, and compressible, and its sphygmographic tracing shows a respiratory wave (*see* p. 300). Other types of cardiac irregularity may also be present. There may be dilatation of the right auricle with impairment of note to the right of the sternum.

**Symptoms of Mitral Disease.**—There are certain general symptoms, largely due to venous congestion, which are common to both mitral regurgitation and stenosis, and these will be considered here. The early symptoms are noticed particularly after exercise. They are shortness of breath and a feeling of exhaustion; to these may be added pain over the heart, palpitation and swelling of the feet; and this earlier stage may last for several years. The transition to *a later stage is often coincident with the occurrence of one of the previously*

described forms of irregularity of the heart's action, and especially of auricular fibrillation. But what particular external influence or what change in the functional conditions or in the structure of the myocardium determines the onset of these irregularities it is not easy to say. When the later stage is reached, the symptoms are in great part the result of the circulatory disturbances, and of the *retardation* of the blood flow, the effects of which upon the organs of the body have been described (*see* p. 337). Thus, as a result of the passive congestion of the lungs, the patient suffers from cough, from mucous expectoration, from occasional hæmoptysis, which may arise from the pulmonary infarcts above described, from orthopnœa at night or continually, and from dyspnœa on the slightest exertion. On examination crepitations will be heard at the bases of the lungs, and in advanced cases there will be dulness, with deficient vesicular murmur, and deficient tactile vibration. General venous stagnation is shown by a rich red colour or actual cynosis of the lips, cheeks, ears, and extremities, and by the occurrence of anasarca. The congested liver is large and smooth, reaching perhaps to the level of the umbilicus; and the skin is slightly jaundiced, the yellow tinge of the forehead combining with the deep red of the lips and cheeks to give a very characteristic appearance to the sufferer. Other results of the hepatic stagnation are ascites, congestion of the spleen, and more or less frequent vomiting. The secretion of the kidneys is also affected, the urine being scanty, reduced perhaps to 10 or 15 ounces daily, high-coloured, depositing large amounts of urates and containing albumin and fibrinous casts; the quantity of albumin is generally small, and varies inversely as the efficiency of the heart. Drowsiness or restlessness, and in advanced cases occasionally delirium, show the effect upon the circulation of the brain. Death takes place ultimately from cardiac failure, from œdema of the lungs and exhaustion, or from malignant endocarditis, or other complication (*see* also p. 346).

## MITRAL STENOSIS

In pure mitral stenosis, the primary effect on the heart is hypertrophy of the left auricle, but later on this leads to dilatation as well, especially when there is incipient heart failure. The right ventricle hypertrophies so as to cause a rise of blood pressure in the pulmonary system which acts against the resistance of the narrowed valve.

As compensation begins to fail, not only is there congestion in the lungs, but the left ventricle receives less than the normal quantity of blood. The cavity becomes smaller, and the ventricle wall may actually atrophy to some extent, and the aorta become narrower than usual in long-standing cases.

In early stages the impulse may be in its usual situation, and the condition is revealed only by the characteristic pre-systolic murmur (*see* p. 289), which is often followed by a reduplicated second sound. It has already been explained that the reduplicated second sound heard at the apex is often a very short mid-diastolic murmur (*see* p. 288). At the point on the chest where the murmur is best heard there is often a palpable vibration, or thrill, which is, like the murmur, pre-systolic in rhythm.

In later stages mitral constriction is apt to be complicated by either tricuspid or mitral regurgitation; the pre-systolic murmur is less often observed, being replaced by a murmur lasting all through diastole, which may also be accompanied by a thrill (*see* p. 288). Sometimes this and the pre-systolic alternate on succeeding days in the same case. Exceptionally the murmur may be early diastolic (*see* p. 290). Not infrequently there is no diastolic murmur, but a reduplicated second sound and a short systolic murmur, which is due to associated mitral regurgitation.

In the latest stages of cases of mitral constriction, when the auricle is fibrillating, the heart's action is most irregular and tumultuous. The rhythm of the diastolic

sound is difficult to recognise, and sometimes when the murmur is cut short by a fresh beat, it may be wrongly regarded as auriculo-systolic (*see* p. 315).

The pulse in early stages may be quite normal, and of medium pressure, corresponding to the regular action of the heart with normal frequency; but in later stages rapidity and irregularity both of time and volume are the characteristic features of auricular fibrillation.

In this condition, as already shown, the auricular impulses reach the ventricle in great number and with great irregularity, so that the ventricular contractions are necessarily irregular also.

The variation in the diastolic murmurs of mitral stenosis may be summed up as follows: When the heart is beating slowly and the stenosis is slight, a pre-systolic murmur is heard due to the hypertrophied auricle. If fibrillation supervenes, the murmur disappears entirely. If the action is slow, but the stenosis is greater, murmurs are heard all through diastole; they consist of diastolic and pre-systolic murmurs. In the case of auricular fibrillation, the murmurs occupy the earlier parts of diastole when the pause between the beats is long, or occupy the full diastolic interval when the latter is short. When the heart's action is quick both with normal rhythm and in auricular fibrillation, the murmurs tend to occupy the whole diastolic interval, but it is often very difficult to hear them at all.

Mitral stenosis often gives rise to hæmoptysis quite early owing to congestion of the lungs, and also to attacks of giddiness and faintness due to cerebral anæmia, and is perhaps more often than mitral regurgitation the cause of hemiplegia from embolism of the cerebral arteries. This arises from thrombi in the left auricle, which are formed owing to the stagnation of blood in the later stages of the disease. The other general symptoms were detailed in the last section.

Mitral stenosis is to be regarded as the final result of chronic attacks or repeated acute attacks of rheumatic endocarditis of the valve, which lead to adhesion and shrinking and thickening of the cusps. It requires some years for its development. This explains the fact that it is not met with in children, though it is met with from puberty onwards. Mitral regurgitation, on the other hand, is a less severe form of valvular inflammation, also due to rheumatism. The valves are thick and rather rigid, but they are not adherent to one another.

When obstruction and regurgitation are associated together at the mitral orifice, they give rise to various combinations of systolic and diastolic murmurs, of which some have been already mentioned (*see* also Figs. 23, 24, 25).

## RIGHT-SIDED VALVULAR DISEASE

*Tricuspid Regurgitation.*—Though tricuspid incompetence is a very common condition, it is usually due to the dilatation of the right ventricle, which is associated with mitral disease, pulmonary stenosis, myocardial disease, or with other conditions causing obstruction in the lungs (emphysema, bronchitis, fibroid phthisis). Much more rarely is it due to organic disease of the valve segments. It is commonly accompanied by the evidence of dilatation of the right auricle, and by the various degrees of œdema, anasarca, and venous congestion which indicate a difficulty in the return of blood to the right heart and lungs. These have already been enumerated under the later symptoms of mitral disease. The signs more characteristic of tricuspid regurgitation are the systolic murmur and the jugular and hepatic venous pulsations. The murmur of tricuspid regurgitation has been already described (*see* p 292); it is sometimes accompanied by a systolic thrill over the lower end of the sternum. The pulsation of the internal jugular vein, which occurs in these circumstances, may be very pronounced, causing an extensive undulating movement of elevation and subsidence at the side of the neck between the ear and the clavicle posterior to the course of the

carotid artery. The external jugular vein may pulsate at the same time. Usually auricular fibrillation is present

In tricuspid regurgitation also the force of the right ventricular contraction may be transmitted to the hepatic veins, so as to cause *hepatic venous pulse*, or *pulsating liver*. The organ is commonly much enlarged, and can be felt throbbing over its whole surface; and the pulsation is sometimes even conveyed behind into the right loin under the last rib, so that the liver can be felt to expand between the hands placed in front and behind.

*Tricuspid stenosis* (*see* p. 292) is less common, and is generally observed in conjunction with disease of some other valve, especially mitral stenosis. No special group of symptoms can be referred to it apart from those seen in tricuspid regurgitation.

*Disease of the pulmonary valves* is mostly congenital if of old standing, and if acute it is the result of malignant endocarditis. *Pulmonary stenosis* is the usual condition in the former case: the valves are united together to form a cone, with only a small opening for the passage of blood, but they may close perfectly. The murmur is systolic (*see* p. 292), and is often accompanied by a systolic thrill over the same area. The obstruction leads to dilatation of the right ventricle, and when accompanied by a patent septum it hinders access to the lungs, so that a very imperfectly aerated blood circulates, and the patient is habitually cyanosed (*see* Congenital Malformations).

*Pulmonary regurgitation* sometimes occurs as a result of mitral stenosis, the valves yielding before the great pressure in the pulmonary artery; a diastolic murmur is heard along the left side of the sternum.

Malignant endocarditis may cause a double (systolic and diastolic) murmur at the pulmonary orifice like that of aortic disease, the respective murmurs having the positions previously noted. The symptoms under such conditions have already been detailed (*see* Malignant Endocarditis).

## DIAGNOSIS, PROGNOSIS, AND PREVENTION OF CHRONIC VALVULAR DISEASE.

**Diagnosis.**—In the diagnosis of valvular heart disease many questions have to be considered. It has to be determined (1) whether a murmur is due to a valvular lesion or to some other cause, endocardial or exocardial, the former including change in the muscular walls; (2) at which orifice it arises, and if there are two, whether one is dependent on the other; and (3) what is the functional capacity of the heart, *i.e.* its response to exercise and the condition of the several cavities of the heart. Very important information as to the heart's position, action, and valvular working can be got by the eye and the hand; and these should always be used in conjunction with the stethoscope. The Röntgen rays will also assist in the estimation of changes in the size and shape of the heart's cavities (*see* Fig. 27).

1. The murmurs of valve disease are apt to be confounded with those due to other conditions. *Anæmia* produces a harsh systolic murmur over the pulmonary area. Seeing the rarity of organic pulmonary disease, this is mostly distinctive enough, but with very considerable anæmia murmurs extend over the whole præcordial area, arising, no doubt, at other orifices besides the pulmonary As in such cases the patients are short of breath, with a tendency to palpitation and to swelling of the feet, the diagnosis may be difficult. The marked pallor of the anæmic cases, the absence of history of rheumatism or other precursor of heart disease, and the diminution of the murmur under the use of iron tonics, are points which will help. But anæmia may also undoubtedly itself be a cause of mitral regurgitation; the deficient quality of the blood causes malnutrition of the wall of the ventricle; this dilates, the mitral orifice yields, and regurgita-

tion is the result. This is, indeed, an actual lesion, and the murmur is immediately due to structural changes in the orifice, if not in the valve itself; but inasmuch as they are primarily due to a condition of the blood which, together with its results, is curable, this murmur is often spoken of as *functional* or as *hæmic*. The diagnosis must, at any rate, be made between this and chronic valvular disease, and it can generally be effected by a consideration of the preceding and associated circumstances—viz. the absence of rheumatism and the decided anæmia.

*Aneurysm* of the aorta frequently gives rise to a murmur at the base of the heart which may be mistaken for that of aortic obstruction. Indeed, a simple systolic murmur in the aortic area, unaccompanied by regurgitant murmurs, is much more often due to aneurysm than to stenosis of the valves. Abnormal pulsation to the right of the sternum and increased area of dulness should be sought for as further evidence. If the murmur is localised at a point not strictly corresponding to the known areas of valve disease, aneurysm is still more probable.

*Pericarditis* often gives rise to a to-and-fro sound, very like the murmur of double aortic disease. It is, however, usually rougher, less uniform in loudness over a large area, not strictly localised to the usual area of aortic disease, and perhaps here and there not strictly synchronous with the two periods of the heart's beat. A short history of acute illness, unusual pain, or distress at the heart, increased area of præcordial dulness in an upward direction, and absence of splashing pulse, point to pericarditis.

Another difficulty arises from *exocardial* murmurs, which are sounds synchronous with the heart's action, but produced outside the heart (*see* p. 293). But the recognition that a murmur is endocardial and produced at a valvular orifice does not carry with it the diagnosis of disease of the valve. Ventricular dilatation not only from anæmia, but from any cause whatever, may lead to an apex systolic murmur; and such an occurrence is most common in Bright's disease, in alcoholism, and in arterio-sclerosis, and acutely in the myocardial disorders of infectious disease.

*Chronic renal disease* may bring about hypertrophy of the heart, and even dilatation and murmur; and the case will then closely resemble one of mitral disease with secondary albuminuria. The difficulty is increased by the fact that the kidneys in a state of chronic congestion from heart disease may become granular; and that a heart dilated, as a result of extreme arterial tension in renal disease, will cause secondary stagnation in the venous system, like one affected with primary mitral disease. In primary heart disease one must look for the history of rheumatism or other cause of endocarditis. The urine has the characters described (*see* p. 342), and the pulse is small and of low tension But in renal disease the urine is more likely to be pale, though also scanty, and to have a more uniform quantity of albumin; and the pulse is one of high tension. In enlargements due to arterio-sclerosis and to alcohol, conditions which are often combined, the arterial tension is variable, and albumin is often absent; the diagnosis may have to depend on the history or associated conditions.

Conversely a valvular lesion is sometimes present when no murmur can be heard; this is most frequently the case in the later stages of mitral constriction, when the strength of the auricle is failing.

2. The diagnosis of the different forms of valvular disease one from another depends for the most part on the character of the murmurs, and the extent to which they are audible over the præcordial area. A murmur may be conveyed beyond the area of one valve into the area of another, when it will be necessary to compare carefully the intensity of the sound at different points. Aortic regurgitation and mitral regurgitation are nearly always indicated by their characteristic murmurs. Mitral obstruction frequently exists without its characteristic murmur, as above stated. Pre-systolic murmurs and diastolic murmurs,

when heard at the exact impulse (and not heard at the base), are very strong evidence of mitral obstruction. But murmurs identical with these are sometimes heard at the apex in association with aortic regurgitation—Flint's murmurs (*see* p. 340) with adherent pericardium, and with a dilated ventricle under other conditions. The explanations of these anomalies are various: vibrations of the anterior mitral cusp from impaction upon it of the aortic regurgitant current, or from its being driven in upon the auriculo-ventricular current; mingling of the above two currents; the formation of a "fluid vein" in consequence of the dilatation of the left ventricle, while the mitral orifice is of normal size, a condition sometimes called relative stenosis. The last is the more likely explanation.

3. The estimation of the heart's response to exercise is perhaps the most important point in diagnosis. The amount of breathlessness or exhaustion after exercise is observed (*see* p. 294). The size of the heart provides a useful guide as to the extent of the valvular defect. This may be determined by palpation and percussion. X-rays may be used, and also the electro-cardiograph to indicate the proportion of right and left-sided hypertrophy (*see* p. 303).

No diagnosis can be made without an examination of the heart and lungs; but it is interesting to note that there is often in children and young people a superficial resemblance between mitral disease and phthisis, since the former may produce marked pallor, emaciation, and hæmoptysis.

**Prognosis.**—The condition of the heart muscle and the extent of the valvular lesion are the two most important points in prognosis, as on these factors will depend the efficiency of the heart as a propelling organ. The former is estimated by the response to exercise (*see* p. 294); the latter is determined by the amount of hypertrophy and dilatation of the heart. It is very unsafe to draw conclusions from the loudness of the murmurs.

A very important point is the nature of the infection. In syphilitic disease, which affects particularly the aortic valves, the prognosis is bad, because the process tends to progress both in the arteries and the valves. On the other hand, in rheumatic infection the lesion may have healed, and so may remain *in statu quo* for years. Repeated attacks of rheumatism, or other infections such as tonsillitis, are dangerous because they may either lead to an acute or subacute infection of the valves, and cause further damage. As to the particular valve affected, pure aortic stenosis is usually regarded as the least serious, and aortic regurgitation as the most serious, lesion; but this may be because so many cases of aortic regurgitation are due to syphilis. Mitral regurgitation is often of no importance. In the case of soldiers invalided with the effort syndrome the presence of a mitral regurgitant murmur was of no value as an indication whether the man would be able to return to full duty or not. Mitral stenosis is a serious lesion, as it is the end result of chronic or repeated acute attacks of rheumatic mitral disease. In slight cases, however, it may remain stationary for years if the infection is arrested.

The prognosis is naturally more grave when two or more lesions co-exist; aortic disease rapidly terminates when the mitral valve becomes secondarily involved, and tricuspid regurgitation is a serious complication of left-sided disease.

Analysis of the cause of death in eighty-one cases of chronic valvular disease of all ages at the Royal Infirmary, Glasgow, gave the following results (**14**). The maximum age incidence of death was between thirty and fifty. There were two groups of cases. (1) No fewer than 48 per cent. were found to have acute or subacute endocarditis in addition to their chronic valvular lesion, and in most of these cases the onset of the fatal illness was insidious. In more than three-quarters obvious septic foci were present outside the heart, which may have been responsible for the superadded infection, such as sepsis round the tonsils and teeth in most cases, and atrophic rhinitis, otitis media, cholecystitis, gastric ulcer, dermatitis, pulmonary and uterine infections. (2) The remaining 52 per cent. were classified as follows: In 7·5 per cent. death was independent of the heart

condition. In the remainder heart failure had been present for some time, and in some cases the patient could trace its onset to particular incidents, *e.g.* an attack of influenza or bronchitis. In a surprisingly small number, 7·5 per cent., was the onset of failure definitely attributable to increased muscular exertion. In one-half of this group failure was aggravated by abnormalities of cardiac rhythm, auricular fibrillation occurring most frequently. The immediate cause of death was, in most cases, the direct failure of the heart due to associated myocarditis.

**Prevention.**—Since most of the cases of heart disease in young children are due to acute rheumatism, prevention consists in counteracting this disease. Unfortunately the ætiology of acute rheumatism is still obscure, but there is a general consensus of opinion that the rheumatic infection commonly gains an entrance to the body through the tonsils, so that particular attention should be paid to the nose and throat, and obviously diseased tonsils should be enucleated, especially if the tonsillar glands are palpable (*see* p. 59).

In older subjects the teeth should be examined, particularly in people who already have chronic valvular disease, as any infection they may pick up may lead to infective endocarditis. Pyorrhœa alveolaris, when there is a free discharge of pus, is not so dangerous, but care should be taken to exclude abscesses hidden away at the roots of the teeth, from which the pus is not discharged, and which may lead either to an acute attack of endocarditis, or to a chronic smouldering infection with progressive deformity of the valves.

Another very important point in prevention is to ensure that the child who has suffered from the mildest rheumatic manifestations is put to bed whenever there is any indication of acute infection, such as a sore throat, tonsillitis, or a common cold, because there is always the chance that the heart may be simultaneously affected. In fact, it is the greater care taken about children of the better classes in this respect that probably ensures their relative freedom from severe heart disease compared with children of the working classes.

Of the specific infections syphilis is the commonest cause of heart disease, and early intensive anti-syphilitic treatment should be carried out, if the disease is contracted.

Arterial disease leads commonly to myocardial degeneration, and the appropriate prophylaxis (*q.v.*) should be undertaken.

## TREATMENT OF CHRONIC CARDIAC DISEASE

The following remarks apply not only to chronic valvular disease of the heart, but also to cases of adherent pericardium and myocardial degeneration.

Before considering treatment it is necessary to find out (1) whether there are early signs of cardiac failure—distress, breathlessness and substernal or præcordial pain after exercise; (2) whether the later signs are present—engorgement of the veins in the neck and of the liver, cyanosis, and œdema; (3) whether the heart is enlarged, and whether there are signs of valvular disease or myocardial changes; (4) whether there is any cardiac irregularity, particularly auricular fibrillation; (5) whether there is evidence of active infection in the heart (Lewis).

The principles of treatment consist in ordering the life of the patient, so that the work the heart is called upon to do is well within its capacity.

In early cases of cardiac failure the patient's symptoms are the main guide. It is necessary to find out the amount of work that causes the onset of undue fatigue, or distress, or breathlessness, or of cardiac pain.

In the perfectly healthy individual the sesymptoms are only experienced after the most severe muscular exercise. Recent experience has taught that there are many individuals with systolic murmurs audible over the præcordium who have no history of rheumatic or other infection, and show no enlargement of the heart,

who can take the most severe muscular exercise with no more distress than the normal person experiences. Such persons need not be restricted in regard to the exercise they take. When, however, there is reason to suspect aortic regurgitation or mitral stenosis, it is wise not to allow the patient to call forth his whole cardiac reserve, even if violent exercise can be undertaken with no more than the normal amount of distress. Only the milder forms of exercise should be permitted. The same thing applies to cases of mitral regurgitation and aortic stenosis when there is definite cardiac enlargement.

Rest in bed is indicated in all cases where actual infection is present, in all cases of advanced cardiac failure, in cases showing signs of venous congestion, in cases of auricular fibrillation where the heart is rapid, and it is required to administer a full course of digitalis, and, most important of all, in patients who show signs of distress when standing or walking leisurely. The patient should lie recumbent, unless there is orthopnœa, when he should be propped up in bed. Complete quiet and freedom from anxiety and excitement should be enjoyed. All unnecessary movement should be forbidden, and, in particular, sleep should be encouraged, as this is the condition that gives the heart its most complete form of rest. This is one of the most important points to be attended to in the treatment of any cardiac patient, whether some exercise is allowed or not. Nine to ten hours in bed should be aimed at, even though the number of hours of actual sleep is less than this. Whisky, 1—2 ozs., may be given as a soporific, or paraldehyde in doses of 1—2 drachms. The diet should be sufficient, simple, and readily digestible; it may be mixed solid and liquid, in quantity at any one time not to overload the stomach, and of a nature not to cause flatulence or distension. Fluids should not be given in excess.

In patients who exhibit symptoms after moderate exercise, such as running or walking upstairs or uphill quickly, or walking quickly along the level, the exercise causing the symptoms should be forbidden. At the same time such exercise as can be tolerated should be allowed. It is bad practice to under-exercise his heart. However, the patient should be told to remain quite still supposing the symptoms come on while out for exercise.

When a patient has obtained relief after rest in bed for some time, exercise should only be begun gradually. Graduated exercises may be given by allowing him to move his arms and legs while still in bed. Another plan, when the patient is up, is to increase the amount of walking from day to day until his exercise tolerance is reached.

In cases of advanced cardiac failure with congestion of various organs and œdema, there are three main lines for treatment: (1) Removal of œdema fluid or bleeding; (2) administration of oxygen; (3) of drugs, especially digitalis.

(1) If much fluid is present in the pleural cavity, its removal by tapping will bring great relief. If there is much anasarca, the legs may be punctured in ten or twenty places with a large flat surgical needle, or drained by Southey's tubes. The patient should sit upright in a chair for twelve or twenty-four hours with the legs down, so as to allow the fluid to gravitate into them. They must be cleaned scrupulously and painted over with a weak solution of iodine. The fluid is allowed to run down into a bath, which should be surrounded with a blanket for warmth, but the blanket must not touch the legs where they have been punctured. In ascites the abdomen may be tapped, and by these proceedings the pressure on the circulation is diminished. The indications for *venesection* are severe orthopnœa, and cyanosis with distended veins. This means that there is a fall of general arterial blood pressure and a corresponding rise in venous pressure, so that the right side of the heart has become so engorged that it has difficulty in contracting upon its contents. In such circumstances, auricular fibrillation is often present; the withdrawal of blood to the extent of 20 or 30 ounces by a cut into the median basilic vein relieves the distress by diminishing the blood flow to the heart. A still more rapid effect may be obtained in extreme cases by

opening the external jugular vein at the bottom of the neck. In this case the blood will flow from the central end if the venous pressure is high, and the right side of the heart will receive direct relief. It is not always easy to obtain enough blood by a simple cut into the vein. A more effective method is to use a short wide hollow needle connected to a closed bottle by means of rubber tubing. Suction can then be applied to the bottle.

(2) Oxygen should be used whenever there is dyspnœa and cyanosis. It cannot be given in too great concentration (*see* p. 234).

(3) The *drugs* which act directly upon the heart in a favourable sense are comparatively few. Digitalis is the most valuable and the one whose action has been most studied. In auricular fibrillation its use has already been described (*see* p. 315). In these cases the ventricle is beating rapidly and irregularly, and digitalis acts like a specific. The pulse is slowed. The urine is increased in volume, and the œdema disappears. But in other cases, even when auricular fibrillation is not present, digitalis may be used with success, especially for its diuretic action. The powdered leaves are often combined with mercury in the form of a pill which is used for such cases. It cannot be said that its action is so certain as in auricular fibrillation. Digitalis may be given by mouth in powder, infusion, or tincture, or as one of its active principles, digitalin or digitoxin. In serious cases 2 drachms of the infusion or 10 or 15 minims of the tincture may be given every three or four hours at first, and after twelve or twenty-four hours less frequently or in smaller doses. The dose of digitoxin is $\frac{1}{250}$ grain to $\frac{1}{60}$ grain. Given in these doses, digitalis takes two or three days to produce its full effect. Much more rapid action is obtained by giving massive doses by mouth, a treatment that may be carried out when there is auricular fibrillation (*see* p. 316). Toxic symptoms arising during digitalis treatment are described on p. 316.

Some other drugs have an action like that of digitalis. The most important of these is strophanthus (dose, 2 to 5 minims of the tincture). The active principle of strophanthus, strophanthin, is valuable where there is no time to be lost in cases of serious failure. $\frac{1}{250}$ grain may be given intravenously. It may also be injected intramuscularly or subcutaneously. The treatment of auricular fibrillation by quinidine has already been described (*see* p. 316). Strychnine has been regarded in the past as a valuable cardiac tonic. A carefully controlled series of observations has shown that strychnine has no effect either in acute or chronic cardiac failure (**15**). Oubaine ($\frac{1}{4}$ mg. intravenously, or 1 mg. by mouth, once or twice a day) has been recently used for myocardial degeneration in France, to increase the tonicity of the left side of the heart.

Other symptoms and complications may have to be treated. If dropsy does not yield to digitalis, theobromine sodium salicylate, in doses of 10—20 gr. t.d.s., is the best drug to use, either in the form of tablets or in a mixture well flavoured with syrup of ginger. Pain over the heart is often severe, and may be relieved by belladonna plasters, by small doses of morphia internally, or by subcutaneous injection of gr. $\frac{1}{4}$. Cough may be treated with small doses of expectorants and sedatives, and vomiting by effervescing salines. In aortic disease with hypertrophy, one of the most distressing symptoms is the violent action of the heart and the throbbing of the great vessels in the neck and over the body generally: and this may be much relieved by the use of a small dose of tincture of aconite (1 to 3 minims), by bromides, or by a small dose of morphia. Pulmonary hæmorrhage is rarely sufficient to threaten life, and does not require styptics. In myocardial disease of elderly subjects (*see* p. 325) paroxysmal attacks of breathlessness cause much distress. Oxygen inhalations (*see* p. 234) usually provide relief, and subcutaneous injections of morphia are also valuable (**18**).

Cerebral embolism is beyond the reach of direct treatment, but embolism of an artery in one of the limbs must be met by the application of warmth, to avoid as far as possible the supervention of gangrene. Throughout, the diet should be in moderate quantity, light, easily digestible, and unstimulating.

With a much-weakened heart, and in late stages, however, stimulants, in the form of brandy, whisky, or sherry, will form an essential part of the treatment.

Syphilitic myocarditis should be treated over an extended period with anti-syphilitic remedies. Deep subcutaneous injections of sulpharsenol (0·3 to 0·48 gram) may be given weekly. A course consists of 6 grams, and six weeks are allowed to elapse between the courses. During the course yellow iodide of mercury, gr. $\frac{1}{8}$, may be given three times a day as tabloids, and increased to 8 or 12 a day. Potassium iodide may be given between the courses.

Mention may be made of the fact that the operation of cardiotomy and valvulotomy for mitral stenosis has been performed, with recovery of the patient (**19**). The writer is informed that the stenosis has reappeared.

**Heart Disease and Pregnancy.**—The question not uncommonly arises as to whether pregnancy should be allowed in patients who on auscultation are found to have murmurs characteristic of mitral regurgitation, mitral stenosis or aortic regurgitation. Pregnancy should be permitted: (1) if there is evidence that the valvular defect is of long standing and that there has not recently been any inflammation in the valves; (2) if the response to effort is good; (3) if the heart is not enlarged or unduly excitable; (4) if the rhythm is normal; (5) if in aortic regurgitation there is no great difference between the systolic and diastolic blood pressure, and the apex beat is not too far out or too forcible; (6) if in mitral stenosis no persistent crepitations are heard in the lungs after coughing or deep breathing, indicating commencing œdema. No attention should be paid to the occurrence of extra systoles in pregnancy; but the presence of auricular fibrillation should be looked upon as an absolute bar (Mackenzie). If pregnancy has been begun against advice the case must be carefully watched and pregnancy terminated if unfavourable symptoms present themselves. When the heart shows some signs of functional inefficiency the patient should rest, propped up in bed, taking deep breaths at intervals during the day so as to aid the circulation through the lungs. Induction of labour during the later months of pregnancy is often a long business and so is more of a strain on the heart than if labour comes on spontaneously. In such a case Cæsarean section may be considered, especially as sterilisation can be performed at the same time. Mackenzie states that no degree of heart disease in a married woman is a bar to sexual connection, supposing she feels the desire for it, and ability to perform it.

## EFFORT SYNDROME

(*Disordered Action of the Heart, Soldiers' Heart*)

During the late war the effects of the war strain upon the heart of the soldier have been constantly the subject of observation and study. Thousands of soldiers were at different times invalided for symptoms suggestive of a weak or diseased heart, and in such cases the nature of the cardiac or other lesion, the best method of treatment, and the prognosis as to the patient's future in relation to military service, whether discharge or return to duty, had to be considered. The subject is dealt with in a report by T. Lewis to the Medical Research Committee (Special Report Series, No. 8, 1918); and in this important report are given the results of the study of 1,000 soldiers returned as sick during training or on active service for actual or supposed defects of the cardio-vascular system.

**Ætiology.**—There is a definite symptom complex, called the "effort syndrome," which occurs under a number of different conditions, such as tuberculosis, exophthalmic goitre, valvular disease of the heart, and in other conditions where there is no obvious lesion in the body. It is this latter type of case that is known as "disordered action of the heart"—a name that is

objected to, because it points solely to the heart, and thus gives a bad impression to the patient. Lewis is inclined to think that the heart action itself is only a subsidiary feature in the disease.

Before study and treatment it is necessary to separate off those other diseases causing the same symptoms. In particular, valvular disease of the heart is held to include cases exhibiting aortic or mitral diastolic and pre-systolic murmurs, but not those with systolic murmurs, whether at the base or apex, owing to the uncertainty of the significance of this type of murmur. Systolic murmurs in soldiers only exceptionally mean valvular disease, and often the amount of damage to the valve is but slight. Further, patients who are invalided on the ground of systolic murmurs alone are found when tested to be fit for active service in nearly all cases.

The malady is found chiefly in men of sedentary occupation, and especially among the more intellectual class of workers. Temperamentally they are unusually sensitive. There is a prevalence of teetotalism among them (53 per cent. in 454 cases). A history of venereal disease is very uncommon; on the other hand, there is often the certainty of self-abuse.

The action of the vagus is probably abnormal, as shown by the variation in the pulse rate during respiration, and occasionally there is profound slowing of the pulse, with fainting fits.

The sympathetic system is more easily stimulated and depressed by adrenalin and apocodeine than normal; this suggests that sympathetic stimulation may account for the cardiac acceleration.

There is no evidence that hyperthyroidism is the cause. Experiments have shown that these patients are just as tolerant to thyroid administration as normal people.

The high incidence of rheumatic histories in the patients (23 per cent.), in spite of the absence of physical signs in the heart, suggests the possibility of early myocardial change as the cause in many cases. However, there is no indication of heart block immediately after exercise, as indicated by the electro-cardiograph, which is usually a valuable sign.

Blood cultures are negative.

It does not seem probable that tobacco smoking is the cause of the condition, though it has been proved that tobacco increases the pulse rate in these cases more than in normal people. In the first place, it is as commonly met with in Sikh soldiers, who do not smoke, as in other Indian troops. Again, the consumption of tobacco is not usually inordinate, and, curiously enough, the heavy smokers return to duty in a higher percentage of cases than the light smokers do, but the non-smokers do best of all. An obvious explanation is that the light smokers are not so much accustomed to tobacco as the heavy smokers. There is no doubt that tobacco smoking aggravates the condition.

It is possible that some toxæmia may be at the bottom of the condition; the symptom complex is very similar to that met with in early tuberculosis, and there is often a history of previous infection. In 50 to 60 per cent. of cases infections are legitimately suspected to have played a large part in the production of the malady.

**Symptoms.**—The "effort syndrome" consists of the following symptoms: *Breathlessness.*—This is a constant feature, especially on exertion. A respiration rate of sixty to eighty is by no means uncommon during the waking hours. During sleep the rate is normal, and it is usually not increased when the patient is lying at rest in bed. *Pain.*—This occurs in about three-quarters of the cases, and varies in character from uneasiness over the præcordia to pain of anginal distribution; it is especially associated with exercise. *Exhaustion* is an almost constant symptom; it is provoked by sustained effort, and is far in excess of that due to fatigue in healthy men. *Giddiness and Fainting.*—Giddiness is almost constant, and is associated with change of posture and with effort. Attacks of

fainting are less common. *Palpitation* is frequent, especially with exercise. It is usually due to rapid and vigorous heart beats, and not often to extra systoles, or other well-defined cardiac irregularity. However, extra systoles do occur, and sinus arrhythmia with respiration is not uncommon. *Headache* is almost constant. *Sweating and coldness of the extremities* is common. The response to pilocarpine is greater than normal. Irritability of temper, sleeplessness, inability to fix attention, shakiness, tremor of the hands, and flushing are common. A disinclination to take alcohol in any form (sometimes for conscientious reasons, but as commonly for reasons of distaste) is to be reckoned as a frequent and remarkable association.

The following are the physical signs :—*Increased heart rate*, especially marked in response to emotions, exercise, or alteration of posture, *i.e.* changing from recumbent to erect position. A notable and widely recognised sign is the slow return of pulse rate after effort. *Blood pressure* is usually normal when the patient is at rest, but there is an exaggerated response to emotion or effort, and high readings are often obtained. *Diffuse apex beat* is common, and this may or may not be associated with increased force of the impulse. It is commonly regarded as a physical sign of dilatation of the heart, but X-ray examination by the orthodiagraph shows that there is no enlargement of the heart, so that the physical sign is untrustworthy. The *deep reflexes* are usually exaggerated. The *urine* in 60 per cent. of cases is hyperacid, the quantity being reduced, and the ammonia and amino acids are increased. In 20 per cent. the volume is also reduced, and the urine deposits phosphates. The ammonia is normal in amount, but the amino acids are increased. Speaking of the urine generally, calcium oxalate crystals are often found and spermatozoa are found in large numbers in the morning urines in 15 per cent. of cases. The *blood* shows a leucocytosis, the average being 12,100 c.m., the lymphocytes being increased. A much larger leucocytosis than normal is observed after exercise, the lymphocytes being increased, and the severity of the symptoms runs parallel with the leucocytosis. The electro-cardiograms show no abnormality.

**Prognosis.**—The following criteria decide whether the patient will in future only be fit for sedentary work : a history of rheumatic fever, persistently severe breathlessness on exertion, præcordial pain sufficiently severe to prevent exercise, a pulse rate of 120 and more even in recumbency, symptoms which have been present for many years, even though of only moderate severity. A method of testing patients is for them to walk up a flight of thirty steps; indications to be looked for are an anxious expression, a respiratory rate of thirty-five or over, which persists while the patient lies and is interrogated from time to time, a pulse rate which fails to fall within five beats of the pre-exercise level on lying for two minutes.

All the men who are not excluded so far are treated with graduated exercises ; selected army exercises are used, with which the men are mostly familiar, and these are supplemented by route marches in light or full kit. The exercises are arranged in seven groups of increasing severity, and a patient moves to a higher grade every three or four days. The exercises last fifteen or thirty minutes daily. The men are classed for duty on the highest grade of exercise they are able to stand without distress. The average period required for classifying the men is a month and a half. Of 220 soldiers discharged from the hospital, 182 (83 per cent.) were still fit for duty in one capacity or other three months afterwards.

**Prevention.**—A long and gradual training is advisable in recruits whose occupations have been sedentary, for these form a very high proportion of the patients, a longer convalescence from febrile affection or affections of the bowel, with a graduated system of retraining.

**Treatment.**—This consists in clearing up local foci of infection (extraction of carious teeth, removal of tonsils, etc.). The removal of a latent dysentery

infection by emetine bismuth iodide has several times ameliorated the condition. If the symptoms are recent, a period of rest from drill and exercise is desirable, but rest in bed is harmful, and should always be avoided, except in severe præcordial pain, headache, or giddiness. Occupation is clearly called for, especially in the open air, such as gardening. They should be encouraged by the assurance that their malady is curable, and that improvement is visible. No particular attention should be paid to the heart. Tobacco increases the resting pulse rate and the symptoms after exercise. Bromides are valuable in the early stages. The most important treatment is by graduated exercises. Digitalis is valueless.

## CONGENITAL MALFORMATIONS

Malformations of the heart arise from defects in its development, which is normally not complete until the closure of the ductus arteriosus and foramen ovale some days after birth. Arrest of this process in any stage will lead to a congenital malformation. It has occurred so early as to leave the heart with only two cavities—an auricle and a ventricle ; or with three cavities—a ventricle and two auricles. But these are very rare cases, and the children mostly live but a short time after birth. One of the commonest lesions is a combination of pulmonary stenosis and deficiency of the interventricular septum. The stenosis is caused either by union of the pulmonary valves, or by a constriction of the ring just below them, or of the infundibulum itself, or by an imperfect septum dividing the infundibulum from the ventricle. Such conditions are regarded by Keith as invariably due to defects in development or to want of expansion of the bulbus cordis, and never to intra-uterine endocarditis. If the outlet of the right ventricle is thus obstructed in early fœtal life, the pressure in that cavity is relieved by overflow into the left ventricle through the still unclosed septum ; and this opening then becomes permanent. According to the stage of development at which the arrest has taken place, the deficiency may be a very large one or a mere perforation in the upper part ; and in this latter case the aperture occupies the *pars membranacea*. When the deficiency is a large one, the aorta frequently arises from the right ventricle, or from both right and left ventricles, and the foramen ovale and ductus arteriosus may one or both be pervious. Both pulmonary stenosis and patent interventricular septum may recur independently of one another. In a few rare cases the communication between the two ventricles has been below the anterior aortic valve (over which arises the right coronary artery) instead of the right posterior valve ; it is then in front of the pars membranacea and opens into the infundibulum of the right ventricle (**20**).

Constriction or obliteration of the aortic orifice or of one auriculo-ventricular orifice sometimes occurs, and similarly interferes with the course of the circulation and the normal development of the heart ; and complete transposition of the aorta and pulmonary artery and other complicated transpositions of vessels have been found.

The ductus arteriosus and the foramen ovale may remain unclosed without any obvious reason —probably, however, from a temporary obstruction to the circulation at the time of birth ; but more or less patency of the foramen ovale occurs in about 30 per cent. of healthy persons, a mere fissure or narrow valvular opening being insufficient of itself to allow of any free passage of the blood from one cavity to the other.

Instead of three sigmoid valves in the aorta or pulmonary artery there may be only two, or there may be four. This change may exist in association with other deformities, but if alone it is less likely to give rise to difficulties at birth than to lay the foundation of disease in later life.

**Pathology.**—No more is really known of the cause of arrested or faulty development of the heart than of congenital malformations in other parts of the body. One of the most characteristic symptoms of congenital heart disease is

*cyanosis*, and the disease has been called the *morbus cæruleus*. Some recent observations have thrown light on the cause of cyanosis in general (**10**). (1) It is due to slow circulation of blood in the peripheral capillaries, so that the returning venous blood contains less oxygen than usual; (2) it is due to the arterial blood not being fully saturated with oxygen. Of course both these factors may be combined, and the cyanosis is intensified if the red cells of the blood are increased. There seems little doubt from determinations of the oxygen content of blood taken direct from the radial or brachial artery through a fine needle that, although in marked cases there may be some arterial unsaturation, it is the peripheral stasis factor that is chiefly responsible for the cyanosis of acquired heart disease. The arterial blood may contain less than the normal quantity of oxygen (*a*) if there is a hindrance in the lungs to the exchange of gases, such as may occur in œdema, pneumonia, bronchial spasm, or chronic pulmonary disease; (*b*) if the arterial blood is a mixture of blood from the lungs and from the systemic veins, as may occur with a deficient septum ventriculorum or patent foramen ovale, associated with congenital pulmonary stenosis. Slowness of the blood through the lungs, such as occurs in acquired heart disease, will by itself tend to complete saturation of the arterial blood, because there is plenty of time for oxygen to be taken up. It was found in certain cases of congenital heart disease with marked cyanosis that the arterial blood was only 70 to 80 per cent. saturated with oxygen. When the patient breathed pure oxygen for half an hour through a mask and valves, the saturation was raised to just over 90 per cent. This is compatible with the arterial blood being composed of a mixture of pulmonary and venous blood, because it was impossible to saturate it completely by giving oxygen. At the same time the peripheral stasis factor is also present in congenital heart disease and increases the cyanosis.

The common lesion is the association of pulmonary stenosis and deficient septum. The stenosis may be so extreme that very little of the blood can reach the lungs by this route. Nearly all of it passes into the aorta and mixes with blood from the left ventricle. In these extreme cases the bronchial arteries are dilated and a fair supply of blood reaches the lungs by means of the anastomoses of these arteries with the small branches of the pulmonary artery. In one case the pulmonary valve was found to be entirely closed and no systolic murmur was heard over the pulmonary area; the patient was extremely blue. The heart was only slightly larger than usual, since all the blood travelled into the aorta, and there was no extra resistance (**10**, Case 15). Where the pulmonary valve is patent, though stenosed, less blood will pass into the aorta; the right ventricle may be hypertrophied and dilated, and there will be a systolic murmur and perhaps a thrill in the pulmonary area. In certain cases a patent foramen ovale may act instead of a deficient septum in short-circuiting the blood. A patent foramen ovale or a patency of the septum will be of no functional significance if the pulmonary valve is normal, because there will be nothing to prevent the blood going through the lungs in the ordinary way. At most a little blood may pass through from the left to the right ventricle, and so travel through the lungs a second time. A patent ductus arteriosus associated with a pulmonary stenosis and deficient septum will provide an extra path for the mixed arterial blood to pass through the lungs; if present by itself it will be of little functional significance.

**Symptoms.**—Cyanosis when present is most marked in the prominent parts of the face—the cheeks, lips, nose, and ears, and in the fingers and toes. In slighter cases it is only richer red than natural; in the severest cases it is purple almost to blackness, and any exertion at once increases the distension of the vessels and deepens the colour. It is a remarkable fact about this disease that, though the cyanosis may be extreme, there is no shortness of breath while the patient is at rest. The chronic stagnation will cause thickening of the parts affected, and the nose and lips are coarse, while the ungual phalanges of the fingers

or toes are thickened much beyond the rest of the fingers, or "clubbed" (*see* p. 672). The blood shows in a remarkable degree the great excess of red corpuscles (*polycythæmia*) which is met with in many forms of cyanosis; thus the corpuscles have been found to number from 8,000,000 to 9,000,000 per cubic millimetre, and the hæmoglobin may reach 110 to 160 per cent. of the normal. The patient sometimes suffers from attacks of giddiness, faintness, convulsions, and loss of consciousness. He is incapable of much exertion, from the readiness with which dyspnœa supervenes; and he is also peculiarly susceptible to cold or exposure, and easily suffers from attacks of catarrhal bronchitis. In later stages œdema of the legs, ascites, enlarged liver, and albuminuria are found; or the patient succumbs to bronchitis; or tuberculous disease of the lung is the cause of death.

The **Physical Signs** do not always give data as to the malformation present. The præcordial region is sometimes prominent, and the dulness may extend to the right of the sternum in consequence of the dilatation and hypertrophy of the right side of the heart. Most commonly a systolic murmur is heard, loudest over the pulmonary area in congenital pulmonary stenosis, but it may also be heard over adjacent spaces. It may be accompanied by a thrill. In deficiency of the septum ventriculorum a systolic murmur is usually described, of maximum intensity in the third left space near the sternum, and this is conducted outwards. There may also be a thrill. But, as already pointed out, cases of marked cyanosis may occur without any cardiac murmur. A patent ductus arteriosus often causes a prolonged murmur running through systole into diastole, and waxing and waning in loudness.

The various types of congenital heart disease may give characteristic appearances when examined by X-rays. Thus in pulmonary stenosis the shape of the heart resembles a French sabot (*see* p. 296), owing to hypertrophy of the right ventricle, and there may be much enlargement to the right, owing to dilatation of the right auricle. There may be dilatation of the pulmonary artery beyond the obstruction; the reason for this is quite obscure, but it has been several times observed at post-mortems. Both left and right sides of the heart may be enlarged; but, on the other hand, there may be no recognisable change in the shape of the heart.

**Prognosis.**—Congenital malformations are always unfavourable. Cases of severe defect live but a few hours or days; others of slighter degree survive five, ten, or twenty years; and even persons with very ill-developed organs have occasionally reached middle age. In any given case the prognosis must depend upon the evidences of cardiac efficiency in the history of the patients rather than upon any opinion as to the nature of the malformation. A good proportion of cases of patent ductus arteriosus live to thirty or thirty-five years of age.

**Treatment.**—This is entirely palliative. The patient must be kept always thoroughly warm, and protected from exposure to cold and from undue exertion. The symptoms of the later stages must be dealt with as in cases of acquired valvular disease.

## PERICARDITIS

**Ætiology.**—Inflammation of the pericardium may result from a general blood poisoning, or it may occur from direct irritation or infection of the serous sac.

Among the first class of cases, acute rheumatism is its most frequent cause; it occurs in Bright's disease, in pyæmia, in leukæmia, in tuberculosis, in influenza, in general pneumococcal infection, and in other conditions of septicæmia and toxæmia. Its local causes are the growth of cancer nodules into its cavity, the rupture into it of abscesses and hydatid cysts, and the contiguity of a source of infection, such as empyema or pneumonia.

**Anatomical Changes.**—If we take as the type the pericarditis which occurs in the course of acute rheumatism, we find the following changes: In early stages the membrane loses its smooth, glossy surface, and becomes more vascular, so that it is injected with a fine network of vessels. Some shreds of lymph from the exudation of corpuscular elements and fibrin from the blood vessels are next seen, and a complete layer forms upon the pericardium. Ultimately the two opposed surfaces of the sac may be separated by a layer of lymph $\frac{1}{8}$ or $\frac{1}{4}$ inch in thickness, which is sufficiently soft to allow the parietal and visceral membranes to be peeled from one another, and the lymph is often of such a consistence that the separation of the surfaces leaves a curiously honeycombed or reticulate appearance. Generally, at the same time, some serum is formed, of yellow colour, and turbid from corpuscular elements. This may accumulate to a considerable amount and further separate the two layers of the pericardium, while it allows the formation of long, shaggy processes of lymph, stretching from surface to surface. After a time the fluid generally disappears, and the lymph is either itself absorbed, or it becomes organised, and unites the parietal and visceral layers of the sac more or less completely together. In this process new vessels grow in the investing lymph, and fibres of connective tissue are gradually developed. The amount of firm connective tissue thus formed and the completeness of the union effected vary much in different cases; there may be a few fibrous bands crossing the cavity, or a dense layer of tissue $\frac{1}{2}$ inch thick. The mediastinal connective tissue is sometimes involved in inflammation at the same time, forming *mediastino-pericarditis* or *mediastinitis fibrosa* (*see* p 384. *See* also Polyorrhomenitis).

Variations in this process take place. Under certain circumstances, mostly in pyæmia or septicæmia, the fluid contents of the pericardium are pus, instead of serum, constituting *purulent* or *suppurative pericarditis*. This is often secondary to abscess of the cardiac muscle, which is known frequently to result from acute necrosis of the long bones. Sometimes the new-formed vessels in the inflammatory formation rupture, and small petechiæ or larger patches of hæmorrhage cover the surface of the membrane, forming *hæmorrhagic pericarditis*. And, occasionally, tubercles are formed both in the new tissue and in the original membrane covering the heart's surface; this is known as *tuberculous pericarditis*, and forms part of a general tuberculosis.

The micro-organisms of pericarditis vary with its origin. Streptococci, staphylococci, pneumococci, and tubercle bacilli have been most often found. Poynton and Paine found their rheumatic diplococci in the pericarditis of rheumatism.

*Effect on Circulation.*—Cohnheim's animal experiments show that the effect on the circulation depends on the tension in the pericardial sac. If fluid is injected slowly when the pressure reaches a definite point the arterial pressure falls, and the venous pressure rises. This is an expression of a diminished efficiency on the part of the heart as a pump, the output of blood at each beat being diminished. The higher the tension in the pericardium, the smaller the output.

**Physical Signs.**—Since pericarditis so frequently arises in the course of some infectious disease, like rheumatism, its symptoms may be entirely masked by those of the disease which it accompanies, and its presence may be only revealed by the alteration in the heart sounds, and other physical signs which it produces. These, however, are generally characteristic. In the early stages, a slight shuffling sound is heard in addition to, though partly obscuring, the normal sounds. The shuffling consists of two sounds occurring during systole and diastole respectively, but not always absolutely synchronous with the first and second sounds; it is heard over the præcordial region, often first at the base, later over the whole of the area corresponding to the anterior surface of the heart and pericardium. Frequently this *pericardial rub* has a triple character, like the pace of a cantering horse. Sometimes the loudness of the sound may be modified by the pressure of the stethoscope. After a short time the sound becomes louder

and harsher, resembling the friction of hard, rough surfaces upon one another, and when it has reached this stage the friction can often be felt by the hand placed over the præcordial region.

If liquid is effused into the pericardium, as is frequently the case, the præcordial dulness is increased. It extends upwards to the upper border of the third rib, the upper border of the second rib, or even to the clavicle; towards the right for 1 inch or more beyond the sternum; and towards the left it may reach right into the axilla. The præcordial dulness has a more or less triangular shape, with its broad base upon the diaphragm, and a rounded apex at the upper part of the sternum and the left upper intercostal spaces. In this extension of præcordial dulness, the small area of resonance in the fifth right intercostal space normally corresponding to the sharp angle between the convex border of the right auricle and the upper surface of the diaphragm becomes obliterated. This is called *Rotch's sign;* but its diagnostic importance is somewhat doubtful.

As the liquid increases, the impulse of the heart becomes diffuse. An important distinction must here be mentioned between pericarditis and pleurisy in the effect of effusion of liquid on the occurrence of a friction sound. In pleurisy the effusion of liquid results in the disappearance of the pleuritic friction sound. In pericarditis the friction sound persists commonly throughout the illness, even to the period of greatest distension of the sac, and during the subsequent absorption of the liquid. This is probably because the fluid collects chiefly behind the heart, as the patient lies on his back, leaving the two inflamed layers of pericardium rubbing against one another in front.

A pericardial effusion often compresses the base of the left lung, causing a dull note on percussion and bronchial breathing on auscultation.

Another result of pericarditis in some cases is inhibition of the action of the diaphragm. Either abdominal respiratory movements cease, or there is a little recession during inspiration, with upward movement of the higher abdominal viscera and of the heart, collapse of the bases of the lungs, and it may be distension of the stomach and colon.

**Symptoms.**—The local symptoms which may accompany pericarditis and pericardial effusion are pain, anxiety or distress at the præcordia, tenderness on pressure over that region, shortness of breath, with shallow respirations and short hacking cough. The pulse may not at first be much affected, but it tends soon to be faster and fuller, and in the later stages of effusion to become feebler, and even fluttering and irregular. Occurring in the course of a febrile disease like rheumatism, pericarditis may not notably add to the existing pyrexia, but with its rapid onset occasionally there is considerable elevation of temperature—for instance, to 105° or 106°; and in other cases it may be accompanied by the usual conditions of pyrexia, loss of appetite, dry tongue, thirst, and scanty urine.

In the worst cases the cardiac feebleness increases, the pulse becomes irregular and fluttering, or may take the form of the *pulsus paradoxus* (*see* p. 300), præcordial anxiety is severe, and the face becomes drawn and pinched; and the dyspnœa is no doubt aggravated by the pressure of the distended pericardial sac upon the left lung. Ultimately the nervous system fails, and delirium, jactitation, convulsion, or coma ends the scene. But in the majority of instances the symptoms gradually subside; the dulness diminishes from above downwards, while the rub often remains till a late stage. In many cases, no doubt, adhesion of more or less of the pericardial surface takes place.

The changes of pericarditis occur rapidly, effusion may reach its height in two or three days, and subsidence may be well established in three or four more.

Suppurative, tuberculous, and hæmorrhagic pericarditis are not essentially different in their symptoms and physical signs.

**Diagnosis.**—Under ordinary circumstances this presents no difficulties, the double or triple friction sound being very distinctive. A double pericardial rub may, however, sometimes be simulated by a *double aortic murmur*, and if there

# PLATE VII.

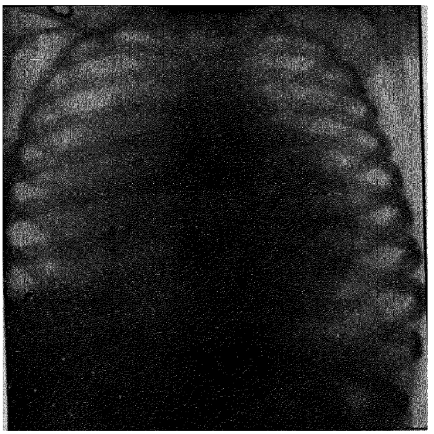


FIG. 1.—Radiogram of Chest in case of Pericardial Effusion. There is a large pear-shaped shadow, with a well-defined margin which does not show pulsation.

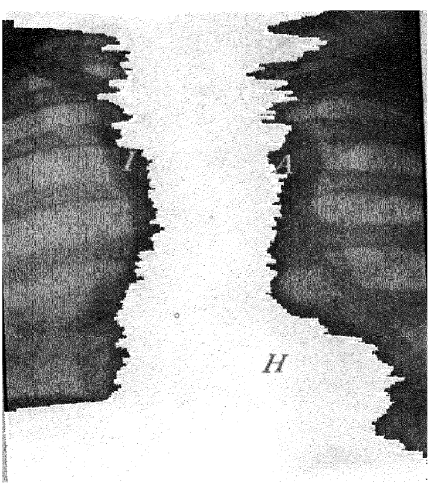



FIG. 2.—Skiagram of an Aneurysm of the Innominate Artery (*I*) with General Dilatation of the Aortic Arch (*A*). (*H*) the heart. (Taken by Dr. A C. Jordan.)

[*To face p.* 358.

height. If a considerable quantity remains for some time, iodide of potassium may be given. If the liquid is in excessive amount and threatens a fatal result, exploration by means of a needle and syringe may be carried out. The best place is in the axilla, where the percussion note is completely dull. This will be beyond the apex of the heart, so that the risk of wounding it will be slight. If the liquid is purulent, a free opening may be made by removing the fifth costal cartilage, and the sac can be washed out and drained.

## ADHERENT PERICARDIUM

Reference has been already made to this condition as arising from pericarditis. The degree to which the two surfaces may adhere varies much in different cases; there may be merely a few filaments running from the surface of the heart to the parietal pericardium, or there may be complete union of the pericardial sac to the surface of the organ, and every intermediate condition occurs. When the union is complete, the tissue uniting the two surfaces may form only a thin layer; or it is a dense, firm, fibrous, more or less vascular coat, $\frac{1}{4}$ inch or even $\frac{1}{2}$ inch in thickness. In rare cases, also, calcareous matter is deposited in the adhesions, so that a complete investment by it may take place. Although the serous sac of the pericardium seems especially devised to allow the free movement of the heart, the simple adhesion of the two layers is not necessarily followed by any ill effects upon the form and size of the heart, and in a certain proportion of cases the heart has its normal size. But in others hypertrophy or dilatation is present. In many of these last valvular disease is associated with the pericarditis, and sufficiently accounts for the changes in the heart walls. If the pericardial adhesion is very extensive and dense, dilatation and hypertrophy may occur without being accounted for by any valvular disease, and it is here probable that the muscular substance of the ventricle has been injured by the occurrence of myocarditis at the same time as the pericarditis. In some cases there is not only obliteration of the pericardial sac, but the external surface is firmly fixed to the surrounding pleura and to the sternum, and the adjacent pleural layers are also adherent (*mediastinitis*).

**Symptoms and Physical Signs.**—The symptoms associated with adhesion of the heart to its pericardial sac are for the most part, if not entirely, due to dilatation and hypertrophy of the cardiac walls: cardiac pain, palpitation, and dyspnœa may be especially mentioned. Physical signs cannot be relied upon to reveal the presence of the adhesion itself; it has been often found post mortem when unsuspected; and it may only be inferred from the knowledge that an acute pericarditis has previously occurred. But when the more extensive external adhesions are also present, one or more of the following physical signs may be recognised: (1) systolic retraction at the point corresponding to the apex of the heart; (2) systolic retraction of the lower end of the sternum; (3) systolic retraction of the third, fourth, and fifth intercostal spaces to the left of the sternum; (4) systolic retraction of the lower ribs at the side or back of the left chest (J. Broadbent); (5) a diastolic rebound or shock after the systolic retraction at the apex; (6) absence of alteration in the præcordial dulness, and in the position and force of the impulse during respiratory movements; (7) sudden collapse of the veins of the neck during ventricular diastole (Friedreich); (8) failure of the sternum to advance during inspiration (Wenckebach). These are not very reliable; certainly systolic retraction of intercostal spaces is not peculiar to adherent pericardium: much less is systolic recession of the epigastrium. Some of the others are difficult to verify in particular cases, and others are not constantly present. Since adherent pericardium so often occurs in company with valvular and myocardial lesions, the physical signs as well as the symptoms of the latter are apt to be credited to the former. But a systolic, and sometimes

even a pre-systolic, murmur may occur without valvular disease when the heart is dilated as a result of adherent pericardium. Adhesions may sometimes be inferred when the patient is suffering from evidence of failure of the right ventricle, such as dyspnœa, dropsy, enlargement of the liver, and albuminuria, without any obvious cause for the right-sided failure, such as mitral or pulmonary disease. This diagnosis is still more reasonable if there are, or have been, pleurisy, pleuritic effusion or adhesions on one or both sides, as this increases the probability of a combined pleural, pericardial, and mediastinal inflammation (*mediastinitis*) having been present.

X-rays may give valuable indications of adherent pericardium. They consist in alterations of the normal movement and shape of the heart on breathing deeply or leaning to one side, and alterations in the movement of the central part of the diaphragm on breathing, due to adhesions with the pericardium and mediastinum.

The **Prognosis** and **Treatment** of pericardial adhesions must be considered chiefly in reference to the changes in the structure and functions of the heart which result from them (*see* p. 346). In exceptional cases, with pronounced evidence of external adhesions, other measures may be taken (*see* Mediastinitis)

## PERICARDIAL EFFUSIONS

### HYDROPERICARDIUM

This term is intended to denote the presence of an excess of serum in the pericardial sac, and is generally used to distinguish the passive secretion of dropsy from that of inflammatory effusions already described under Pericarditis. The pericardium naturally contains a very small quantity of serum, and after death from any cause it is common to find a few drachms of pale yellow fluid in it. When this reaches 5 or 6 ounces or more, it constitutes dropsy of the pericardium, or hydropericardium. The causes of serous effusion, apart from inflammation, are those of general dropsy, such as Bright's disease, and such local interference with the venous circulation of the pericardium as valvular disease of the heart itself, chronic lung disease, and pressure of growths upon the veins which return blood from the pericardial surfaces. The liquid contained in the sac resembles that of dropsical effusion into the other serous cavities, being pale yellow, or more or less pink from exudation of blood-colouring matter, with a small quantity of fibrinogen and from 1 to 3 per cent. of albumin.

The **Physical Signs** of hydropericardium are the same as those of effusion in pericarditis. As a rule, no special **Treatment** directed to the pericardium is required where the condition forms part of a general dropsy, or where it results from local interference with the circulation; the general dropsy or the valvular disease must be dealt with. In rare cases the effusion may be so rapid or abundant as to require paracentesis of the pericardium.

### PNEUMOPERICARDIUM AND PNEUMO-HYDROPERICARDIUM

These signify respectively the presence of gas, and the presence of gas and liquid together, in the pericardium. Gas in conjunction with liquid has been observed as a result (1) of decomposition of the liquid of pericarditis, and (2) of the communication of the pericardial sac with air-containing cavities. This communication may be traumatic, as in the case of a juggler who, in attempting to swallow a blunt sword, perforated the pericardium from the œsophagus; as in the case recorded by Flint, where the pericardium was punctured by a stab through the pleura; and after the operation of paracentesis pericardii. Or the communication may be effected by disease; and cases are on record of cancer of the œsophagus ulcerating into the pericardium, of a phthisical cavity opening into it,

and of a hepatic abscess communicating at the same time with the pericardium and with the stomach. Gas can never be observed alone in the pericardium, as its entrance from without is almost immediately followed by pericarditis with liquid effusion.

The **Physical Signs** of pneumo-hydropericardium are resonance on percussion over the præcordial area and splashing, churning, or gurgling sounds, synchronous with the movements of the heart.

### HÆMOPERICARDIUM

In slighter degrees, the effusion of blood into the pericardium occurs in so-called hæmorrhagic pericarditis, from the rupture of the new-formed vessels; but larger quantities, when not directly traumatic, result from rupture of the myocardium, of an aneurysmal sac, or of vessels in a cancerous growth. Scurvy and allied conditions may also give rise to pericardial hæmorrhage.

**Symptoms.**—When sudden effusion of blood into the pericardium takes place, the patient is seized with more or less oppression of the chest, pallor, syncope, unconsciousness and death in quick succession; or with the same pallor and with collapse, feeble pulse, dyspnœa, and orthopnœa he may remain for twenty-four or thirty-six hours before the fatal termination; or presumably, with a less degree of hæmorrhage, death may be still further delayed, and a pericarditis may develop which contributes to the final result. Walshe refers to cases, probably of a scorbutic nature, or at any rate not dependent on rupture of aneurysms, or of the heart itself, in which recovery has taken place.

The **Physical Signs** are those of a large pericardial effusion, extensive præcordial dulness, and enfeeblement or absence of the heart sounds. The **Diagnosis** would be assisted by a knowledge of the previous existence of aneurysm, or attacks of angina pectoris.

**Treatment.**—Absolute rest and judicious use of stimulants would give the only chance.

## ANGINA PECTORIS

This name is given to an intense pain beneath the sternum, which comes on with great suddenness, and occasionally proves fatal.

**Ætiology.**—It may occur in boyhood, but it is uncommon before the age of thirty, increases in frequency with every year, and is most common between the ages of fifty and seventy-five. It is more frequent in men than in women, in the proportion of about four to one. As a large majority of the cases present some lesion of the heart or arteries, the conditions which lead up to these changes may be regarded as predisposing causes of angina pectoris, and especially obesity, sedentary occupations, the gouty habit, and interstitial nephritis. Heredity also seems to have an influence. The immediately exciting causes are mostly such as may be supposed to act prejudicially upon the functions of the heart, whether through its muscular or nervous apparatus. These are: (1) Physical exercise, especially going uphill or against the wind, and, later on, even the mildest forms of exertion; (2) a large meal; very often these two factors are combined; (3) emotional excitement, whether depressing or exhilarating, and mental strain; and (4) exposure to cold. The last is important, since undressing at night in a cold room may bring on an attack in bed. Occasionally the attack begins during sleep.

**Pathology.**—When death has taken place in an attack, the heart has generally been found relaxed, with its cavities full of blood. In the majority of cases, some disease of the heart or aorta has been found, and mostly of the following kinds: myocarditis or fatty degeneration of the myocardium; syphilitic aortitis, or atheroma, or dilatation of the aorta, or aneurysm; atheroma or calci-

fication or shrinkage of the aortic valves; and arterio-sclerosis or calcareous deposit in the coronary arteries, or their obliteration from endarteritis or thrombosis. Coronary embolism may cause death rapidly, with the most severe anginal symptoms. Disease of the mitral orifice, on the other hand, is rarely the sole cause of angina pectoris. In some cases of death from angina the heart muscle and coronary arteries have been found to be perfectly healthy.

Allbutt has brought forward the view that the pain of angina pectoris is due to tension of the inflamed coats and outer investments of the aorta (aortitis) above the sigmoid valves, and that death is caused by vagal inhibition of the heart. He regards the myocardial changes and coronary sclerosis so commonly found as the cause of death, but not the cause of the angina itself (**21**). Mackenzie's view is that angina depends on the cardiac muscle (**22**). In this connection it may be noted that the usual pain in mitral stenosis, which arises in the left auricle, occupies a band on the left side of the chest, extending from the nipple to below the ensiform cartilage; the sensory nerves from the skin of this area pass to the sixth and seventh dorsal segments of the spinal cord. Pain arising in the ventricle and aorta, on the other hand, is felt higher up in the chest, over an area corresponding to the higher dorsal segments, because, in the embryonic cardiac tube, the ventricular part lies anterior to the auricular part, and becomes bent over and downwards later on (**23**). The theory that anginal pain comes from the ventricle is the most satisfactory in the opinion of the present writer, because it brings this type of pain into line with pain produced by other muscular organs. The fact that pain is sometimes caused by a healthy cardiac muscle need cause no more difficulty than the pain which is often produced by healthy gastric muscle when there is pyloric stenosis. Cardiac pain is due to overwork of the cardiac muscle, and it seems reasonable to suppose that the muscular fibres must be sufficiently healthy to be able to produce it; they must be capable of hard work. The substernal pain felt by healthy but untrained persons during a hard game of football may be of similar origin. Angina may occur with fibroid heart because, though healthy, the muscle fibres are greatly reduced in number by replacement with fibrous tissue. Angina may occur, while there is no breathlessness on exertion, because the muscle, though overworked, still keeps up the circulation. When the circulation fails angina may disappear, to reappear when the circulation is restored by treatment. However, the commonest condition is, undoubtedly, the simultaneous occurrence of both pain and breathlessness after exertion. On the analogy of œsophageal and gastric pain (*see* p. 410), cardiac pain would be due to a tonelessness and consequent stretching of the muscle, and amyl nitrite, which relieves all but the severest type of pain in a few seconds, may possibly act by stimulating the muscle to contract down on its contents. It certainly increases the pulse rate, and though it causes a peripheral vaso-dilatation, with marked flushing of the face, this cannot always be the cause of the relief, because it often happens that the relief occurs before the blood pressure falls. In a case of mitral stenosis and regurgitation, with pain beneath the nipple, amyl nitrite also caused relief. In one case an electro-cardiogram taken during the attack showed temporary delay in the right branch of the A.V. bundle, which disappeared after the end of the attack (Bousfield). This suggests that the muscle is responsible for the pain, though it can be accounted for by vagal action on Allbutt's theory.

**Symptoms.**—The patient is seized quite suddenly with acute pain in the front of the chest, situated beneath the upper or lower part of the sternum, or rather to the left-hand side of it, but not over the heart itself. The pain radiates thence to the left side and back, or through to the scapula; up to the left shoulder, and down the left arm to the hand; or less frequently the pain is on the right of the sternum and radiates to the right shoulder, arm, and hand. The pain may occur simultaneously on the right and left side. It may ascend on either side of the neck as high as the scalp, and this is explained by the fact that the trigeminal

nerve is the sensory counterpart of the afferent bago-glosso-pharyngeal nerve from the heart and so becomes the seat of "referred pain" (**23**). The pain may be felt in the throat. The pain in the chest is described as "tearing" or "piercing," or "burning as hot as fire," or "constricting." Tingling or numbness may accompany the pain in the arms or fingers. There is considerable variety in the onset of the pain in different cases: thus it may begin in the arm or arms and spread up to the chest; or it may begin in the upper abdomen (epigastric angina) or lower down in the abdomen (angina abdominis). In one such case the pain was brought on by exertion, was most severe in the umbilical region to which it was at first confined; but it gradually increased in severity, and spread all over the front and back of the chest. During the attack the patient's expression is anxious or solemn; he is obliged to stop if he is walking; he becomes covered with clammy perspiration; the saliva may be increased; there may be a sense of impending death. The pulse is usually unaltered, but it may be slowed, or irregularities such as extra systoles may be noticed; it becomes quick towards the end of a fatal attack. There is no characteristic alteration in the blood pressure; in some cases it is raised by 20 mm. or so. After lasting a few seconds or minutes, the pain quickly passes off, but it may occur again frequently in the course of a few hours, or it may not be experienced again for several months or years. Angina may be fatal in the first and only attack. Between the attacks, and lasting for weeks after the severe attacks have finally ceased, tenderness may be experienced on pressing various spots on the chest wall and arms. These spots vary from day to day. At the same time the patient's attention may be drawn to sensations of fulness or compression inside the chest, which should be regarded as warning signs that the severe pains may come on unless care is taken.

The attack is often accompanied by a feeling of fulness in the stomach, particularly if it is brought on by exercise after a meal, and it may be relieved if wind is successfully eructated. The frequency of this symptom has led to the view (**24**), which however few people accept, that the primary cause of the trouble is distension of the stomach or œsophagus with wind. In one observed case (**25**) attempts at eructation regularly relieved the pain, but it was found by X-rays that air had entered the stomach. There is, however, a very close reflex connection between the seat of angina and the upper alimentary tract, since a full meal will precipitate an attack, and some of the pains felt during anginal attacks are produced in the œsophagus, and may be momentarily accentuated or relieved by swallowing. This is to be explained by the peristaltic wave which passes down the œsophagus after swallowing (**25**). John Hunter made a clinical observation on this subject in his own case. The term "pseudo-angina" may perhaps be used for such pains. Angina is sometimes associated with Raynaud's disease.

Slight attacks of angina (angina minor) are commonly met with. The patient, as the result of exercise or of exposure to cold, may feel some substernal pain for a few seconds, and be obliged to remain still. These attacks have sometimes incorrectly been called "pseudo-angina"; but this term should not be used in this connection, as it tends to mask the gravity of the condition.

**Diagnosis.**—The character of the pain, its occurrence as the result of exertion, its relief by amyl nitrite, and the evidence of a cardiac or arterial lesion (valvular disease or arterio-sclerosis), are generally conclusive. It must be distinguished from cardiac pain felt over the præcordia, which is so common an accompaniment of a heart failing from valvular disease or myocardial degeneration. It must be distinguished from neuralgic pains, especially in neurotic women. In this case the pain often occurs during rest, lasts much longer than angina, and may be accompanied by tumultuous action of the heart and palpitation. The milder forms of angina are frequently mistaken for indigestion, or gastritis; and this is partly accounted for by the fact that an attack often occurs when the patient gets about after a meal. Tobacco angina presents rather similar features to angina pectoris. Very severe pain may be caused by sudden

embolism of a coronary artery, and there is often dyspnœa present as well, presumably due to acute circulatory failure. Finally, acute distension of the stomach has caused similar symptoms, and this has been relieved by passing a tube into the stomach.

**Prognosis.**—Death may occur during, or shortly after, an anginal attack, or during sleep, or suddenly. It may occur from heart failure or independent causes. The outlook depends on how well the case responds to treatment. A patient may have an attack and yet with proper care live for years afterwards. Unfavourable signs are a poor response to exercise, pulsus alternans, and certain alterations in the electro-cardiograms (*see* p. 303), which all help to indicate that the functional power of the heart is below par.

**Treatment.**—The patient must remain quite still during an attack. The most efficacious remedy for an attack of angina is nitrite of amyl. From 3 to 5 minims are contained within a small glass capsule, covered with linen: the capsule is crushed between the finger and thumb, and the vapour is inhaled freely. The face flushes, the cranial vessels throb, and the pain is often relieved at once. The dose may have to be repeated. A similar effect may be obtained by the administration of nitro-glycerine (trinitrin) internally, but its action is not so rapid. One may begin with $\frac{1}{100}$ minim, given in the form of tabella, or with a 1 per cent. solution in alcohol, of which 1 minim in a little water is the required dose. Much larger quantities may have to be given, equivalent to two, three, five, or ten-hundredths of a minim. The first administration of even small doses of nitro-glycerine is often followed by a throbbing headache, but after a time tolerance is established, and the larger doses can be borne. Sodium nitrite ($2\frac{1}{2}$ grains in tabella) and erythrol tetranitrate (1 grain in 1 drachm absolute alcohol suitably diluted) are also good vaso-dilators. If these measures fail, the hypodermic injection of morphia, or even the inhalation of chloroform, may be used; and much collapse will require brandy or ether. Morphia is specially useful when the attacks are precipitated by excitement or mental anxiety. Sal volatile diluted with an equal volume of water may be useful when the attacks are precipitated by a full stomach or are accompanied by flatulence. Allbutt advises the subcutaneous injection of atropine if the attack is severe, so as to paralyse the vagus and prevent sudden arrest of the heart.

When angina has once declared itself in a patient, absolute rest in bed for some weeks and freedom from anxiety and excitement will be required on the theory that the condition is due to overwork of the heart. Food should be given in small amounts at a time. On getting about again the patient's life must be altered so as to avoid those factors—excessive muscular exertion, etc.—that have caused the attack. In regulating his life he will be helped by the premonitory sensations of fulness and compression in the chest which will appear if he is again beginning to do too much. The attacks should be arrested in the first place by drugs; but later on it is probably best not to use nitrites prophylactically, as they may remove the patient's warning signs, so that he may be led to overwork his heart again, and this may lead to circulatory failure. Amyl nitrite should be carried for an emergency. Smoking in excess must be stopped. In severe cases, where the reserve of the heart is low, such treatment may fail to ward off attacks, and these will still come on whenever the patient gets about at all. In such a case nitro-glycerine will be valuable; the dose may be $\frac{1}{100}$ minim three or four times daily, gradually increased to $\frac{1}{20}$ or $\frac{1}{10}$. Iodide of potassium (5 to 30 grains) is also beneficial in some cases, especially where syphilitic aortitis is suspected. Arsenic and iron may be used as general tonics. The exposure of the body surface to light from the electric arc has sometimes warded off attacks. Ammonium bromide in doses of 10 to 20 grains is useful as a mental sedative. Surgical treatment, *i.e.* division of the depressor nerve, etc., is not to be recommended in the present state of our knowledge.

# DISEASES OF THE BLOOD VESSELS

The diseases of arteries are mainly comprised in inflammation and degeneration, and the mechanical results of those lesions. *Amyloid* or *lardaceous* degeneration is dealt with elsewhere (*see* p. 489). Inflammation of the veins, or phlebitis, and thrombosis and embolism will be considered in this section.

## ARTERITIS

*Acute and Subacute Local Arteritis.*—This may be caused by the direct spread of some neighbouring infection, as when the artery lies in the neighbourhood of a suppurative process. In a tuberculous lung the arteries are directly invaded from without by tubercle. The artery may be the subject of embolism; the outer or inner coat in this case is infected with organisms; inflammatory changes take place which result in softening, yielding (aneurysm), or perforation (with hæmorrhage) of the arterial wall. In malignant endocarditis the inner coat of the aorta is sometimes infected from the valves, and undergoes the same pathological processes.

*Endarteritis.*—This is a term usually applied to small arteries. There is a proliferation of the subendothelial connective tissue of the intima, which projects into the lumen, and may block it completely. It is a normal process in certain cases, as when the uterine arteries atrophy after childbirth. It is particularly characteristic of all stages of syphilis, and affects the arterioles in the primary chancre as well as in later manifestations. When the process is well marked, it is called endarteritis obliterans. This is of special importance in the case of the cerebral arteries, as it may lead to cerebral thrombosis. Endarteritis also occurs in tuberculosis and certain other infections.

*Syphilitic Arteritis.*—This term is usually applied to syphilis of the aorta and other large arteries. The whole thickness of the wall is attacked, but in all probability the process starts in the adventitia and spreads inwards. Endarteritis of the vasa vasorum is present, but there is also destruction of the elastic and muscular tissue of the aorta with replacement by white fibrous tissue. Focal aggregations of plasma cells and lymphocytes are seen round the small vessels in the coats of the artery, and these vessels extend further in towards the intima than normal. The intima remains intact, but the results of the arteritis are seen as white scars beneath it, and since these are made up of fibrous tissue, they readily stretch under the arterial pressure, giving rise to aneurysms.

*Thrombo-angiitis Obliterans.*—This affection occurs chiefly among adult Jews of young or early middle age, especially those from Eastern Europe. It is not due to syphilis. Cigarette smoking may be partly responsible. The blood pressure is not raised. The arteries are found to be thrombosed; but the clot is partially organised, new vascular channels being formed, so that nutrition of the part supplied, though defective, is still possible. Intermittent claudication (*see* p. 382) and erythromelalgia (*see* p. 383) are characteristic symptoms. There is absence of pulsation in the arteries, notably the dorsalis pedis artery (**31**). Ischæmic gangrene may supervene.

*Atheroma* (*Nodular Sclerosis*).—This is a patchy degeneration of arteries involving a local proliferation of the intima, with degeneration of the underlying media. It affects arteries of all sizes. It is probable that the primary change takes place in the intima in some cases, and in the media in others. (1) Fatty deposits in the intima unassociated with any medial change are very common in the aorta and main arteries of young people after acute illnesses, and these may develop into ordinary atheroma. In the earliest stage the superficial cells of the intima proliferate, and these are either of endothelial or connective tissue origin.

The intimal thickening is permeated by elastic fibrils, and later on fatty degeneration occurs, possibly due to sufficient oxygen not being able to diffuse in through the thickening. (2) In elderly people patches of degeneration in the media are sometimes seen alone. If these become more extensive, the deeper layers of the intima may proliferate in order to fill up the gap in the underlying media. On this hypothesis, the patches of atheroma which are seen to project into the lumen of the artery post mortem really lie flush with the surface during life.

The changes that take place in atheroma are characteristic. Subsequent to fatty degeneration the fat slowly becomes converted into calcium soap, and eventually the fatty acid is destroyed and calcium carbonate and phosphate remain behind. At any stage in this process the mass may break down, the superficial layers being discharged into the circulation, and leaving an *atheromatous ulcer*, which may be ragged and soft, or may have a smooth, hard calcareous base. There is evidence that high blood pressure leads to atheroma, particularly if it is intermittent, and there is a belief that it occurs particularly among those who carry out laborious occupations, which cause frequent rises in the arterial pressure, owing to muscular exertion. At the same time infection probably plays a part in its production, presumably owing to the presence of toxins in the circulation, and among these the most important is syphilis, which may produce a condition of the aorta indistinguishable to the naked eye from the atheroma due to other causes.

*Diffuse Hyperplastic Sclerosis* (*Arterio-capillary Fibrosis*).—This change occurs most commonly in the kidneys, and next in the spleen and other organs, including the brain, pancreas, liver, suprarenal glands, and less often the stomach and intestines, but not the heart or skeletal muscles. It affects the arterioles and smaller arteries. In the arterioles there is primarily a proliferation of endothelial cells which degenerate to form a hyaline swelling of the intima, and subsequently there is fatty degeneration, and there is also hypertrophy of the media. The vessel is tortuous and the lumen is very small; but this latter appearance is probably due to constriction of the vessel after death. In the smaller arteries there is also hyaline swelling of the intima and hypertrophy of the media, but there is no fatty degeneration. Diffuse hyperplastic sclerosis has rather a patchy distribution in organs, and in the kidney the glomerular tufts and afferent arterioles are chiefly affected. The cause of the condition is not known, but arguments have been brought forward for regarding it as inflammatory in nature and possibly due to bacterial toxins (**26**). It occurs in people from thirty to fifty; it is associated with a high blood pressure and hypertrophied heart, which are to be regarded as compensating for the increased peripheral resistance due to the arterial disease. It is regarded as being the cause of *primary contracted kidney* (*see* p. 630); but it also occurs in the kidney of chronic Bright's disease (secondary contracted kidney), where it is definitely secondary to the renal changes. Although some bacterial toxin may be the primary cause of diffuse hyperplastic sclerosis, the hypertrophy of the media may well be the natural response to a permanently raised blood pressure, because the smaller arteries are kept contracted, while the heart beats more powerfully than usual, and the contraction leads on to hypertrophy.

*Senile Degeneration.*—This degeneration, which occurs in people over fifty, affects the middle coat of larger and smaller arteries. It is particularly obvious in such arteries as the radial and those at the base of the brain, but it is as general in its distribution as diffuse hyperplastic sclerosis. There is first of all fatty degeneration and atrophy of muscle fibres, and later there is fibrosis and calcareous deposits are laid down, and these may extend to the deeper layers of the intima. The vessels become tortuous. In an advanced stage the artery may feel like the stem of a clay pipe; microscopically, fatty changes are seen in the media of quite small arteries. Senile degeneration is usually accompanied by atheroma. The blood pressure is not raised in senile degeneration.

*Mönckeberg's Sclerosis.*—Like senile degeneration, this is a primary affection of the media characterised by calcification; it is like an extreme form of senile degeneration, because the intima hardly proliferates at all. But it does not occur in old people; it is chronic wasting disease that causes it, such as cancer, phthisis, heart disease, and other chronic infections.

*Arterio-sclerosis.*—This term means hardness of arteries. It is used at present in rather a vague way, and so it is best to keep the term to indicate all the various chronic pathological conditions in arteries, just described, which lead to hardness of their walls. Hence arterio-sclerosis embraces the various syphilitic diseases of arteries, as well as diffuse hyperplastic sclerosis, atheroma, senile degeneration and Mönckeberg's sclerosis. It is a term of great value in clinical diagnosis, because it is at present impossible in most cases to differentiate the various arterial degenerations by the bedside.

**Ætiology of Arterio-sclerosis.**—Something has already been said on this subject under the previous headings. Probably it is primarily due to infection or intoxication. In syphilitic arteritis the nature of the virus is known; but syphilis may cause the other types of arterio-sclerosis as well. If arterio-sclerosis is due to infection or intoxication, it is possible that the different types are not so much due to different viruses as to the different soils in which the virus acts. Thus in young people the inflammatory changes result in great internal proliferation (diffuse hyperplastic sclerosis), while in the elderly or cachectic such an intense reaction is impossible, and the blood pressure remains low (senile degeneration and Mönckeberg's sclerosis) (**26**). The various factors that play a part in the ætiology are, besides syphilis already mentioned, excessive muscular exertion, over-eating in particular of protein foods, gout, alcohol and lead poisoning, malaria and other acute infections, particularly typhoid, hypothyroidism, Bright's disease, and possibly intestinal intoxication. Arterio-sclerosis may be associated with diabetes in elderly people, but in this case it is possible that the diabetes is secondary, owing to the failure of blood supply to the pancreas, because the vessels are sclerotic.

**Symptoms.**—Allbutt has pointed out that there is no tendency for primary arterial changes, such as atheroma and senile degeneration, to cause high blood pressures, and that, as a matter of fact, in these cases the pressure is not raised, nor the heart hypertrophied. On the other hand, diffuse hyperplastic sclerosis is associated with a high blood pressure, so that, if all cases with hard arteries are grouped together under the term *arterio-sclerosis*, in some the blood pressure will be normal, in others it will be raised.

In the earlier stages the artery is palpable, and can be rolled under the fingers when all the blood has been squeezed out of it by pressure from the fingers. The artery feels thick. In the later stages the wall may feel hard from calcification. The transverse diameter may be increased, and the vessel may become tortuous, owing to lengthening of the artery. Pulsation is often much diminished, and sometimes is absent altogether; thrombosis may occur. Since the velocity of the pulse wave increases with the rigidity of the artery, the determination of the former by means of the "hot wire" sphygmograph should indicate the amount of arterio-sclerosis in a given case (**30**).

Ophthalmoscopic examination often provides a valuable means of estimating the degree of arterio-sclerosis. In particular, it is possible to infer from the appearance of the retinal arteries what is the condition of the cerebral arteries. The following description is largely based on a series of cases, in which almost without exception the blood pressure was high (**27**), and hence it cannot be applied to Allbutt's senile or "decrescent" group with pipe-stem radial arteries and normal or low blood pressure. In an early stage the changes are confined to the arteries. In general, the lumen is reduced in size when compared with the veins. The walls of the retinal arteries normally reflect the light to some extent, and this is seen as a bright streak along the middle of the vessel. In

arterio-sclerosis this reflection is increased owing to the thickness of the walls, and the smaller arteries appear burnished like *copper wire*, and later on, as the opacity of the wall increases, they look bright like *silver wire*. This bright streak is often irregular and has a dotted appearance. The arteries are tortuous, but owing to extreme variability of the normal artery in this respect this feature is of no value in diagnosis; their lumen is irregular; they are often greatly reduced in size, sometimes looking like fibrous threads. Sometimes they are of normal width, but are sheathed in places by white plaque-like deposits which look like pieces of pipe stem; these may disappear after a time.

Characteristic appearances are seen at the arterio-venous crossings. The line of the vein is displaced if it crosses the artery obliquely, so that it lies along the artery for a short distance on each side of the crossing. When the artery crosses in front of the vein, the latter seems to disappear at the crossing, because it becomes hidden behind the thick walls of the artery. The veins are pressed upon, so that there is a swelling of the vein on the distal side of the crossing, but this appearance, called "banking," is by no means very common (*see* Plate VIII.).

The retinal appearances of arterio-sclerosis (*arterio-sclerotic retinitis*) are also met with in chronic interstitial nephritis. Small flame-shaped areas may be seen, due to minute hæmorrhages in the nerve fibre layer; when present in the deeper layers of the retinæ the hæmorrhages are roughly circular. In a later stage there may be small bright spots with sharply defined edges scattered about, very often in the macular region, round which they may form a rough *star*-figure, or they may lie between the macula and the optic disc as a fan-shaped figure. They may be grouped round the radicals of the retinal veins, but more often they are scattered irregularly about. Often a large number are grouped together so closely as to resemble a piece of mosaic. Histologically these patches consist of round or oval masses of hyaline exudate in the internuclear layer of the retina. It is perhaps possible to regard them as of essentially the same nature as white infarcts due to blocking of the smaller branches of the retinal arteries.

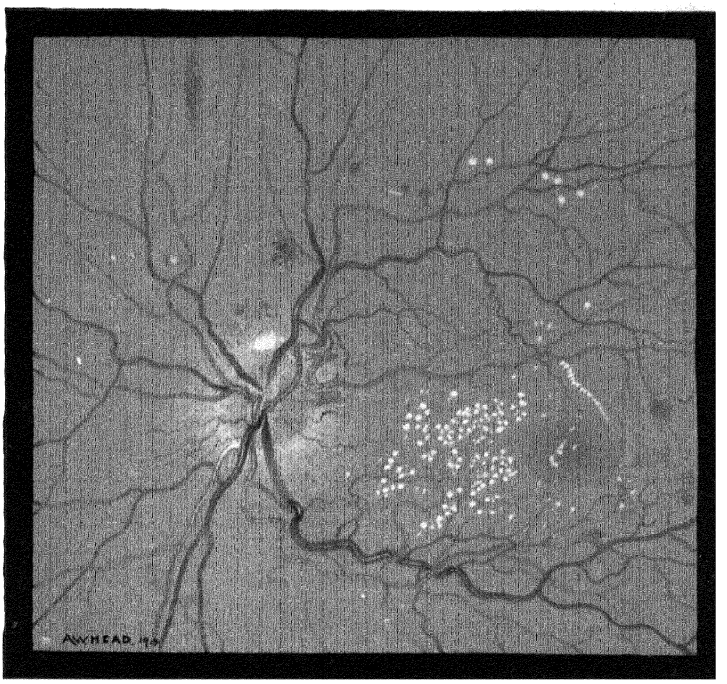
Probably, in the majority of cases, the retinal changes persist until the patient's death; but improvement may take place, effused blood being absorbed, and even the spots of degeneration disappearing. In the earlier stages there is no loss of vision. Gradual impairment of vision may be due to degeneration of the nerve elements in the retina from defective blood supply. The fields of vision may become restricted. Optic atrophy may be seen and may be preceded by some slight swelling of the disc. Sudden temporary loss of vision (*amaurosis fugax*) has been attributed to spasm of the thickened retinal arteries. Sudden permanent blindness, quite painless, may result from thrombosis of the central retinal artery. The retina appears pale; the macula shows up in contrast as a cherry-red spot. The blood column in the vessels is broken up into small blocks of red cells with intervening clear spaces of plasma (cattle-truck appearance).

In arterio-sclerosis the coronary arteries are very commonly atheromatous, so that the nutrition of the heart muscle is affected, leading to myocardial degeneration. Consequently symptoms of early cardiac failure may be present—exhaustion, breathlessness, and pain on exertion.

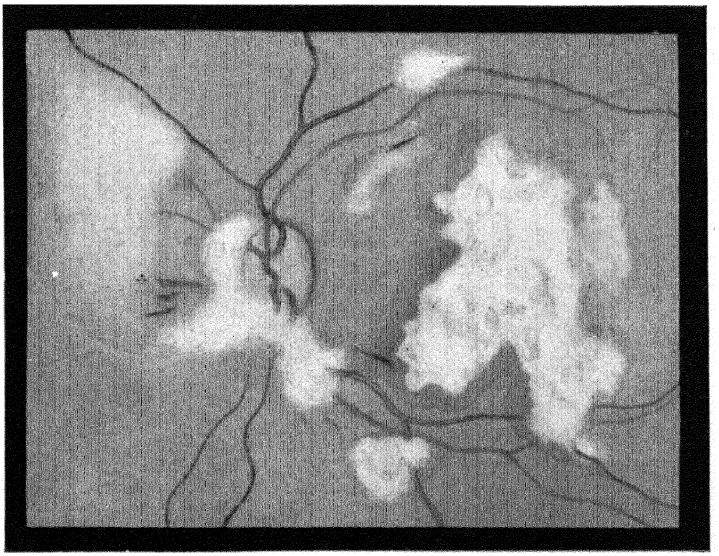
Arterio-sclerosis may cause local symptoms from its presence in certain organs. In the brain it may lead to thrombosis or hæmorrhage, and reproduce the characteristic symptoms. Apart from these complications the lack of nutrition of the brain due to arterio-sclerosis may lead to the gradual onset of mental deterioration, characterised in the early stages by forgetfulness, inability to concentrate, emotional instability, headaches, attacks of giddiness and insomnia. In the kidneys arterio-sclerosis may lead to fibrosis with loss of function and uræmia. In the limbs it may lead to intermittent claudication or thrombosis and gangrene. The arteries themselves may dilate, producing aneurysms.

**Prevention and Treatment.**—Prophylaxis consists in treating the causes of the condition—in particular, syphilis, and hypothyroidism, and alcohol, and

## PLATE VIII.




Arteriosclerotic Retinitis from a woman whose systolic blood pressure was persistently higher than 300 mm. Hg, and who died of a "stroke" about four years after the drawing was made.
(R. Foster Moore.)



To show "cotton wool patches" of Renal Retinitis as seen in parenchymatous nephritis.
(W. T. Holmes Spicer.)

[*To face p.* 368.

lead poisoning. Over-eating must be avoided. It is very important in early cases that all brain work and manual work should be stopped, and sources of worry removed. Little can be done by way of treatment when the disease has become established. Iodides are commonly given, and these may be beneficial if there is an element of hypothyroidism present, which is not uncommonly the case. Thyroid extract may also be tried. If it can be reasonably supposed to be due to high arterial pressure, the cause of that may be sought out and met, or the reduction of the pressure may be considered (*see* p. 370).

## HIGH ARTERIAL PRESSURE

(*Hyperpiesia*)

The normal range of blood pressure in young, healthy adults is from 95 to 140 mm. (*see* p. 301). It has frequently been taught that the blood pressure increases with the age of the patient; in fact, formulæ relating age and blood pressure have been constructed. This view arises from faulty weighing of the evidence. There are many elderly people with perfectly normal arteries in whom the blood pressure is normal. On the other hand, arterial disease, which itself often leads to a rise of blood pressure, becomes increasingly prevalent as age advances, and this explains the fact that the average blood pressure of a group of elderly people is found to be increased.

An investigation on a normal man, whose systolic blood pressure was actually below 100 mm., showed a rise of about 10 mm through the day and a corresponding fall during the night. The rise was chiefly due to taking tea, coffee, and to smoking. Exercise, mental work, worry and excitement increased the rise, and alcohol diminished it. A marked rise was observed during a holiday in the Alps (**26**). This suggests that the lack of oxygen, due to the rarefaction of the air, stimulates the circulation.

Arterial pressure is within normal limits influenced by more than one factor; thus the pressure will be increased by over-action of the heart, by increased capillary resistance, by narrowing of the smaller arteries and arterioles, and by an increase in the viscosity or volume of blood, though an increased volume is probably always a temporary condition.

In hyperpiesia the blood pressure is raised, and values from 150 to 300 mm. and more have been recorded. The rise usually begins in middle life, *i.e.* above thirty, and is fully developed by the time old age comes on; so that it is at this period of life that it is most commonly met with. The morbid anatomy of the fully developed form is what has just been described under the term *diffuse hyperplastic sclerosis*, and points in its ætiology have also been considered under *arterio-sclerosis*. Further, it has been pointed out that the primary cause of hyperpiesia is an increase in the peripheral resistance, due to internal proliferation in the arterioles of an inflammatory character, whether due to infectious or other poisons. The presence of pressor substances, an excess of adrenin or pitutrin in the blood, which might act on the vasomotor centre, has also been postulated, but without evidence. Whatever its cause, there is plenty of evidence that hyperpiesia may arise as a primary deviation from health. In the early stages of the disease, at any rate in some cases, the hyperpiesia is not constant, but occurs spasmodically. It may be an aggravation of the normal changes in blood pressure, already described. When once hyperpiesia is established, the hypertrophy of the left ventricle follows as a necessary consequence, because a greater effort is required on its part to carry on the circulation. The red cells and hæmoglobin of the blood are greatly increased in some cases (polycythæmia). The high blood pressure in these cases may be partly secondary to the increased viscosity of the blood.

**Symptoms.**—It is not necessary to repeat what has been said under

Arterio-sclerosis, or to anticipate the description of the state of the patient with chronic Bright's disease. The patient may complain of headaches, insomnia, tinnitus aurium, and giddiness. Pain at the back of the neck near the occiput is very characteristic. The characteristics of high tension are: (1) the records on the instruments employed (*see* p. 300); (2) the sensation to the finger of a firm well-filled artery, with long-sustained systole, never completely empty during diastole, capable of being rolled under the finger, but not necessarily palpable as a thick structure when emptied by pressure of the finger; (3) the evidence of enlargement of the heart and forcible apex beat; (4) the modified heart sounds, lengthening and muffling of the first sound at the apex, and accentuation or ringing character of the second sound at the aortic cartilage. Dieulafoy notes the rapid return of colour in the white line caused by scratching the skin of the abdomen; (5) the evidences of retinal arterio-sclerosis already described.

Most of the other conditions found in such cases are not so much evidence of the high tension as the results of the circulatory difficulties which ultimately supervene in the worst instances. They are slight albuminuria, due to secondary renal involvement (*see* primary contracted kidney, p. 630), though in most cases the blood urea is either not at all or only a little raised, which shows that there is little or no impairment in the kidney function; hæmorrhages in different parts of the body, as, for instance, epistaxis, hæmoptysis, retinal and vitreous hæmorrhages, small cerebral hæmorrhages causing so-called slight strokes or transitory aphasia, bleeding from piles; angina pectoris; mental depression; sometimes undue sleepiness; dead fingers; itching; cramps in the calves of the legs. Dieulafoy notes the increased sensibility to cold (*cryæsthesia*, κρύος, cold) which these patients suffer: it is felt especially in the lower extremities, and drives the patient to wear thick clothing, even in the warm seasons.

It is especially in these conditions of high arterial tension that sudden death is likely to occur; and as it is well known that in many cases of sudden death no macroscopic changes can be found post mortem to account for it, it is conceivable that in some of them undue and undiscovered arterial tension has been a cause.

Though it is common experience that many persons suffering from chronic Bright's disease are pallid or anæmic, in many cases of primary hyperpiesia it is just the other way. There is a high degree of congestion of the surface blood vessels. Such persons with advancing years may have the appearance of robust health, from the rich colour of the face, and may seem to be only instances of the somewhat florid colour which so many healthy old persons present. But in course of time the face, which was only unduly florid, becomes turgid, and later even congested; and at the same time the other circulatory and respiratory difficulties are making themselves felt. Thus these cases terminate eventually by sudden death, by an attack of angina pectoris, or in the same way as cases of chronic Bright's disease by cerebral hæmorrhage, by failing heart, with dropsy and œdema of the lungs.

**Treatment.**—If the cause of the high blood pressure can be recognised, attempts must be made to remove or reduce it. Where there is good reason to believe that the mode of life is contributing to the result, help may be sought by the avoidance of butcher's meat and highly nitrogenised foods or those containing purin bodies, by abstention from alcohol, tea, and tobacco, and from excessive mental or physical strains. It is advisable to give calomel (2 or 3 grains) or blue pill (3 to 5 grains) as an occasional purge, followed by a morning laxative saline; and diuretics such as the salines, and theobromine (1 to 5 grains) with thyminic acid (2 or 3 grains). The blood pressure is often reduced promptly when the patient is compelled to take complete rest in bed. Vaso-dilators, like nitroglycerine, amyl nitrite or sodium nitrite, have little effect in the permanent reduction of pressure, though they may be useful if acute symptoms, such as angina, occur. Potassium iodide and other potassium salts may be of use; and massage, muscular exercises, high frequency currents, and hydrotherapy will

sometimes be beneficial. Where the high blood pressure is compensatory to a lesion which is itself incurable, such as chronic interstitial nephritis, no attempt should be made to lower it. Digitalis is not suited for early stages, but may be helpful when the heart has reached an advanced stage of dilatation and there is œdema. In cases of hyperpiesia, *i.e.* where the blood pressure itself is the primary condition, the question of venesection should be considered, if other methods of treatment are unsuccessful, especially if symptoms such as headache are present, and there is no anæmia. Relief may be obtained by removing a pint of blood, and this may be repeated at intervals. Venesection may also be employed when there is dilatation and failure of the heart.

## LOW ARTERIAL PRESSURE

Much less often than high blood pressure does an abnormally low blood pressure come before us as a cause of definite symptoms requiring treatment. It occurs in the course of acute infectious diseases, and in other forms of intoxication, it is the result of myocardial failure or degeneration, whether as a part of the different forms of valvular lesion or independently. As a persistent condition it is present in Addison's disease. In these circumstances the systolic pressure falls to 90, or 80, or 70 mm., a figure which is not uncommonly registered in the latter disease.

**Symptoms.** In this also the results of an extreme condition of low arterial tension are illustrated: the patient may be incapable of any but the slightest exertion, and readily faints on attempting it. The pulse is soft, feeble and small, and probably dicrotic; the colour returns slowly to an area of the skin which has been rendered pale by pressure (*see* Addison's Disease).

It is only occasionally that low arterial tension requires separate treatment in the same way as the opposite condition. In infectious diseases, in heart disease, and in Addison's disease, one should look to the primary lesion or infection. When, exceptionally, a low arterial tension presents itself as a condition to be treated, the indications are complete rest, adequate food, stimulants, digitalis and strychnia. Suprarenal extract or adrenin are clearly suggested, though, as a matter of fact, suprarenal extract has not been very successful in the treatment of Addison's disease.

## ANEURYSM

This name (ἀνευρύνω, to widen) is applied to dilatation of an artery for a more or less limited extent of its course. Aneurysms are divided, according to their shape, into *fusiform* and *sacculated*, the fusiform being a more or less uniform dilatation of the whole circumference of the vessel; the sacculated forming a globular projection from one side of the vessel, and connected with it in advanced cases by a constriction or neck. Sometimes, especially in the limbs or the abdomen, a sacculated aneurysm ruptures at a prominent point; blood oozes slowly out into the tissue around and forms a coagulum, bounded by a kind of cyst of inflammatory tissue. This has been called a *diffused* aneurysm. Lastly, a *dissecting* aneurysm is formed when at a part of the artery affected with atheroma the blood penetrates the inner and middle coats, and forces its way between them and the outer coat.

**Ætiology.** Aneurysms arise from any cause that weakens the vessel at one point. The most common cause is atheroma, especially in the large vessels, in which the inner and middle coats are weakened, and the whole wall yields to the pressure of the blood at that point. In smaller vessels, such as those of the brain and lungs, the vessel may be weakened by the local causes of arteritis already mentioned—viz. embolism or the invasion of tubercle. Surgical injuries of the outer coat also lead to aneurysm. Irritation is another predisposing cause, and accounted for the frequency of popliteal aneurysm in the old days, when

horse-riding was more common than at present. Of the more general causes disposing to aneurysm syphilis holds an important place, and probably also excessive strain acting through the circulation.

**Results.**—The following description of results applies chiefly to the sacculated forms. One result is the *coagulation* of the blood in the sac itself. As this is out of the direct current, it moves more slowly, and its coagulation is favoured by the roughness of the aneurysmal sac. The sac thus becomes lined, or nearly filled, with successive layers of pale buff fibrinous deposits; and it is by the complete filling of the sac with these fibrinous layers that aneurysms may be obliterated and cured. The greater the freedom of communication with the main vessel, the less the liability to the formation of fibrin; and in a fusiform aneurysm no deposits take place.

Another result of aneurysm is its *pressure* upon the parts around it. The sac may attain an enormous size; an aneurysm, of which there is a model in the museum of Guy's Hospital, springing from the aortic arch, measured 8 inches in diameter. As it enlarges, the growth presses with irresistible force upon adjacent parts, displacing the various organs, compressing and obstructing the blood vessels, the trachea, the bronchi, or the œsophagus, flattening and stretching nerves, and causing thereby pain, numbness, or paralysis, according to the nerve involved and the degree of its compression. When it comes into contact with unyielding bone, an aneurysm causes absorption of the osseous tissue, and excavates or actually perforates it. The vertebræ are frequently eroded in this way, and it is remarkable that the intervertebral cartilages are more resistant than the bone, so that when the aneurysm is large enough to cover more than one vertebra the cartilage projects between the two cavities which have been made in the adjacent bones. The ribs and sternum, at first slightly raised by the advance of an aneurysm, are subsequently perforated and allow the pulsation of the tumour directly under the skin. Analogous results occur in other parts of the body, but it is chiefly in the thorax that the pressure effects of aneurysm are manifested, because the bony walls allow no room for their important contents to escape.

A third effect of aneurysm is *hæmorrhage*, which is the cause of death in a large percentage of cases. The great distension of the coats and the degeneration which precedes this sufficiently explain why hæmorrhage occurs. Even the deposit of layers of fibrin within the sac will not always prevent it; the clot, if at all abundant, does not organise, and the blood may force its way into fissures and meshes of the coagulum, and so finally reach the surface and slowly ooze out. The rapidity and extent of the hæmorrhage further depend upon the support the aneurysm has from without. Ruptures into hollow viscera and serous cavities are often rapidly fatal, though a man has lived ten days after a rupture with hæmorrhage into the pleura. Ruptures into connective tissue or intermuscular spaces are often much slower in their effects, and in the limbs may allow time for successful treatment.

**Symptoms.**—They may be divided into those common to all aneurysms and those determined by the locality; of the latter only the special symptoms of thoracic aneurysm will be now considered, those characteristic of abdominal aneurysm being discussed elsewhere (*see* Abdominal Tumours).

The symptoms common to aneurysm in any part of the body are—(1) tumour; (2) pulsation; (3) murmur; (4) pain; (5) other effects of pressure.

*Tumour*, or some kind of swelling, is a necessary part of an aneurysm, but it may, of course, be entirely unrecognisable during life in such parts as the cranium, the thorax, the deeper parts of the abdomen, or the gluteal region.

*Pulsation* is the characteristic symptom, showing the connection of the tumour with an artery. It is nearly synchronous with the cardiac systole and the radial pulse, mostly rather slow and heaving, expansile—that is, a sudden increase of pressure is experienced by the fingers on palpating the tumour on all sides, and

not only in one position, as would be the case with an independent tumour lying over the artery. Pulsation is affected by the amount of coagulum lining the sac, and is in some cases absent. It must be specially noted that the pulsation of the healthy abdominal aorta can be felt on palpating the abdomen in spare individuals; sometimes this pulsation has been noticed by the patient, and may be complained of by hysterical or hypochrondriacal individuals. It must not be confused with aneurysm of the abdominal aorta, a much rarer occurrence.

*Murmur.*—On listening with the stethoscope over an aneurysm in the abdomen or limbs a murmur is mostly audible, synchronous with the beat of the pulse, varying in quality, soft, harsh, or loud. It is due to the passage of the blood from the normal artery into the wide cavity of the sac. In some cases a diastolic murmur is present; and with a feeble current of blood, or with much coagulum in the sac, and perhaps under other circumstances, the murmur may be absent.

*Pain* is frequently present, and results from the stretching of, or pressure on, parts in the neighbourhood. It occurs at the seat of the aneurysm, and often radiates in different directions from it—to the shoulder and down the arm, for instance, in thoracic aneurysm, and along the course of the intercostal nerves when the spine is eroded.

The other effects of pressure will vary with the seat of the aneurysm.

## ANEURYSM OF THE THORACIC AORTA

Aneurysm may occur at any part of the thoracic aorta, from the sigmoid valves to the diaphragm; but the first part is most frequently involved, and here especially all varieties occur, from irregular dilatations of the whole calibre to true sacculated aneurysms.

The results of the former, *dilated aorta*, are not seen so much in the pressure effects. If the part adjacent to the valves is affected, the orifice may be dilated, the valves are thus rendered incompetent to close it, and regurgitation will take place, followed by dilatation and hypertrophy of the left ventricle. In other cases of dilated aorta there is a systolic murmur, and the patient suffers from attacks of severe cardiac pain, with many of the characteristics of *angina pectoris*, on anything beyond the most moderate exertion. Such patients sometimes die suddenly.

A *sacculated aneurysm* causes symptoms which depend on its position and the direction of its growth. Aneurysm of the ascending aorta, which often justifies its title as the "aneurysm of physical signs," may grow forwards and present itself as a pulsating tumour in the second or third right intercostal space, less commonly in the second or third left space. The tumour is slightly tender, often the seat of pain, which is aggravated by exertion; and commonly a soft systolic murmur is heard over it. Growing towards the right, a tumour in this region presses upon the superior vena cava, causing œdema of the arms, or it grows into the upper part of the right chest, compressing the upper lobe of the right lung or the bronchus leading to it, and producing deficient breath sound and at a later stage dulness over the corresponding area. Towards the left an aneurysm may press upon the pulmonary artery, cause dilatation of the right heart, and ultimately open into the pulmonary artery. Aortic aneurysms have also opened on rare occasions into one or other main pulmonary branch, into the right ventricle, into the right auricle, into the left auricle, and into the superior vena cava (*varicose aneurysm*).

In nearly all cases of such communications, when the patient has lived sufficiently long, a murmur is heard; and in some it has exceptional qualities in that it is a continuous or wavy murmur, apparently covering both first and second sounds, and being especially harsh, blowing or roaring. In other cases the murmur is double, or only systolic. A thrill is frequently present. Rupture into

the pericardium is a not infrequent termination of aneurysms of the ascending aorta.

As a diagnostic feature of an aortic aneurysm, importance is attached by some to the exceptional loudness of the second sound, and to the sense of shock (*diastolic shock* or *rebound*) which is communicated to the hand or to the ear resting upon the old wooden stethoscope. When it occurs, it must be due to unusual force of closure of the valves due to recoil of the aneurysmal walls, or, quite as likely, to unusual intensity of vibrations because they occur in the aneurysmal cavity.

Aneurysm of the arch of the aorta, which often justifies its title as the "aneurysm of symptoms," especially affects the convex border at the site of the origin of the great vessels, and frequently grows upward to the base of the neck, where it forms a pulsating tumour and is with difficulty distinguished from aneurysm of the carotid or innominate artery. The pressure effects, if present, are mainly directed towards the trachea, producing stridulous breathing and dyspnœa; while the tumour itself is indicated by dulness over the upper end of the sternum and by a murmur. A large aneurysm in this position may drag the larynx downwards and to the left by pressure on the trachea. Aneurysms arising from the concave part of the arch come into relation with the left bronchus and with the left recurrent laryngeal nerve, which winds round it. Pressure on the bronchus causes in the corresponding lung diminution of the movement of the tidal air, in some cases early distension of the lung sufficient to displace the heart and depress the diaphragm, in all cases ultimately collapse of the lung with retention of bronchial secretions and bronchiectasis. The physical signs are absence of vesicular murmur with exaggerated breathing on the opposite side, hyper-resonance on percussion, later dulness with loss of voice sounds and of tactile vibration, later again localised bronchial or cavernous breathing and moist râles (*see* pp. 223, 230). Compression of the lung itself may occur with localised dulness and loss of respiratory murmur, and in some cases gangrene. Another result of pressure on the bronchus is that when, with the patient in the upright position, his mouth closed, and his chin raised to its fullest extent, the cricoid cartilage is grasped by the finger and thumb and gently lifted, the pulsation of the aneurysm is conveyed to the fingers holding the cricoid. This sign, called *tracheal tugging*, has been said positively to indicate an aneurysm of the transverse arch; but an aneurysm adherent to the trachea may cause it as well as one pressing on the bronchus. Slight tugging can be felt in some healthy persons, but pronounced movement is a valuable sign of aneurysm. The pressure of the aneurysm on the recurrent laryngeal nerve causes abductor paralysis of the left vocal cord with subsequent "paralytic contracture" of the adductor, so that the cord occupies the middle of the glottis, and a certain amount of stridor and clanging cough may be the result.

Aneurysms of the aorta *below the arch* may press upon the œsophagus and cause dysphagia, or regurgitation of food; salivation after food has been noticed in some cases. The aneurysm may rupture into the œsophagus, and Sir Frederick Taylor knew one in which the blood rupturing the outer coats found its way between the coats to the stomach and finally ruptured into that organ. Directed backwards, an aneurysm erodes the spine, presses upon intercostal nerves so as to cause intense pain, and later causes paraplegia by implicating the spinal cord. A murmur is often heard over the spine behind under these circumstances. Growing laterally, the aneurysm may compress a large bronchus or the lung.

Some other symptoms may be mentioned which are common to two or three of the situations discussed.

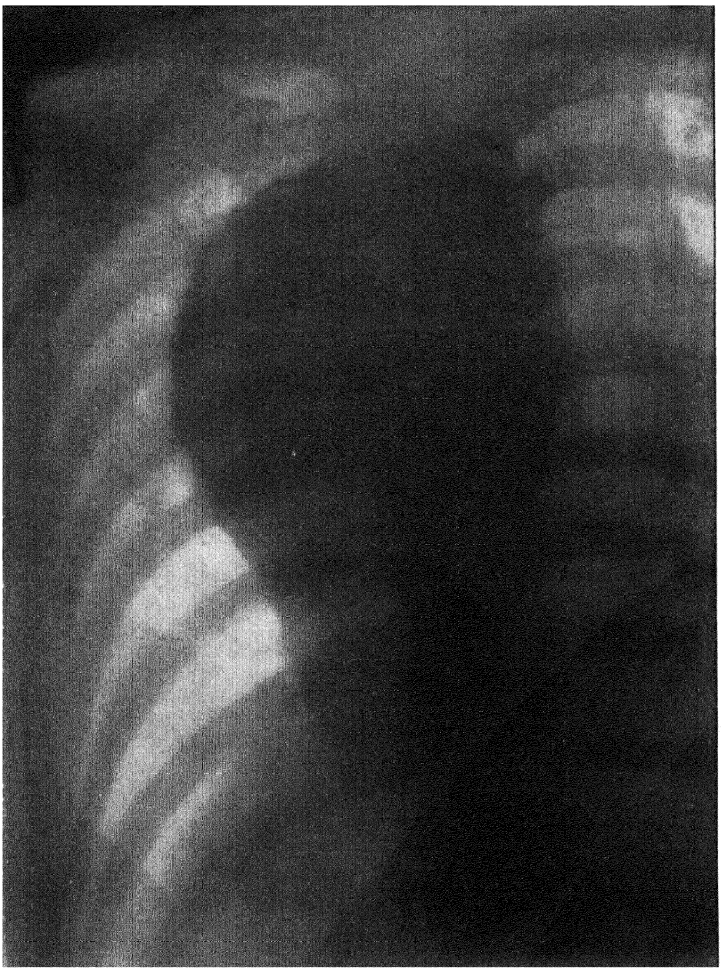
*Inequality of the Radial Pulses.*—The radial pulses may be unequal in volume, in sharpness of rise, and in blood pressure. If the aneurysm compresses the innominate or the left sub-clavian artery, or obstructs either of these vessels at

PLATE IX.

Radiogram of an Aneurysm involving the Ascending and Descending Aorta and the Arch of the Aorta. This is shown by the increase in the shadow both to the right and left-hand side. (Taken by Dr. A. C. Jordan.)

[*To face p.* 375.

PLATE X.



Radiogram of a large Aneurysm of the Descending Part of the Arch of the Aorta, taken from the back (Taken by Dr. A. C. Jordan.)

[*To face p.* 375.

its origin by the formation of coagulum, the corresponding pulse is smaller and less sharp in its rise, and shows in the sphygmographic tracing a sloping upstroke, or even complete absence of the percussion wave (Fig. 47, A, B, C). The blood pressure as measured by the sphygmomanometer is usually higher than normal in aneurysms of the aortic arch or innominate artery. A difference between the pressures in the two radial pulses of 5 or 10 mm. Hg is common in these conditions, but also not infrequent in arterio-sclerosis. A constant difference of more than 20 mm. is in favour of aneurysm; but a difference of this degree was found only in 10 per cent. of cases (two out of twenty) of such aneurysms (O. K. Williamson).

*Inequality of the pupils* (*anisocoria*) has usually been attributed to interference with the sympathetic nerve fibres. Drs. Wall and Walker show reason for doubting if this explanation will meet every case; and they attribute the inequality to local pressure on the vessels, pointing out that normally pressure on the carotid will cause dilatation of the pupil of the same side.

Loss of flesh, cough, and dyspnœa on exertion or in paroxysms, and pain, are the common accompaniments of thoracic aneurysm of any size. Death often takes place from associated myocardial changes which are secondary to coronary sclerosis; but it sometimes follows from interference with necessary functions from pressure upon such parts as the œsophagus, trachea, or bronchus; from inflammatory and septic processes from pressure on the lung; and, lastly, from rupture of the sac and hæmorrhage, either externally through the skin, or into the œsophagus or pericardium or pleural sac.

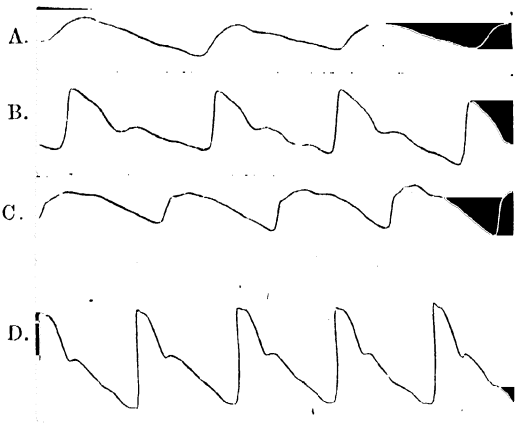



FIG. 47.—Sphygmographic Tracings. A. Right radial pulse in a case of aortic aneurysm compressing the right innominate artery. B. Left radial pulse in the same case. C. Radial pulse in compression of the subclavian artery. D. Atheromatous artery, showing normal tracing. Pressure, six ounces.

**Diagnosis.**—The diagnosis must be made from a careful consideration of the above facts, remembering that the symptoms vary with the seat of aneurysms. Some of the combinations which may give rise to suspicion of aneurysm may be enumerated: (*a*) pain like that of angina, with systolic murmur over the base of the aorta; (*b*) systolic murmur occurring at a part of the chest not corresponding to the seat of any valvular orifice; (*c*) pulsating tumour in or near the præcordial region; the points at which a normal or enlarged heart may beat must be remembered, as well as the fact that pulsation of the top of the right ventricle in the second left interspace is quite common; (*d*) obstruction of one or other radial pulse, but, of course, one or other subclavian artery may be obstructed by other means so as to produce a local murmur and a delayed pulse; (*e*) obstruction of veins; (*f*) obstruction of trachea, bronchus, or œsophagus; (*g*) paralysis of one vocal cord, especially the left. The diagnosis may now often be confirmed by the use of the Röntgen rays (*see* Plates VII., Fig. 2, IX. and X.); and if dysphagia is the only symptom the question of aneurysm should be determined by this means, if possible, before the passage of a bougie, which might produce a fatal rupture. The appearance of the normal aorta is shown in Plate I., facing p. 257.

Aneurysms near the base of the aorta are more likely to be confounded with

valvular disease, those in the deeper parts of the chest with new growth (*see* Mediastinal New Growths). The extremely rare pulsating pleurisy or empyema has been referred to (*see* p. 274).

The diagnosis of the existence of a communication between an aneurysm and the right heart, pulmonary artery or vena cava, is often difficult. Even the time at which rupture takes place cannot always be recognised. In some cases there is a sudden aggravation of dyspnœa, cyanosis and dropsy. The most characteristic sign is the wavy murmur covering both systole and diastole, like that heard in cases of patent ductus arteriosus (*see* p. 355); but it is present in less than half the cases.

**Prognosis.**—It is necessary to take the condition of the myocardium into account. The ultimate prognosis is unfavourable in the case of thoracic aneurysms. Complete consolidation with cure is rare; relief of symptoms and postponement of the fatal result for some years may be often effected.

**Treatment.**—The objects of treatment are to delay enlargement, to prevent rupture, and to favour coagulation of blood in the sac. Everything which will quiet the circulation and diminish the force of the heart's contraction will act favourably in these directions. The surgical methods employed in aneurysms of the limbs are rarely applicable in the case of thoracic aneurysms. Proximal ligature of the vessel is out of the question, and distal ligature or compression can only be applied to branches such as the carotid and subclavian. Insertion of horsehair, wire, or needles, and the injection of astringent fluids into the sac have had too little success in aneurysms of the limbs, where the operation is more under control, to be recommended for aortic aneurysms. Galvano-puncture has, perhaps, been more successful. Two needles, connected with the poles of a battery, are introduced into the sac of the aneurysm; on the passage of the current fibrin is deposited on the poles, but the danger of portions being detached, and causing embolism in remote parts, is present here, as it is in the methods just mentioned. The treatment of aneurysm in the chest generally resolves itself into—(1) rest; (2) restricted diet; (3) the use of anodynes and sedatives; (4) iodide of potassium. Absolute rest has been urged by some, the patient being in the recumbent position, and not allowed to sit or stand up for any reason whatever. However, the wisdom of such a course is very doubtful, especially as in many cases the myocardial changes ultimately cause death. However, it is very important to avoid excessive exercise. The diet which was recommended by Tufnell, who strongly advocated absolute rest, consisted per diem of 10 ounces of solid, including 3 of meat, and 8 ounces of fluid, divided into three meals; but it is extremely trying, and few patients will submit to it. Opium or morphia is generally given to ease pain, to produce sleep, or calm restlessness, but other sedatives may also be of use, such as bromide of potassium, chloral, paraldehyde, or sulphonal. Pain may also be relieved by belladonna applications, or by cold, or by venesection to a small amount. Iodide of potassium appears to have a special influence upon the coagulation of blood in aneurysms, as great improvement in diminution of pulsation and of pain has followed its use, even though unassisted by restriction of diet. It should be given in increasing doses, up to 60, 90, or 100 grains daily. Gelatin in solution has been injected subcutaneously with some success; but accidents have occurred from the presence of tetanus spores, so that the method has been seriously discredited. From 2 to 6 ounces of a 2 per cent. solution in normal saline, carefully sterilised, are injected into the gluteal region every five or six days up to thirty or forty injections; the patient must be kept strictly at rest. Where the vocal cords are implicated by pressure on the recurrent laryngeal nerve, tracheotomy may be required to prevent fatal asphyxia. But aneurysms also cause dyspnœa by tracheal compression, for which tracheotomy would afford no relief.

## ABDOMINAL ANEURYSM

**Pathology.**—The usual seat is between the diaphragm and the origin of the superior mesenteric artery, and it often involves the origin of the cœliac axis. In its growth it may interfere with adjacent organs, press upon the vena cava, or erode the vertebræ. Aneurysms of the superior mesenteric, or of the iliac arteries, are less common, and will not be specially considered here.

**Symptoms.**—These are pain, the presence of a pulsating tumour, with murmur, and sometimes evidences of pressure. The *pain* is situate in the abdomen, is often severe, paroxysmal or neuralgic in character, and may radiate to either side, into the groin or the back. The *tumour* varies, of course, with the seat of the lesion; it is more common in the epigastric region, in the middle line, or slightly to the left; it is globular or ovoid, pulsatile, and expansile; it is scarcely, if at all, affected by the movements of the diaphragm. A systolic murmur can generally be heard over it. The pressure signs other than pain are not common, since the several organs readily yield to its progress. But it may exceptionally cause jaundice by obstructing the bile duct, or dropsy by pressure on the vena cava. It may press on the colon, or on the stomach, and vomiting is sometimes present. The duration may be two or three years, and death results, as a rule from rupture of the sac into the retro-peritoneal tissue, into the peritoneum, or into one of the hollow viscera, or from the exhaustion of pain, sleeplessness, sickness and malnutrition.

**Diagnosis.**—It is very common to feel a pulsation of the normal aorta in women on palpating the abdomen, particularly if the muscles of the abdominal wall are weak. This is often mistaken for abdominal aneurysm by the inexperienced. Abdominal aneurysm must also be distinguished from tumours lying in front of the aorta, especially *carcinoma of the stomach*, or less commonly cancer about the *gall bladder*, to which pulsation is communicated from the healthy aorta. Tumours over the aorta do not expand laterally, and are often irregular or nodulated in shape; the pulsation in some cases ceases when the patient is placed prone, or on his hands and knees, so that the tumour may fall away from the aorta. Carcinoma of the stomach is displaced more than an aneurysm by a deep inspiration.

**Treatment.**—This must follow the lines indicated under the head of Thoracic Aneurysm (*see* p. 376). But an abdominal aneurysm is sometimes open to treatment by proximal or distal compression by the tourniquet or other surgical means.

## CONGENITAL COARCTATION OF THE AORTA

In this rare condition there is stenosis or complete obliteration of the aorta at the point of junction with the ductus arteriosus just beyond the origin of the left subclavian artery. If the child survives, the circulation in the trunk and lower limbs is maintained by anastomosis between branches of the subclavian and axillary arteries on the one hand and the thoracic arteries and epigastric arteries from the femoral on the other. The anastomosing arteries become enormously enlarged to carry the necessary amount of blood, and they form large tortuous pulsating vessels which can be felt beneath the skin of the thorax, in front or behind, or in the axillary region and flank. The abdominal aorta and iliac and femoral vessels may be devoid of pulsation, or pulsate but feebly, from the diminution of the force of the current by the time the blood reaches them through the anastomoses.

The patients may live for some time in comfort, but commonly die young; a few, however, survive to a great age. The heart is often, but not always, dilated and hypertrophied; valvular lesions and other cardiac defects are often present; and a murmur may be heard over the aortic valves, or over the præcordia generally, and sometimes behind.

## PHLEBITIS

Inflammation of the veins, or phlebitis, results in thickening and infiltration of the walls with leucocytes, which may be in such numbers as to constitute a real suppuration of the coats. The terms *endophlebitis* and *periphlebitis* have been used to indicate inflammation of the intima and the adventitia respectively. Periphlebitis arises from contact with inflammatory foci outside the vein, or from injury. Endophlebitis is most often set up as a result of thrombosis or coagulation of blood in the vein itself. This occurs from a variety of causes (*see* Thrombosis). The clot may then adhere to the vein wall, becoming at the same time organised, and the vein may be completely obliterated. On the other hand, the clot may become channelled, and allow the continuation of the circulation; or in other cases it softens down into a puriform liquid. Periphlebitis extending inwards itself leads to thrombosis; on the other hand, abscesses may form in the tissue around the vein.

**Symptoms.**—Phlebitis is accompanied by pain and tenderness in the course of the affected vessel, with some reddening of the surface in the case of superficial veins. The vein can be felt as a prominent hard cord, and a varying amount of febrile reaction accompanies the local disease. The formation of abscesses will be indicated by hardening of the surrounding tissue, redness and œdema of the skin, and subsequently fluctuation. The secondary effects which result from breaking down, and transportation of the particles of thrombus, are described below.

The **Treatment** of phlebitis consists in complete rest of the part affected, the application of warm fomentations or of glycerine and belladonna to ease pain, and the administration of opiates, if necessary, for the same purpose. The risk of detachment of a thrombus must always be borne in mind (*see* Thrombosis and Embolism). If abscesses form the pus will have to be evacuated by incision.

## THROMBOSIS AND EMBOLISM

*Thrombosis* is the name applied to the coagulation of blood within living vessels, whether arteries or veins, or in the cavities of the heart; and the clot itself is called a *thrombus*.

*Embolism* means the transference of a portion of clot or other substance (particles of tumour, parasites, fat globules) from one part of the circulation to another, and its impaction when it arrives at a vessel too narrow for its further progress. Embolism may take place along the arteries to the periphery of the systemic circulation, along the systemic veins and pulmonary artery to the lungs, and along the portal vein to the liver. The transferred particle is called an *embolus*.

Besides the conditions of fibrin formation which usually determine coagulation, two important factors in *thrombosis* are—(1) undue slowness of the current of the blood, whether from diminished cardiac force, from local obstruction in the vessel, or from increased viscosity of the blood, and (2) some lesion of, or irregularity on, the lining membrane of the vessel or cavity concerned; but it must be allowed that there is often an intimate relation with infective disorders in which micro-organisms or toxins may have a share, and with the conditions of health present in gout and in the puerperal state.

Thus we see that blood coagulates in the heart upon its inflamed valves, or in its cavities when dilated or contracting with extreme feebleness. It coagulates in the vessels if their walls are injured, or are in connection with septic or gangrenous processes; in the arteries especially when their walls are the subject of syphilitic or atheromatous lesions, or of aneurysmal dilatations; in the veins when the current of their blood is slowed by pressure, and with the slightest local

disturbance in the subjects of various infective, cachectic, and anæmic disorders. The first step in the process seems often to be the accumulation of blood platelets at the determining spot, and later an aggregation of leucocytes or the formation of fibrin. The effect of the coagulation of blood in a vessel is naturally to cause an obstruction, which will have different effects according as it is in an artery or in a vein. The coagulum once formed receives further deposits of fibrin from the blood circulating above and below, and so the thrombus may extend into larger and larger vessels. When first formed, it is soft and fills the veins; but after a time it may shrink, and thus allow the re-establishment of the circulation. But the termination is not always so favourable. The thrombus commonly sets up some endarteritis or endophlebitis, adhesion to the wall of the vessel takes place, and ultimately the clot becomes organised, with permanent obliteration of the channel. In other cases septic micro-organisms may cause the coagulum to break down into a puriform fluid, which consists of pus corpuscles, micrococci, and fine granular particles An important result of thrombosis in the heart and in the veins is the detachment of fragments from the coagulum and their transference to other parts of the circulation. They then become *emboli*, as above stated. The results differ according to the position and character of the original thrombus. Portions detached from venous thrombi are carried by the current of blood into the right auricle, thence into the right ventricle, and into the pulmonary artery, which they may block according to their size, either quite at its commencement, or in the substance of the lung. Thrombi in the right side of the heart will similarly cause embolism of the pulmonary artery; but thrombi on the aortic or mitral valves, or in the right auricle, will cause embolism of the systemic arteries in the brain, spleen, kidneys, limbs, or elsewhere.

The result of embolism, unless a collateral circulation be promptly established, is the death of the tissue within the area of the vessel obstructed. The portion of tissue so affected is called an *infarct*. In some organs, such as the liver, the anastomosis of vessels is so complete that infarcts are never formed; but in other organs, although there may be some anastomoses, there are not sufficient for the nutrition of a part. Consequently when an artery, which is known in this case as a *functional end artery*, is blocked, an infarct is formed. In the solid organs it usually has a conical shape, and is therefore triangular or wedge-shaped on section; and as seen post mortem it is either hard, of white or yellowish-white colour (*white infarct*), or softer and blood-red in colour (*hæmorrhagic or red infarct*). In the former case the change is chiefly one of *coagulative necrosis;* the tissue deprived of its blood supply is permeated with lymph from the surrounding living tissue, and coagulative changes take place in this. If the coagulable material is sufficient the infarct is hard, as seen in the kidney and spleen; if it is less abundant, the infarct is softer, as in the brain. The white infarct is sometimes surrounded by a narrow margin of hæmorrhage. In the hæmorrhagic infarct the first process is also one of coagulative necrosis; but to this is added more or less complete hæmorrhage by diapedesis of red corpuscles Infarcts of the kidney and retina are commonly of the white variety; those of the lung and intestine are constantly hæmorrhagic, because, owing to the presence of air in these two organs, the capillary walls are not supported, and blood oozes from them, when the cells of the lining membrane die from lack of nutrition. Infarcts of the spleen and heart may be either white or red. While infarcts in early stages are often somewhat swollen and project on the surface of the tissues, they subsequently, if not septic, become shrunken and contracted, as seen especially in the kidneys; the elements undergo fatty degeneration and are replaced by connective tissue. If the embolus comes from a suppurating thrombus, or is the product of malignant endocarditis, then the contained organisms may set up septic processes in the infarcts. These become purulent in the centre, forming abscesses, such as occur in the lungs in pyæmia, or in the brain and kidney occasionally in malignant endocarditis. Septic emboli, by infecting the

arterial wall, sometimes determine the weakening and dilatation of the artery at the seat of impaction, and so the formation of an *embolic aneurysm*. If the main vessel of a peripheral part (foot, leg, or hand) is obstructed, to which no surrounding living tissue can supply coagulable material, the result is not coagulative necrosis, but gangrene.

The following are the more usually recognised forms of thrombosis and embolism:

*Femoral Thrombosis.*—Thrombosis of the femoral vein arises in the last stage of phthisis, cancer, and other exhausting diseases, in convalescence from typhoid fever and influenza, and after confinement ("white leg"). The leg becomes swollen, and the vein can be felt to be obstructed; there is generally also some tenderness from co-existing phlebitis. The detachment of a portion of clot, followed by its impaction in a large branch of the pulmonary artery with sudden death, is an occasional accident. To prevent thrombosis in the limbs during convalescence from fevers the movements of the patient must be regulated with care. He must not be allowed to lie so that the veins are compressed. Support can be obtained with soft pillows.

*Jugular* thrombosis and thrombosis of the *lateral sinus* result from disease of the internal ear, or mastoid cells. From contact with the external ear, septic organisms are frequently present, severe phlebitis is set up, and the clot becomes septic. Particles are then conveyed through the right side of the heart to the lungs, in which pyæmic abscesses are formed (*see* p. 51). Other cerebral sinuses (longitudinal and cavernous) are sometimes thrombosed as a result of more general conditions, such as marasmus in infants, and chlorosis and anæmia in adults (*see* p. 848).

Thrombosis of the *pelvic veins* arises from disease of the pelvic viscera in women, or from gonorrhœa in both sexes.

Thrombosis of the inferior vena cava is not incompatible with life. In one case who lived for twenty-five years, the vein was converted into an impervious ribbon from below the entrance of the hepatic veins. Blood was returned from the kidneys by way of the capsule and the lumbar and azygos veins. There is always great varicosity of the veins of the leg and abdominal wall, with a tendency to thrombo-phlebitis, hæmorrhoids, and varicose ulcers. The causes are infection, traumatism and malignant disease (**29**).

Large clots sometimes form in the *heart*, just previous to death, when the circulation is failing, and in recesses of the walls in cases of dilatation. They may hasten death by hampering the action of the organ, or they may supply emboli to the pulmonary or systemic circulation.

Embolism and thrombosis of the *cerebral arteries* are described under Diseases of the Brain (*see* p. 825).

Embolism of a large artery in a *limb* is not a very common event. It causes sudden acute pain, followed at once by numbness, coldness, and loss of power in the limb; the pulse is imperceptible below the seat of embolism, and, as already stated, gangrene may result. In the *spleen* and *kidneys* the occurrence of embolism is not so commonly recognised. Sometimes there is sharp pain in the left side from embolism of the spleen. Embolism of the kidney causes hæmaturia and frequently albuminuria too, and in malignant endocarditis there is often a condition of embolic nephritis present (*see* p. 619). Cases of embolism of the *mesenteric artery* have occurred in which the patient has been seized with severe abdominal pain and distension, followed by collapse and death in one or two days; and blood has been found in the bowel and in the peritoneal cavity. Very similar results may follow thrombosis of this vein, but the symptoms are more slowly developed.

The effects of embolism and thrombosis of the vessels of the *liver* are described under Pylephlebitis. Pulmonary embolism and thrombosis, and infarction and fat embolism are described on p. 269.

Embolism of particles of *new growth* is the cause of fresh growths in remote parts.

**Treatment.**—This is chiefly palliative. The pain of embolism may be relieved by local anodyne applications; if the large artery of a limb is obstructed, the limb should be wrapped in cotton wool or oiled lint; and surgical measures may have to be considered. Citric acid has been credited with solvent properties, and it may be given internally in doses of 15 or 20 grains for the resolution of thrombosis; but it is of doubtful value.

## VASOMOTOR DISORDERS

### RAYNAUD'S DISEASE

This disorder, first described by M. Raynaud in 1862 as local asphyxia and symmetrical gangrene of the extremities, is due to a spasmodic contraction of the arterioles, whereby the circulation in the affected parts is retarded, so as to cause a temporary "deadness" or lividity of the part, or is obstructed to such an extent or for such a long period as to be followed by actual gangrene.

**Ætiology.**—It is much more frequent in women than in men, and is first noticed commonly between the ages of fifteen and thirty, or even in childhood. Cold and emotional disturbance are exciting causes. Many patients are delicate or anæmic, nervous or hysterical, but some seem to have been in good health until the occurrence of the disease. Associated with Raynaud's disease, hæmoglobinuria, peripheral neuritis, and various skin eruptions, particularly scleroderma or sclerodactylia, have occurred. In the latter condition the skin of the fingers becomes thick, smooth and glossy, and eventually atrophies.

**Symptoms.**—Raynaud describes these as occurring in three degrees of severity. The simplest and least severe is one of *local syncope*, in which spontaneously, or from cold or mental emotion, one or more fingers turn white, cold, numb, and insensible to touch. The condition lasts from a few minutes to several hours, and recovery is accompanied by a good deal of pain. This is one form of the condition known as *dead fingers*, which, however, in a less pronounced form occurs in many people who are not to be regarded as having Raynaud's disease; local syncope results from closure of both arteries and capillaries.

In the second degree, *local asphyxia*, the fingers are more or less cyanosed; they are bluish-white, violet, slate-coloured, or even black. Pressure upon them produces a white spot, which only slowly regains the former livid colour. The adjacent part of the extremity is often slightly swollen, and there is a livid marbling of the limb for some distance above it. There are, with this, always much pain and complete anæsthesia. Recovery is accompanied by tingling and pricking, and the livid tint gradually passes through scarlet to the natural pink colour. In local asphyxia the arteries are shut and the capillaries open.

The third degree is the condition known as *symmetrical gangrene*. Sometimes this begins with pallor of the fingers, which then become lilac, and afterwards violet, with acute pain, tingling, and sensation of burning heat, though the finger ultimately becomes quite cold to the touch. In other cases the finger is at first livid red, with itching and tingling, and finally is the seat of severe pains. Then in either case there is livid mottling of the adjacent limb, and the fingers become black and insensible to touch; vesicles or bullæ containing sero-purulent fluid form on them, and burst, leaving small ulcers, which shortly heal, while the lividity gradually subsides. With a repetition of this process, numerous small cicatrices may form on the affected part, and the fingers acquire a shrivelled, pinched, parchment-like aspect. The skin may desquamate, and the nails may fall off. In other cases, without the formation of bullæ or phlyctenulæ, the fingers and toes become black, shrivelled, and gangrenous; and then a superficial layer of skin, or even some portion of the deeper tissues, separates as a slough in the

course of a few weeks. The most marked symptom accompanying these severe cases is intense pain, of paroxysmal character, radiating to other limbs; the pulse may be thin or compressible, but is always perceptible, and the general health of the patient may be remarkably little affected. The toes are attacked as well as the fingers, and sometimes before them; and the nose and ears may be livid, but do not often slough. When the ulcers heal, it is remarkable what slight scarring remains behind.

The attacks occur at intervals of weeks or months, and in some cases, after repeated slight attacks, the fingers remain in a permanently benumbed or shrivelled condition.

**Diagnosis.**—Senile gangrene is distinguished by the age of the patient, by the gangrene affecting a single limb, and generally a lower limb, by its progressive course, and by the diseased condition of the artery of the limb. *Chilblains* present a certain resemblance to local asphyxia, and perhaps may have an allied pathology; they occur from definite exposure to cold.

**Prognosis.**—Many cases recover. Death is rare as a direct result of the gangrene.

**Treatment.** A strong continuous galvanic current should be tried, with the anode at the back of the neck, and the kathode over the sacrum and lower lumbar region; or the affected limb should be immersed in a basin of salt and water, in which one electrode of the battery is placed, while the other is applied to the top of the limb. Shampooing the limb and diffusible stimulants internally may also be employed. Cold and excitement should be avoided. Einhorn claims complete success by administering very large amounts of warm sterile normal saline through a duodenal tube.

## INTERMITTENT CLAUDICATION

Intermittent limping or *claudication* may be considered under functional disorders of vessels, although in most cases there is an underlying structural change. In this rare condition, the patient finds that after walking a certain distance he has weakness in one or other leg, with stiffness, heaviness, numbness, pricking sensations, pains and cramp, so that he necessarily limps in his gait. The painful sensations increase as he progresses, and he is at last obliged to stop. The foot or leg shows signs of circulatory disturbance; it becomes red or mottled, and swollen; and the toes may be white and "dead." After a period of rest the symptoms gradually subside. In the majority of cases there is evidence of sclerosis of arteries or veins, or of obliterative arteritis or atheroma, or thrombo-angiitis obliterans; and in nearly all there is an absence of pulse in the dorsalis pedis artery, or in the posterior tibial of the affected limb. It is, accordingly, a disease of adult life; and gout and syphilis, and indulgence in tea, tobacco, or alcohol, are often among the antecedents. It is more common in men than in women. The symptoms are due to the affected arteries being unable to carry the increased flow of blood that the limb requires during muscular exercise.

In some cases slight muscular wasting and degeneration of the peripheral nerves (peripheral neuritis) have been observed. In many instances the complaint has resulted in dry gangrene of the limb, and it has been associated in a few cases with Raynaud's disease of the upper extremities.

In such cases as do not present any evidence of arterio-sclerosis or obliterative arteritis, it is assumed that the condition is due to arterial spasm.

The prognosis is bad, but the attacks may go on for years.

**Treatment.**—The patient must limit his exercise, and avoid quickening the circulation up to the point at which the obstruction in the vessel will begin to operate. Frequent rests in bed may be advisable. Iodide of potassium may be tried in syphilitic cases; and the local remedies used in Raynaud's disease

may be employed here, also the constant current, electric baths, warm baths, high frequency currents, and gentle massage.

If gangrene occurs, the pain may be relieved by morphia, and the part should be amputated at a suitable opportunity. The condition of the arteries, *i.e.* the amount of calcification present, may be determined by radiography, and this will help in deciding whether amputation should be performed high up or low down Gangrene and threatened gangrene have also been treated by removing the sympathetic nerves round the artery, so as to prevent arterial spasm and by injecting alcohol round the artery (**32**).

## ERYTHROMELALGIA

In this condition, first described by Weir Mitchell, there are attacks of acute pain in the feet and legs associated with, or followed by, dilatation of the blood vessels, the part becoming bright red or deep purple in colour, with shiny surface, prominent veins, and perhaps sweating. The upper limbs and trunk are also sometimes affected. The pain is acute, burning and throbbing. The attacks are brought on and aggravated by heat, exercise, and a dependent position of the limbs; and some relief is obtained by cold and by elevation of the limbs. The attacks last at first a few hours; but with the progress of time they are more persistent, and perhaps at the same time less severe It occurs in men of early middle age, and rarely in children.

Two of Mitchell's cases subsequently developed spinal symptoms, and other cases have been seen to be associated with tabes, syringo-myelia, and disseminated sclerosis. Some cases of erythromelalgia appear to be due to ergotism and thrombo-angiitis obliterans. In the latter disease the erythromelalgia has been associated with intermittent claudications, and it has been suggested that it acts as a compensatory mechanism whereby the blood supply to the part is increased by dilatation of capillaries. Erythromelalgia has been observed in the arsenical poisoning of beer-drinkers.

The treatment is mainly symptomatic: by cold, suitable position, and the use of morphia. Faradism and massage have also been of use.

*Acroparæsthesia.*- In this there are disagreeable or painful sensations, tingling or numbness, or "pins and needles" in the hands and feet. It may be accompanied by vasomotor disturbances. It is more common in women than in men. The disagreeable sensations are felt usually in one or both arms when the patient wakes in the morning, and after a time the symptoms subside. Sometimes the hand is paler or redder than normal, or even swollen. In a few cases there appears to be a sufficient cause in much use of the hands in some occupation during the day, or in a faulty position of the arms during sleep; but often no cause can be discovered. The pathology is not clear: it has been attributed to vasomotor spasm, but in many cases there is no evidence of this. It has been seen in general paralysis, tabes dorsalis, and allied disorders; but as a rule it is independent both of these and of hysteria. The treatment consists of rest, tonics, potassium bromide at night, and the constant current.

## ANGEIO-NEUROTIC ŒDEMA

This is an affection apparently connected with the vasomotor apparatus and closely allied to urticaria; but the lesions are larger. Circumscribed swellings appear in various parts of the body- for instance, on the face, the eyelids, the hands or legs, in the throat or in the tongue. They are not inflammatory, and not dependent upon gravity; they are not painful, but may be accompanied by burning, pricking and itching. They appear suddenly, last from two to six hours or more, and recur frequently, even daily; on the skin they are generally harmless, but œdema of the larynx has frequently proved fatal. Gastro-

intestinal symptoms are usually present, such as colic, nausea, and vomiting, and are attributable to an acute œdema of the gastric or intestinal mucous membrane. The disease is often hereditary, occurring in several members of the same family in two or three generations. It is one of the toxic idiopathies, like asthma (*see* p. 224), and is due to sensitisation to a foreign protein.

**Treatment** consists in finding out and avoiding the particular protein to which the individual is sensitive; septic foci must be eradicated. Quinine, nitroglycerine and thyroid extract have given relief in a few cases. Laryngeal œdema may require intubation or tracheotomy

# DISEASES OF THE MEDIASTINUM

## MEDIASTINITIS

Inflammation of the mediastinum may be suppurative or non-suppurative. The former, or *mediastinal abscess*, arises from numerous causes, of which injuries by bullet, stab, or blow, and tuberculosis of the lymphatic glands are the most frequent, while occasionally it follows upon pneumonia, pleurisy, erysipelas, or enteric fever The abscess may be in the anterior or in the posterior mediastinum, more often in the former. The chief symptoms are sternal pain and pyrexia. Physical signs will only be apparent if the abscess reaches a sufficient size, when there may be dulness, localised tenderness, œdema over the sternum, and ultimately fluctuation at the border of that bone. The pus must be evacuated as soon as possible, and, if necessary, the sternum must be trephined or a portion resected.

Adhesive or non-suppurative mediastinitis may also arise from traumatism and general infectious diseases, but its most common associations are pleurisy and rheumatic pericarditis, especially the latter, forming then a *mediastino-pericarditis*, or *mediastinitis fibrosa*. In these instances the tissues of the anterior mediastinum may be matted together to form a dense fibrous substance, and with this there are pericardial adhesion or thickening, sometimes pleural adhesion or liquid, and often ascites, with or without chronic peritonitis. The physical signs are those enumerated under Adherent Pericardium (*see* p. 359). The symptoms are dyspnœa, dropsy of the legs, enlarged liver and ascites, albumin in the urine, and sometimes the pulsus paradoxus. When the mediastinitis is pronounced and extensive, there may be cyanosis and œdema of the neck, face, and arms, and enlargement of the venules on the surface of the chest. The cases thus may present all the appearances of advanced mitral disease; or of heart disease without cardiac murmurs; or of cirrhosis of the liver with pleural complications, if the abdominal distension is out of proportion to the œdema of the legs; or of mediastinal tumour, if cyanosis and œdema of the upper part of the body are marked (*see* p. 386). Apart from these last, most of the symptoms can be explained by the cardiac dilatation and failure consequent on adherent pericardium; but some of the effects may be due to hampered movements of the diaphragm, demonstrable by X-rays, and possibly to fibrous tissue constricting the superior vena cava or the inferior vena cava between the right auricle and the diaphragm (Simon). The physical signs on the side of the heart may be very few: they are sometimes those which indicate hypertrophy and dilatation with adherent pericardium; and if adherent pericardium can be diagnosed on its own merits, the evidence of past or present pleurisy makes mediastinitis very probable. Signs of pressure on the trachea or œsophagus are not common. There may be a dull percussion note over the manubrium sterni if the lesions are extensive, or even, it is said, between the scapulæ. Penez described a rough, creaking

to-and-fro sound of friction, audible when the arms are moved upwards and downwards.

**Treatment.**—This resembles the treatment of valvular disease; purgatives and diuretics will have to be employed, and the ascites often requires paracentesis.

The operation of *cardiolysis* has given much relief in some cases. This consists in resection of the left fourth, fifth, and sixth ribs, perhaps also the third and seventh, with their costal cartilages, from the front of the heart, so as to liberate it in part from the tissues which impede its action.

## TUBERCULOSIS OF THE BRONCHIAL GLANDS

**Ætiology and Pathology.**—The former is similar to that of tuberculous infection in general; and the bronchial glands are infected commonly from the lesions of pulmonary phthisis, but are not much enlarged. However, in children the glands may caseate, enlarge, and even suppurate, and there is also infiltration at the roots of the lungs (*see* Hilum Phthisis, p. 256). In nearly all cases a primary focus can be found in the lung (*see* p. 127). The bronchial glands go through the same changes as other organs when invaded by tubercle. Grey granules are at first formed, and caseation follows; the glands become enlarged, and may suppurate or become calcareous. As a result of their enlargement they press upon surrounding parts, especially the œsophagus and the trachea or main bronchi. If they suppurate they may discharge into one of these passages, or they may lead to abscess in the mediastinum.

**Symptoms.**—Enlarged bronchial glands constitute a mediastinal tumour. They cause pain in the chest, cough, and dyspnœa, and perhaps slight swelling of the face and neck, dysphagia, and hæmoptysis. The cough is of most interest. It is sometimes harsh and altered in quality, as if the larynx were involved; and this is probably from some pressure of the glands on the recurrent laryngeal nerves, producing paresis of one or both cords. The cough may be constantly hacking, or spasmodic like that of pertussis. If the pressure on the trachea is considerable, there is severe dyspnœa with stridor, and impending suffocation; and this is especially likely to be the case if the glands are suppurating and rupture is imminent. When rupture takes place, pus is expectorated, and there is some danger of asphyxia in young children. Occasionally a detached fragment of gland has been impacted in the respiratory passages, and has caused death. The puffy swelling of the face and neck is due to the pressure on the superior vena cava or on one or other innominate vein. There is more or less pyrexia, according to the stage and the rapidity of the caseating process.

**Physical Signs.**—There is the possibility that the glandular mass, if of sufficient size, may cause dulness between the scapula and the spine on one or both sides; or in front over the upper part of the sternum. But this is probably very rare. The auscultatory signs are variable, even independent of any coexisting phthisis. There may be deficient breath sounds over the lung on one or other side, or bronchial breathing and bronchophony in the neighbourhood of the enlarged glands; but the occurrence of bronchial breathing over the upper dorsal vertebræ under normal conditions must not be forgotten (*see* p. 193).

**Diagnosis.**—The symptoms and physical signs of what is practically a mediastinal tumour in a young child, with a family history or other evidence of tubercle, such as the subcutaneous injection of tuberculin (*see* p. 258), may enable the disease to be recognised; but in a small child it may be easily confounded with empyema, until tested by exploration or by Röntgen rays. On the other hand, coarse physical signs may be wanting unless the enlargement is considerable: the Röntgen rays may then be helpful.

The **Prognosis** is not absolutely unfavourable. Health may be restored completely after the discharge of pus; and other cases, no doubt, get well on calcification of the tubercle.

**Treatment.**—This must be conducted on the same principles as that of tubercle in general. Good food, fresh air, and tonics, such as the syrup of iron iodide or of iron phosphate, Parrish's food, or cod-liver oil, should be given. Local stimulants, such as iodine, may also be useful, and cough and pain must be met by small doses of anodynes, Dover's powder, compound tincture of camphor, or syrup of poppies.

## MEDIASTINAL NEW GROWTHS

New growths in this situation arise commonly from the bronchial or posterior mediastinal lymph-glands (**33**), but it may be from the thymus, and possibly from the fatty or subserous tissues, or the periosteum. The most frequent form of new growth is a lymphoma or lympho-sarcoma; spindle cell sarcoma, carcinoma, gumma, and rarely dermoid cysts also occur. Inflammatory infiltration and abscess also produce many of the effects of definite tumours. In the mediastinum these tumours are adjacent to the great vessels, the trachea, the roots of the lungs, and the œsophagus. They may extend above the clavicle into the neck.

**Symptoms.**—These are, for the most part, due to the pressure of the new growth upon the important structures in the chest. This results in (1) pain; (2) obstruction of veins, arteries, air tubes or œsophagus; (3) compression of one or both recurrent laryngeal nerves; (4) displacement of organs; and (5) deformity of the chest.

The *pain* is variable: it may be felt behind the sternum, or in the back, or may radiate along the course of the nerves.

*Obstruction of the veins* is one of the most important indications of mediastinal tumours, since veins have little power of resisting the growth of tumours, which readily press their walls together, and even grow through them and project into the interior, or lead to thrombosis and fibrous stricture. As a result of obstruction of the superior vena cava, the veins of the head, neck, face, arms, and upper part of the chest are dilated, and if it occurs rapidly, these parts become immensely swollen from œdema, contrasting curiously with the lower part of the body. The blood, however, finds its way to the heart, by anastomoses under the skin between branches of the intercostal and the abdominal veins; and these become visible as a close network of small blue tortuous vessels on the chest, and large vertical tortuous veins on the abdomen. Even with this compensation, the venous current may be much obstructed, and, on stooping down or making any exertion, the face becomes still more congested and cyanosed.

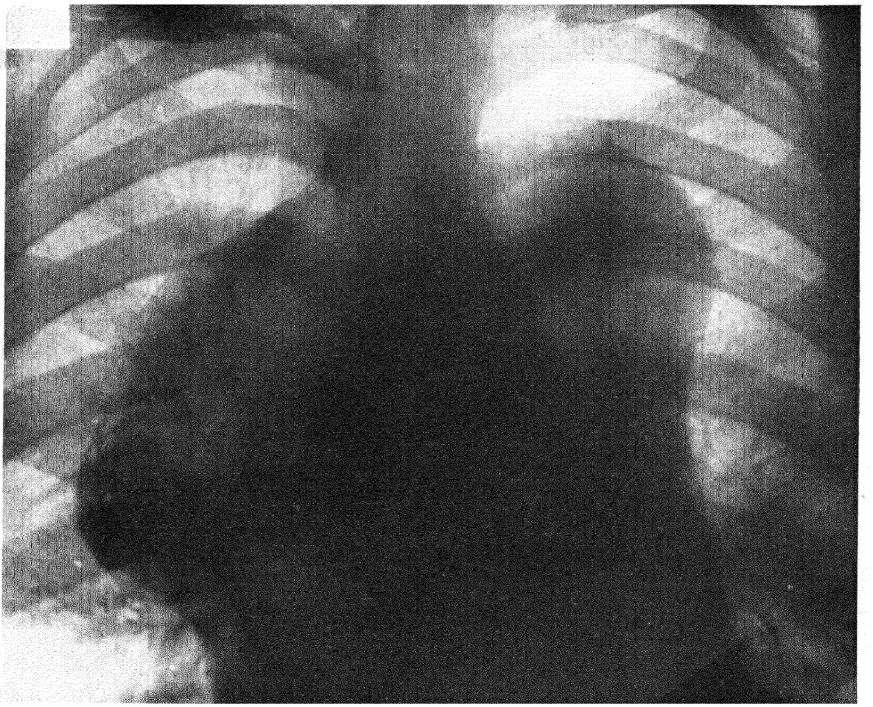
In cases of obstruction of the inferior vena cava a similar communication takes place, but there is a difference in that the flow of blood on the surface is entirely downwards in obstruction of the superior vena cava and upwards in obstruction of the inferior cava. This last, however, rarely results from mediastinal tumour, although it is possible for a malignant growth to reach the inferior cava, just above the diaphragm.

*Arteries* often maintain their course through a tumour unmolested. They are sometimes compressed, with the effect of weakening or obliterating the peripheral pulse.

The results of *compression of the trachea and bronchi* have been already described (pp 214, 230). If the *œsophagus* is involved, there will be difficulty in swallowing, and an œsophageal bougie will meet with resistance.

The paralyses of the vocal cords, which follow pressure on the *recurrent laryngeal nerves*, are similar to those caused by aneurysm. The left nerve is more

## PLATE XI.



Radiogram of Chest showing Mediastinal Growth, which has spread into both lungs. Note the sharply-defined margin of the growth. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 387.

exposed than the right from its lower position, and the results will be abductor paralysis, or complete paralysis, according to the degree of compression. Sometimes there are spasmodic attacks of dyspnœa.

The *displacement of organs* and *deformity of the chest* depend on the size and position of the tumour. The heart may be pushed to the right or left, the liver downwards towards the abdomen, or one lung may be compressed against the ribs. Deformity may be quite absent, especially if the tumour arises in the posterior mediastinum. But there may be enlargement of one side from effusion of fluid into the pleura, caused by the growth pressing on the veins in the root of the lung; or retraction of one side if the growth compresses the bronchus. Tumours in the upper part of the chest may extend into the root of the neck, or be accompanied by enlargement of cervical glands in this situation. Dyspnœa, spasmodic cough, expectoration of mucus, hæmoptysis, faintness, attacks of syncope, and palpitation are additional symptoms in many cases. Paraplegia occurs in exceptional cases from invasion of the spine.

The general condition of the patient is dependent often upon the extent and duration of the lesion. At first he may show little change, but cachexia and wasting must come in due time. Definite and prolonged *pyrexia* is sometimes present, as, for instance, in the following circumstances: if the growth is lymphadenomatous, there may be the characteristic recurrent pyrexia of Hodgkin's disease; carcinoma is sometimes attended by pyrexia, which is also liable to vary in intensity from week to week; if a bronchus is obstructed so that sepsis occurs within it, pyrexia will be caused.

**Physical Signs.**—The most important is the decided dulness which a tumour produces if it reaches the surface of the chest, so as to displace the lung, or if it compresses the lung between itself and the chest wall; but a large tumour may exist in front of the spine without appreciably altering the signs of percussion. The auscultatory sounds will depend on the presence or absence of fluid in the pleura, and on the relation of the tumour to the root of the lung (*see* Aneurysm, p. 375).

**Diagnosis.**—Mediastinal new growth is most readily confounded with an *aortic aneurysm*, which is itself a mediastinal tumour, occupying the middle or superior mediastinum when it grows from the arch, and the posterior mediastinum when it arises from the thoracic aorta. A diagnosis is, nevertheless, very desirable, since the prognosis as to duration of life and temporary relief of symptoms is much more favourable in the case of aneurysm. Many of the symptoms are the same; they both press upon and displace important structures, such as the œsophagus, the trachea or bronchi, and the large nerve trunks. One of the most important distinctions is the fact that aneurysms implicate arteries, and thus may lead to suppression of pulse on one or other side; while new growths commonly spare the arteries, but compress and project into the veins, producing venous distension and œdema. The presence of a murmur is in favour of aneurysm. Enlargement of the cervical glands or the existence of tumour in other parts of the body speaks for new growth; in this connection the mammæ or the testes should always be examined. X-ray examination provides the most certain means of differential diagnosis. An aneurysm has a clearly defined edge, which shows expansile pulsation. The shadow of a glandular neoplasm is also well defined (*see* Plate XI.); but there is no pulsation. The shadow of an irregularly infiltrating neoplasm gradually merges itself into the surrounding lung, and no true edge can be seen at all. The stridor produced by compression of a bronchus may be mistaken for the rhonchus of *bronchitis*. The former is constant in time and position; the latter is variable, and changes from place to place within a few hours, or disappears at intervals. Attention may be called to mediastinal pleurisy (*see* pp. 274, 277).

The **Prognosis** is bad, and the duration rarely more than a few months.

**Treatment.**—The rate of growth has sometimes been delayed by exposing

the chest to large doses of radium in the case of lympho-sarcoma; but usually little can be done beyond relieving pain by opium or morphia. However, deep X-ray therapy holds out hope, especially in sarcoma. Symptoms threatening life, such as laryngeal asphyxia, must be met as they arise.

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# DISEASES OF THE ORGANS OF DIGESTION

## EXAMINATION OF THE ABDOMEN

The abdomen is accessible to the same methods of examination as are employed in the case of the lungs and heart namely, inspection, palpation, percussion, and auscultation; and in most instances it is desirable that the patient should be in the recumbent position, with the head rested.

For descriptive purposes the abdomen is divided into nine areas by means of two horizontal and two vertical lines. The vertical lines are drawn upwards from the mid-point of Poupart's ligament on each side to the costal margin. The horizontal lines are drawn across from the lowest parts of the costal margin, *i. e.* the tenth costal cartilages on each side, and between the highest points of the iliac crests. The names given to the regions mapped out by these lines are in descending order—(1) in the middle, epigastric, umbilical and hypogastric; (2) at the sides, hypochondriac, lumbar and iliac. As in the chest, accurate localisation of a lesion requires measurement from easily recognised parts, like the umbilicus, xiphisternum, middle line, pubes, anterior superior iliac spine, or tip of the eleventh rib.

### INSPECTION

The first thing to notice is the size of the abdomen. This is extremely variable even within the limits of health. It may be uniformly much enlarged; but it requires the help of other methods of examination to determine whether this is due to a collection of liquid in the peritoneal cavity (*ascites*), to gas in the intestines (*meteorism*, *tympanites*), to fat in the parietes and omentum, or to some tumour, such as an ovarian cyst. Uniform and symmetrical retraction of the abdomen is seen in starvation, in emaciating diseases, and in death from cerebral diseases, such as tuberculous meningitis and intracranial tumour.

By inspection may be observed various local enlargements or prominences, such as result from tumours or enlargements of different organs. Many of the tumours which occur in the abdomen may, under favourable circumstances, be visible on the surface; this is much more likely to occur when the patient is thin, and the abdomen empty, than when the patient is fat, and the abdomen is distended with ascites or flatus. Amongst those more commonly observed are enlargements and tumours of the liver, dilated stomach, distended intestines in cases of obstruction, infiltrated omentum and adherent intestines in tuberculous peritonitis, enlarged spleen, hydronephrosis, the pregnant uterus, ovarian and other cysts, and a distended bladder. Local enlargements in the upper part of the abdomen may cause asymmetry of the thorax by driving upwards and outwards the lower ribs on one side, thus enlarging the angle between the costal margin and the middle line. This is especially seen in cases of hydatid, carcinoma, and abscess of the liver.

It is important to note the relation of the abdomen to the respiratory movements already referred to in connection with the chest (*see* p. 188); the descent of the diaphragm is impeded by much distension, and is checked by acute in-

flammation of the peritoneum, so that in these cases respiration is almost entirely thoracic. In other cases respiration affects materially the position of the organs in immediate contact with the diaphragm, namely, the liver, spleen, stomach, and kidneys, whereas organs or tumours situated lower down, or connected with the posterior wall of the abdomen, are much less influenced by the descent of this muscle. The pulsations of the aorta, or of the right ventricle, or of the enlarged liver in tricuspid regurgitation, or very occasionally of an aneurysm, and the peristaltic movements of a dilated stomach or of the intestines, may sometimes be seen, the latter being visible in proportion to the thinness of the abdominal parietes and the vigour of the peristalsis.

## PALPATION

For this method of examining the condition of the abdomen, the abdominal walls should be relaxed as much as possible, and hence the patient should be in the recumbent or semi-recumbent position, and the head should be supported, for if the patient raises his head—*e.g.* to see what is going on—the recti abdominis become tense. The relaxation of the abdominal walls is sometimes assisted by raising the patient's legs, but they must be supported in that position by a pillow under the knees. This procedure compels the observer to examine very much from one side, rather hampers the use of the hands, and can often be dispensed with.

In many patients the abdominal muscles are persistently tense, and the abdomen is palpated with difficulty. In such cases the patient should be asked to breathe deeply in and out, and should be engaged in conversation while the hand is on the abdomen, or he may be asked to lift up his head from the pillow, and keep it raised for about a minute, when the muscles will become exhausted, and for a moment afterwards the abdomen may be lax enough for the purpose. Another plan which has been recommended is to palpate the abdomen, when the patient is in a warm bath. If these means fail, and an examination is of the first importance, an anæsthetic should be administered.

The examination of the abdomen should be made with great gentleness; the hands should be warm, and should be laid flat upon the surface, and care should be taken not to force the finger tips suddenly into the abdomen, whereby the muscles are made to contract, and trustworthy results are impossible. During the movements of deep breathing enlargements of organs or new growths, especially in the upper part of the abdomen, may be detected, which would otherwise perhaps escape recognition. When examining the sides of the abdomen or the flanks, the observer should never neglect to employ the *bimanual* method—that is, one hand should be placed under the twelfth rib, and another on the abdomen in front; if one hand be pressed towards the other which is still, the slightest enlargement or resistance can generally be appreciated. In particular cases it may be desirable to examine the patient in the knee-elbow position.

In the normal abdomen there is scarcely any resistance to the movement of the hand in all directions. The solid organs, liver, spleen, and kidneys are almost entirely within the bony thorax; the left lobe of the liver, which lies across the epigastrium, is of small bulk, thin, and soft; the hollow viscera yield readily to the hand, and often nothing can be recognised, except, in thin people, pulsation of the aorta or iliac vessels.

By palpation in disease we can recognise changes in shape or size of the organs and the existence of tumours, and can obtain information on the following points:

The condition of tenseness or relaxation of the abdominal wall, which may be local or general.

The presence of tenderness, general or local; this may be elicited directly the hand touches the abdomen, or only when deep pressure is made.

Various kinds of movement may be felt in the abdomen—the pulsations of

the normal vessels, or of an aneurysm, or of the liver in heart disease; the peristaltic movement of the bowel; the movements of air in the intestine (borborygmi); the crackling or gurgling due to air and liquid, which may be felt sometimes on gentle pressure over the cæcum in enteric fever; the coarser movements or splashing of air and liquid in a dilated stomach when somewhat sudden pressure is made upon it; and the friction of inflamed peritoneal surfaces.

Under palpation also must be included two of the methods by which ascites or liquid in the peritoneal cavity may be recognised—namely, *fluctuation* and *displacement* (*see* Ascites).

## PERCUSSION

It is in reference to percussion especially that we must remember that the abdominal cavity extends up into the lower parts of the bony thorax. In health the abdomen is resonant over so much of the combined surfaces as corresponds to the intestines and to the stomach—that is, all the parts below the ribs, and the costal cartilages and lower ends of the ribs on the left side below the heart. It is dull over the parts which correspond to the liver and spleen—that is, for the liver the ribs of the right side below the upper border of the sixth in front, and the eighth at the side, and for the spleen the ninth, tenth, and eleventh ribs on the left side just behind the anterior axillary line; and with very light percussion one may recognise the extension of the left lobe of the liver across the epigastrium. The relative areas of dulness and resonance may be much altered by changes in the amount of gas in the hollow viscera, and the dull areas of the liver and spleen are moved downwards in inspiration and upwards in expiration. There is, further, much difference in the quality of the percussion note over the stomach and different parts of the intestine.

Alterations in the size of the liver and spleen, or the existence of solid tumours or cysts, will give rise to new areas of dulness, and such dulness will, as a rule, be accompanied by resistance appreciable by palpation. As constant reference to these altered conditions will be made under the diseases of the different organs, it is not necessary to specify them here.

In percussion also we have another valuable method of recognising ascites (*q.v.*).

## AUSCULTATION

Friction sounds are occasionally heard over the liver and elsewhere in peritonitis; abdominal aneurysms may be accompanied by murmurs. If the region over the cæcum is auscultated, sounds are heard at intervals due to the passage of intestinal contents through the ileo-cæcal valve. In acute inflammation the movements stop, so that auscultation may be of value in suspected acute appendicitis.

## EXAMINATION BY X-RAYS

The œsophagus, stomach and alimentary canal are examined by giving the patient a meal mixed up with some salt opaque to the X-rays, such as barium sulphate or bismuth carbonate or oxychloride. Full details are given under the respective organs. For the examination of the colon an opaque enema may also be used. Other organs may be examined by taking plates of the abdomen after the alimentary canal has been emptied as far as possible by laxatives. In this way the presence of gall stones may be determined in favourable circumstances. Recently successful results in mapping out the liver, gall bladder, spleen, kidneys and enlarged pelvic organs have been obtained after injecting oxygen through a cannula passed by means of a trocar through the rectus muscle to the left of and below the umbilicus. The oxygen is allowed to escape again through the cannula after the examination.

# DISEASES OF THE MOUTH, TONSILS, AND PHARYNX

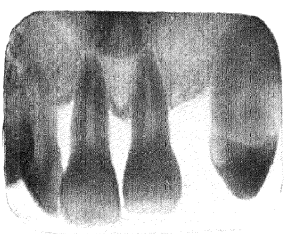
## STOMATITIS

Inflammation of the mouth, or *stomatitis*, occurs as a general catarrhal condition involving the cheeks, gums, tongue, and lips, and in more localised forms, as aphthous, ulcerative, and gangrenous stomatitis, which are almost certainly due to micro-organisms. At the same time it is clear that some special conditions are required for the operation of micro-organisms, since the mouths of healthy persons contain innumerable micro-organisms, among which are staphylococci, streptococci, torulæ, and sometimes pneumococci, and diphtheria bacilli. The lesions of some diseases of the skin may involve also the buccal mucous membrane, such as those of herpes, pemphigus, and urticaria. Stomatitis limited to the gums is called *gingivitis*. Different forms of gingivitis are seen in scurvy and in acute leukæmia, and as local results of dental diseases in the form of *pyorrhœa alveolaris* and *gumboil*.

**Oral Sepsis.**—It is necessary to lay especial stress upon the importance of a healthy condition of the teeth in relation, not only to stomatitis, but to conditions of general ill-health. There are two conditions which are important from the medical point of view. (1) *Apical abscesses*, which form round diseased stumps and the roots of dead teeth and teeth which have been the objects of much prosthetic work, and so have become devitalised. There is no free drainage, so that the infective micro-organisms readily pass into the blood stream, producing lesions in different parts of the body. There may be symptoms of general ill-health with fatigue and anæmia. It is probable that many diseases of obscure origin may in the future be traced to this cause. In particular rheumatoid arthritis, infective endocarditis, recurring gastric and duodenal ulcer, ocular inflammations, *i.e.* iritis, keratitis, choroiditis, may result from apical abscesses. Obviously dead teeth must be removed; but this is not enough, as an abscess may be present when the tooth looks healthy, and there may be no pain and no tenderness on tapping the tooth. The only safe way is to take dental radiograms, when the abscess is easily identified by the clear space at the root (*see* Plate XIII.). On the other hand, the mere presence of an apical abscess without other signs of ill-health does not necessarily indicate the removal of the tooth, since the abscesses are sometimes sterile. All the circumstances of the patient must be taken into account. (2) *Pyorrhœa alveolaris*, where there is chronic inflammation with absorption of the bone surrounding the roots of the teeth. Pockets are formed between the teeth; the floor of the pocket is relatively wide between the roots of the teeth, while the opening of the pocket is constricted, so that it becomes filled with food *débris* and pus (*see* Plates XII. and XIII.). In proportion as drainage is restricted, infection by the blood stream is apt to occur; when there is a free discharge of pus, the micro-organisms may be swallowed and produce various alimentary disturbances. However, blood stream infection is probably more dangerous than swallowing the organisms. According to Goodrich and Moseley, pyorrhœa is due primarily to the leptothrix of the mouth; but other organisms, streptococci, etc., are always associated with it, and are responsible for the various diseases secondary to pyorrhœa.

The *prevention* of oral sepsis consists in keeping the teeth scrupulously clean and free from tartar. At the same time the greatest care should be taken that the gum is not injured habitually by too hard brushing. The treatment of apical abscess consists in the removal of the tooth, and the same applies to well-advanced pyorrhœa, as there is no cure for this condition. When oral sepsis is causing a general infection it is most unsafe to remove more than one or two teeth at one

## PLATE XIII.



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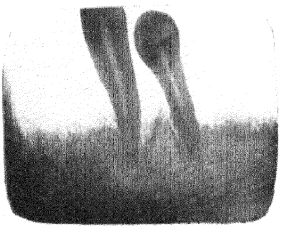

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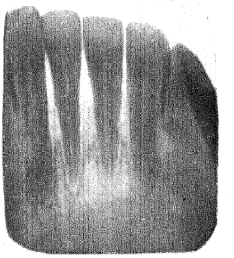
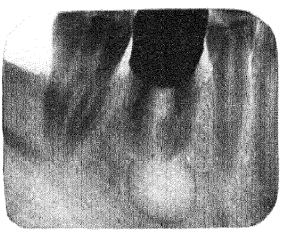


3.

FIGS. 1, 2 and 3.—Radiograms showing advanced Pyorrhœa Alveolaris. There is almost complete loss of the alveolus surrounding the roots.



4.

5.

6.

FIGS. 4, 5 and 6.—Radiograms showing Abscesses round the Roots of the Teeth. These are shown as clear areas into which the roots seem to project. In Fig. 6 the affected tooth contains a filling which has descended into the pulp cavity. The blunting of the tip of the root in the abscess cavity from absorption should also be noticed. The large clear space in this figure is the normal antrum (From films taken by Mr H. M. Worth.)

[*To face p.* 392.

time, since extensive removal may cause an exacerbation of the infection, in some cases with fatal results. It is also safest to scale and otherwise clean up all the teeth some days beforehand, as this will tend to prevent septic absorption. At the same time the extraction of many teeth should only be undertaken after the most careful consideration, especially in the case of poorer patients who may experience great inconvenience, if they cannot afford to buy satisfactory dentures. Palliative treatment consisting in cleaning out the pockets periodically is also adopted.

A solution of thymol in water is a valuable mouth wash, as it is a particularly powerful antiseptic. Thymol is very slightly soluble in water, so that it is only necessary to put two or three crystals into a bottle of water and leave it to stand for some time. If the external temperature is high, the solution becomes stronger, and causes a stinging sensation in the mouth, and so it must be diluted before use.

### CATARRHAL STOMATITIS

**Ætiology.**—Catarrhal stomatitis may be set up firstly by chemical or mechanical irritation, such as contact with acids or alkalies, excessive drinking, or the presence of broken or carious teeth; secondly, by inflammation spreading from adjacent parts, such as the nose or naso-pharynx; thirdly, by the action of some poisons, viz. mercury, lead, and arsenic; and fourthly, in consequence of some general and mostly infectious conditions, such as measles, variola, syphilis, scurvy, leukæmia, and others.

**Symptoms.**—These are swelling and increased redness of the mucous membranes of the gums, lips, and cheeks, swelling of the tongue, salivation and increased secretion of buccal mucus, which adheres as a coating to the surface, and swelling of the neighbouring lymphatic glands. Mastication and deglutition are painful, and the breath may be offensive. In later stages abrasion and superficial ulceration take place.

**Treatment.**—All causes of irritation should be as far as possible removed; and antiseptic washes should be employed, such as boric acid (2 to 5 per cent.); potassium chlorate (3 per cent.): in later stages much more astringent solutions, such as alum (5 grains to the ounce) or glycerine of tannin.

### APHTHOUS STOMATITIS

This occurs in children, especially about the time of the first dentition, and less frequently in adults: it consists in the formation of circular grey patches, or aphthæ, on the gums, tongue, and the inside of the lips and cheeks. They are from 3 to 5 mm. in diameter, slightly raised above the surface, and, though looking like vesicles, are really caused by a fibrinous exudation beneath the epithelium. After a time the epithelium is shed, and small ash-grey ulcers with narrow red margins are left. Children thus affected are restless and feverish, there is slight salivation, and sucking or mastication is painful. The ulcers commonly heal in a few days, but may recur frequently in some patients. In adults the aphthæ are rarely so numerous as in children.

**Treatment.**—Antiseptic washes and glycerinum boracis may be used. The application of nitrate of silver in adults at once relieves pain and often quickly cures.

### ULCERATIVE STOMATITIS

Ulcerative stomatitis is more common between the second dentition and puberty, but is seen at other periods of life. It has occurred in an epidemic form amongst soldiers in camp and prisoners in gaol, and it is probably due to micro-organisms, but none has certainly been identified as specific. In cases occurring among troops both in France and in English camps, Goadby found *Bacillus fusiformis* and *spirochætes*. It will be noted that these are the same as are present

in Vincent's angina. It attacks especially those who are in ill-health or badly nourished. It begins at the free margins of the gums, which become red, swollen, detached from the teeth, and may bleed on slight pressure, or in the movements of mastication. Ulceration then takes place, the ulcers being often deep, ragged, covered with a grey or yellowish purulent coating, and surrounded by a thin red margin. The process spreads irregularly over the gums, and involves the lips and cheeks as well. Sometimes the ulceration extends down to the periosteum, and superficial necrosis of the jaw results. The teeth are loosened, there is free salivation, and the cervical lymphatic glands are enlarged and tender. The tongue and palate are inflamed at the same time, but are not generally ulcerated. The constitutional symptoms are often rather severe, and pyrexia is present. Mastication and swallowing are, of course, painful and difficult.

The **Prognosis** is favourable, recovery commonly taking place in one or two weeks.

**Treatment.**—Goadby recommends swabbing the parts with a warm dilute solution of chromic acid, 1 in 200 to 1 in 400, followed by painting with a mixture of 20 grains of chloretone to the ounce of glycerine and spirit. Tinct. benzoin co. may also be applied to the ulcerated areas. Antiseptic washes, glycerinum boracis and hydrogen peroxide may also be used.

### GANGRENOUS STOMATITIS

This disease, also called *cancrum oris* and *noma*, occurs in debilitated children or those subject to bad hygienic conditions, or those who are recovering from infectious disease, of which measles and enteric fever are most common. It is due to bacterial infection. The changes are very rapid; a small spot of induration appears on the inner side of the cheek, and soon the whole thickness of the cheek is hardened, black in the centre, and reddened around, or, in other words, a slough has formed. If it goes on, the cheek will be perforated, or if it is on the lips, the gum will be invaded, and the teeth will fall out. There is very little pain or fever, but the child rapidly becomes exhausted and dies.

**Treatment.**—The only means of saving the child is the prompt destruction of the part by nitric acid, or its removal by the knife. In addition the child must be supported by food and stimulants.

### THRUSH

Thrush is seen in weak and badly nourished infants, especially in those who are being fed by hand, or are suffering from diarrhœa, and also in adults in the last stages of exhausting diseases, such as phthisis, carcinoma, and enteric fever. Upon the mucous membrane of the lips, cheeks, gums, palate, and tongue, milk-white patches occur, which are irregular in shape, scattered or confluent, slightly raised above the surface, and surrounded by a thin red line. If the patch is stripped off, the mucous membrane beneath is bright red, or even bleeds slightly, and the patch may form again in a short time. It consists of epithelial scales, fat globules, and the spores and mycelium of a fungus, *Oidium albicans*. According to Castellani thrush in the tropics may be due to many different varieties of fungi. The fungus develops first in the middle layers of the epithelium, and spreads thence in both directions to the more superficial and the deeper layers. It is probable that the growth of the fungus is the cause of the stomatitis which accompanies it; but it is stated by Vogel that the deposit is favoured by the secretions of the mouth, which are acid before any white patches appear. Children who have thrush and diarrhœa frequently have excoriations about the anus, which lead to the popular notion that the thrush has "gone through" the child; but though in severe cases thrush may extend to the pharynx and œsophagus, it does not occur on parts covered with cylindrical epithelium. The anal rash is either erythema intertrigo or a con-

genital syphilide. A certain amount of local discomfort, with pain on swallowing or sucking, results from thrush, but symptoms beyond these are chiefly due to the condition of health preceding it.

**Treatment.**—The general condition of the patient must be improved. In infants the food must be rendered suitable, and the diarrhœa checked. After every meal the mouth should be carefully wiped out with a fresh piece of soft linen, and the patches should be touched with a solution of borax (10 grains to ℥j), or a little glycerine of borax should be left in the mouth.

### LUDWIG'S ANGINA

*Ludwig's angina*, or *submaxillary cellulitis*, is a form of severe and sometimes fatal phlegmonous inflammation of the floor of the mouth, and upper part and front of the neck. It is due to infection from some part of the buccal cavity, such as a carious tooth, which is not infrequently the cause. Streptococci, staphylococci, and other organisms may be found. Prompt treatment by surgery is desirable.

## CATARRHAL SORE THROAT

**Ætiology.**—The reader is referred to the account of acute rhinitis on p. 196.

**Symptoms.**—In its mildest form there is only some discomfort on swallowing, and nothing may be visible in the throat. In other cases the soft palate, uvula, pharynx, and tonsils are redder than natural, the uvula is elongated, the soft palate is flaccid, and the pharyngeal veins are dilated. In more severe cases (*ulcerated sore throat*) superficial abrasions occur on the tonsils, palate, and pharynx, the tongue is furred, and there is marked constitutional disturbance. Sometimes there is excess of saliva and buccal mucus; at others there is unusual dryness of the mouth. Talking, as well as swallowing, may be painful.

**Treatment.**—For this we may use iron, quinine, or other tonics internally, and apply glycerine of tannic acid locally, or give rhatany or formalin lozenges. Where it is due to a hospital atmosphere with resulting infection, removal to the country for a few days may be desirable. (*See also* Acute Rhinitis.)

## TONSILLITIS

The tonsils are masses of lymphoid tissue situate one on each side between the anterior and posterior pillars of the fauces, and are now called the *faucial tonsils*, to distinguish them from other masses of lymphoid tissue, some lying at the base of the tongue, or *lingual tonsils*, and others in the naso-pharynx, the *pharyngeal tonsil*, or *Luschka's tonsil*. In the faucial tonsils the lymphoid tissue contains special groupings of cells and reticulum, called follicles, and presents deep fissures called crypts; these open upon the surface and run deeply down nearly to the capsule which lies in contact with the muscular substance of the pharynx. The space in which the tonsil lies is called the sinus tonsillaris, the upper angle of which is the supra-tonsillar fossa. In the crypts even in health may be found numerous species of bacteria, namely, streptococci, staphylococci, the pneumococcus, *Micrococcus catarrhalis*, the colon bacillus, the tuberculous bacillus, Friedländer's bacillus, and others. The tonsils exercise a protective action against infection from the mouth; but when they are infected, the infection may be transmitted to the cervical lymph glands, one of which, situate near the angle of the jaw under the anterior edge of the sterno-mastoid muscle, is distinguished as the tonsillar gland.

Inflammation of the tonsils has been already described in connection with certain infectious diseases, namely, diphtheria, scarlatina, syphilis, and acute rheumatism.

## FOLLICULAR TONSILLITIS

Apart from the infections just mentioned, inflammation of the lymphoid and follicular substance of the tonsils arises apparently spontaneously and in some persons occurs repeatedly at intervals of months or years.

It is probably due to infection, or to increase of virulence of organisms latent in the crypts, combined with depression of the resisting powers of the individual or his tissues.

**Symptoms.**— The tonsil is red and swollen, and presents several yellow or white prominent spots, which are masses of purulent exudate, exfoliated epithelium, polymorphonuclear leucocytes, lymphocytes and bacteria; and the surface is covered with more or less mucus. The swelling can be felt externally behind the angle of the jaw. In severer forms the secretion of the crypts is more abundant, and they are distended with large bright white plugs, which may present a close resemblance to the white material of diphtheria. Both tonsils are frequently affected. There is moderate constitutional disturbance, furred tongue, feeling of malaise, local discomfort, and pain on swallowing. There is often a considerable rise of temperature. As a rule, the tonsillar cervical glands are enlarged.

**Morbid Anatomy.** The cells of the parenchyma of the tonsils and of the follicles are increased in number, and very small abscesses may form in the follicles, and burst into the crypts.

**Diagnosis.**—The occasional resemblance to *diphtheria* is most important. Generally the obvious formation of the plug of secretion within a crypt of the tonsil, or the existence of several on each side, serves to distinguish them. A single white patch of some extent, apparently only on the surface, and an extension of this to the soft palate are in favour of diphtheria or Vincent's angina. Bacteriological cultivation should be employed in doubtful cases (*see* p. 69).

**Treatment** should be nourishing and stimulating, internally quinine and perchloride of iron, and port wine in weakly individuals. The tonsils may be painted with astringent or antiseptic solutions, such as glycerine of tannic acid, tincture of perchloride of iron in glycerine (5 drops to ℥j), liquor sodæ chlorinatæ, chinosol (1 in 500 of water), formalin (2 per cent. in glycerine), solutions of alum, or boric acid. Lozenges of potassium chlorate or rhatany and formalin tablets may also be sucked. Tincture of guaiacum, or tablets of guaiacum, and sodium salicylate are also useful.

## PERITONSILLAR ABSCESS

### (*Quinsy*)

In this condition suppuration occurs between the capsule of the tonsil and the muscular wall of the sinus tonsillaris. The exact position of the abscess in the sinus varies, but it mostly occupies the upper two-thirds of that space, pushes the tonsil downwards and inwards, and encroaches on the palatal tissues.

**Ætiology.**—This is most common between the ages of fifteen and twenty-five; some persons are very liable to it, and have it repeatedly. It is sometimes due to infective conditions of the tonsil itself; in other cases it is less easily explained. Both this and follicular tonsillitis are regarded by some as being part of acute rheumatism, even when occurring quite independently of the articular lesions.

**Symptoms.**—It may affect one or both tonsils. The tonsil becomes red and swollen to twice its natural size, projecting to the middle line, and pushing the uvula aside; if both tonsils are affected, they may meet in the middle line, driving the uvula forwards; the swelling and redness extend to the base of the soft palate. The surface is generally smooth, shining, and deep red or purple in

colour. Externally there is obvious swelling behind the angle of the jaw. The illness often commences with a rigor and sickness, and the constitutional disturbance is considerable. The tongue is thickly furred, appetite is lost, and the temperature rises to 103° or 104°. Swallowing and talking are excessively painful, and saliva and mucous secretion collect in the mouth, and require to be constantly expectorated. In from two to four days suppuration occurs; the tumour, which was at first hard, is now softer, and yields to the finger; or the presence of pus may be detected by placing one finger on the tonsil and another outside behind the angle of the jaw. If left alone, the abscess bursts into the throat, the temperature falls, and recovery quickly takes place in from four to seven days, though convalescence may be protracted for some time longer. Rarely the abscess has burrowed into the neck or chest, or eroded the carotid artery, or caused suffocation by discharging its pus into the larynx.

**Diagnosis.**—Quinsy may resemble *follicular tonsillitis*; it is more often unilateral, the fever is more severe, the redness extends to adjacent parts, secretion does not accumulate in the crypts, and pus may be eventually detected. Sometimes the two occur together.

**Treatment.**—In the early stages, ice often relieves the pain; it should be sucked as well as applied to the throat externally. Salicylate of sodium in 10 to 15-grain doses every three or four hours may be given internally, and guaiacum lozenges (3 grains in each) may be sucked every two hours, to diminish the acute symptoms. If suppuration has commenced, hot fomentations and poultices probably hasten it. When pus is detected, an incision should be made into the prominent part of the abscess with a bistoury, covered up to the last half-inch with plaster, so as to protect the other parts of the mouth. It may be less painful to incise in an upward direction through the upper part of the supratonsillar fossa than directly through the palatal tissues. The patient is generally confined to bed, and can take only fluid food. This should consist of milk, and stimulants are usually required. In later stages quinine and iron may be given in full doses.

### VINCENT'S ANGINA

This inflammatory condition is found in two forms: (1) ulcerative; (2) pseudomembranous, resembling diphtheria. It may spread from the tonsil on to the surrounding mucous membrane. Sometimes it does not affect the tonsil, but instead the soft palate or pillars of the fauces. The cervical glands are swollen, and there are difficulty of swallowing and some fever, and the breath is fœtid. Usually in eight or ten days the membrane disappears. The fever is but slight, the glands never suppurate, and the prognosis is good.

Two organisms are found in these cases: (1) the *Bacillus fusiformis*, which is longer and broader than the diphtheria bacillus, stains best with methylene blue, and not at all with Gram's solution. It is pointed at the ends, bulging in the centre, and measures from 6$\mu$ to 12$\mu$ in length. (2) *Vincent's spirochæte*. Local antiseptics, such as silver nitrate, zinc sulphate, tincture of iodine, trichloracetic acid, and solution of hydrogen peroxide, have been used in the treatment of the angina.

### CHRONIC ENLARGEMENT OF THE TONSILS

**Ætiology.**—This is of common occurrence in children, being due to chronic infection; some, it is true, are weakly in other ways: others maintain good health. Sometimes it can be traced to previous attacks of sore throat; on the other hand, those who have chronic enlargement of the tonsils are liable to temporary acute attacks. It often subsides as the patient approaches middle age, if not earlier. It must be noted, however, that the projection of the tonsil beyond the pillars of the fauces is not a safe measure of the size of the gland: some large tonsils are deeply buried.

**Pathology.**—The change in the tonsil is one of hypertrophy of the parenchyma and follicular tissues with more or less accumulation of secretion in the crypts.

Chronic infection of the tonsils is of importance, as it may provide a focus from which other parts of the body may be infected through the blood stream. Much of what has been said concerning apical abscesses of the teeth also applies here. Dental sepsis is usually more important in adults, while the tonsils are more important as sources of infection in children. In both cases secondary infection is the more likely to occur because the primary focus is deep-seated. In fact, Gardiner regularly obtained cultivations from the deep aspect of tonsils removed at operation. It does not necessarily follow that chronic septic tonsils are always large. They may be quite small and buried between the pillars of the fauces. Acute rheumatism and nephritis may often be secondary to septic tonsils, and they may provide an entrance through which tubercle invades the body; in fact, some enlargements of the tonsil are due to tubercle, and actinomycosis of the tonsil has also been observed.

**Symptoms.**—The tonsils are large, pale pink, lobulated on the surface, and firm in consistence, and pus can be squeezed out on pressing with a spatula. When only of moderate size, they may cause no local symptoms. In other cases the tonsils obstruct the passage of air from the nose through the pharynx, and the breathing is at all times somewhat snoring. The child breathes with the mouth open, and, the nasal passages being little used, the anterior nares are small, and the alæ compressed. Swallowing is laborious and clumsy, and speech is suggestive of something being in the mouth. Hearing is also deficient, from catarrh of the Eustachian tube; and taste and smell are said to be affected. The same symptoms may, however, be due to *adenoid* growths in the naso-pharynx, *i.e.* hypertrophy of the pharyngeal or Luschka's tonsil. Cough, nasal catarrh, restlessness, and headache are other symptoms observed in such cases.

**Treatment.**—The general health should be maintained by cod-liver oil, iron, and other tonics, including sea air. Local applications are of little service. The complete removal or enucleation of the tonsil—*tonsillectomy*—is advisable, where it is thought to be a primary focus of infection.

## NEW GROWTHS OF THE TONSIL

The malignant growths affecting the tonsil are carcinoma and sarcoma, usually lymphosarcoma. The former is often primary, but is sometimes secondary to a growth in an adjacent part. At first infiltrating, it soon ulcerates, discharges pus, and the cervical glands are early involved.

## LINGUAL TONSILS

The lingual tonsils are two or three nodules of lymphoid tissue, situate at the base of the tongue on either side of the middle line. They have the same structure as the faucial tonsil with two or three crypts. They may be inflamed like the faucial tonsils, less commonly have retention of secretion in the crypts, but occasionally hypertrophy, and this more frequently in children than in adults. It is probable that the sensation of "sore throat," which is felt in the middle line and somewhere at the back of the throat, is really in many cases due to inflamed lingual tonsils. The latter are also responsible for the common "tickling in the throat" that gives rise to a "throat" cough, and for the cough in whooping cough. Cough may be allayed by applying to the tonsils a saturated solution of 240 grains of ferric chloride made up to 1 oz. with glycerine, using a wool holder bent at the end. The tongue must be protruded as far as possible during the application (**1**).

## PHARYNGEAL TONSILS

This is a mass of lymphoid tissue situated in the naso-pharynx, together with scattered nodules in the mucous membrane of the fossæ of Rosenmüller and of the posterior wall of the pharynx. The mass may be sessile or pedunculated, consisting of finger-like processes, separated by fissures or clefts, analogous to the crypts of the faucial tonsil. It is covered with a layer of columnar ciliated epithelium. Enlargement is common between the third and tenth years of life; it may follow the infectious diseases of children, and is often associated with attacks of catarrhal rhinitis.

The pathological results of hypertrophy of the pharyngeal tonsil, often called *adenoid growth* or *adenoids*, are important. Chronic catarrh may spread up the Eustachian tube, and cause otitis media, and later chronic inflammation of the mucosa. In the course of the growth of the child occur some of the changes referred to under the head of the faucial tonsils. The face is lengthened; the alæ nasi are collapsed; the upper lip is short and retracted; the mouth is often kept open, and a vacant expression is thus acquired: these constitute the *adenoid facies*. Pigeon-breast and a high palatal arch are often present also.

**Symptoms.**—These, of course, vary with the amount of enlargement and consequent obstruction to the naso-laryngeal passage. They are deafness, habitual mouth-breathing worse at night, snoring and "night terrors," and liability to catarrhal rhinitis, with occasional blood in the secretion. In articulation the consonants M and N are badly pronounced, because these sounds cannot be resonated in the nasal meatus. Laryngismus stridulus, nocturnal enuresis, stammering, epilepsy and infantile convulsions are said by some to be excited by adenoids.

**Treatment.**—If the symptoms are pronounced, the growths should be removed surgically. Slighter cases may be improved by breathing exercises, aiming at teaching the child to breathe through the nose.

## CHRONIC PHARYNGITIS

**Ætiology.**—Chronic inflammation of the pharynx may arise from repeated acute attacks, but more frequently results from certain injurious influences, such as the abuse of alcohol, excessive smoking of tobacco, and the continual use of the voice; and when a public speaker inclines the head downwards in addressing an audience on a lower level, and consequently compresses the parts engaged in vocal utterance, he undoubtedly favours its occurrence. It is constantly associated with a similar change in the soft palate, tonsils, or posterior part of the nose.

**Symptoms.**—The mucous membrane may be reddened, with dilated veins; in some cases there are numerous small grey elevations scattered over the pharynx (*granular pharyngitis*); in others small abrasions or ulcerations occur. The grey projections in granular pharyngitis are the enlarged follicles or mucous glands. In some cases the mucous membrane is covered with increased secretion, and the patient is constantly hawking and spitting; in others the surface is dry, and a certain amount of discomfort and difficulty in swallowing, with pricking pain and desire to cough, is the result.

*Granular pharyngitis* is often spoken of as a distinct affection. It may spread beyond the fauces proper to the top of the pharynx and to the larynx; the mucous membrane is in most cases dry, but sometimes the follicles are covered with viscid mucus. It may cause little or no discomfort; but there may be stiffness and dryness of the throat, constant desire to hawk and spit, and distress and difficulty in swallowing. The effort to talk is also painful, and the patient may be obliged to stop to clear the throat. This condition of things is not uncommon in clergymen, public speakers, and others of like vocation, and has

consequently been called "clergymen's sore throat." The symptoms are aggravated by exposure to cold, and an inherited disposition has been observed by some writers.

**Treatment.**—Local treatment is necessary in granular pharyngitis. Gargles are of little use, as they do not reach beyond the soft palate; but sprays of alum or tannin (4 to 10 grains of each to the ounce of water) may be employed, or the throat may be painted with astringent solutions, such as nitrate of silver (40 grains to the ounce), solution of perchloride of iron (℥j to ℥j), or tannin (℥j to ℥j), or Mandl's pigment of iodised glycerine (iodine, 6 grains; pot. iod., 20 grains; ol. menth. pip., 5 min.; glycerine to an ounce). If these fail, the granulations must be destroyed; and this is best done by the galvanic cautery or Paquelin's thermo-cautery, each nodule being successively touched. This may, of course, require several sittings; the resulting inflammation is checked by sucking ice for some hours afterwards. Anæmia, dyspepsia, and gouty tendencies must be met by suitable treatment.

## RETROPHARYNGEAL ABSCESS

This, though chiefly a surgical complaint, requires short notice here, since it is apt to complicate the diagnosis of some throat complaints, especially laryngeal obstruction. It arises from caries of the spine, or more often from inflammation of the retropharyngeal lymphoid tissue; and it forms a swelling at the back of the pharynx, which may press upon the larynx so as to cause dysphagia, dyspnœa, and asphyxia. Thus it may be mistaken for croup or laryngeal diphtheria, but the cough and voice are not husky and hoarse, as in the latter, but rather "gurgling." In a suspected case the finger should be passed to the back of the throat, when a fluctuating swelling will be felt. It should be opened by the surgeon.

## PAROTITIS

*Primary* or *specific parotitis*, or mumps, has been already described among the infectious diseases (*see* p. 46).

*Secondary parotitis* is an acute form of inflammation which arises in the course of severe illnesses, such as pyæmia, fevers, dysentery, phthisis, and carcinoma. It is also the result of infection from micro-organisms contained in the mouth, and reaching the gland through Stenson's duct. This is apt to occur when febrile patients are kept for a long time on sloppy food, so that there is no mastication and the flow of saliva is diminished. Suppuration is much more common in this form; several small abscesses are formed, and afterwards run together. They may discharge externally, behind the ramus of the jaw, or burst into the external auditory meatus, or burrow deeply down the neck, or behind the pharynx. They sometimes slough.

The **Treatment** is that of acute local inflammations; fomentations will relieve pain, and when pus is recognised, an incision must be made; but recovery depends much upon the primary illness. Prophylactically, patients should at intervals be given something to suck, so as to flush out the glands and ducts.

## CHRONIC ENLARGEMENTS OF SALIVARY GLANDS

Besides being invaded by new growths, the parotids may be infiltrated with lymphocytes as part of Hodgkin's disease, or of lymphatic leukæmia.

*Mikulicz's disease* is a chronic bilateral and symmetrical enlargement of the lachrymal and salivary glands, due to infiltration with round cells, in a subject apparently healthy in other respects. There is thus no leucocytosis, but even some leucopenia, though there may be a relative lymphocytosis. The disease is presumably due to infection, and may recover under arsenic, or iron, or potassium iodide. Recently success has been obtained with deep X-ray therapy.

# DISEASES OF THE ŒSOPHAGUS

## ŒSOPHAGITIS

The œsophagus is much less liable than other parts of the alimentary canal to the various forms of inflammation. It may be injured by chemical substances or hot fluids, or inflammation may extend to it from neighbouring parts. *Chronic* inflammation results from the pressure of tumours, and from valvular disease of the heart. It produces thickness and opacity of the epithelium, or actual warty growths, or in some cases dilatation of the veins and desquamation of the epithelium.

## OBSTRUCTION OF THE ŒSOPHAGUS

This is the most important pathological condition of this part of the alimentary tube. The causes are—impaction of foreign bodies, such as false teeth; compression from outside by mediastinal growths and thoracic aneurysms; the growth of carcinomatous or other tumours in the walls of the tube itself; constriction by the contraction of ulcers following injury by corrosive poisons; functional spasm of the muscular walls; and achalasa of the cardia or cardio-spasm. The last four conditions will be separately considered.

### CARCINOMA OF THE ŒSOPHAGUS

This generally occurs in advanced life, and in males more often than in females. The growth occupies the middle and lower thirds of the œsophagus much more often than the upper third; but it is especially frequent opposite the bifurcation of the trachea, and at the cardiac extremity of the œsophagus. It is always primary. In course of time it forms an irregular ulcerated surface on the inside. The tumour partially or completely encircles the tube, extending vertically from 1 to 4 inches; moreover, it often involves the trachea or the root of the lung, or compresses the recurrent laryngeal nerves. The mediastinal lymph glands are enlarged, and not infrequently quite early the cervical glands.

**Symptoms.**—The first and prominent symptom is dysphagia. The patient finds he has difficulty in swallowing solids when he may get fluids down with comfort. The difficulty increases gradually, and at length solid food has to be given up; liquids can alone be taken, and if more than a mouthful is attempted at a time, it is regurgitated, and the patient may choke. Pain is usually absent. After a few weeks the patient begins to waste, and loses strength and energy. The symptoms are generally progressive, but occasionally temporary improvement takes place from crumbling away of portions of growth from the surface, so as to enlarge again the calibre of the œsophagus. If no relief be afforded, death takes place from simple exhaustion, or from complications. Thus in some cases a communication with the trachea is produced by the spread of the growth; food particles are inhaled, and a septic broncho-pneumonia is set up. In others the lung is directly invaded by the new growth, and gangrene or broncho-pneumonia, with which pleurisy or empyema may be associated, carries off the patient. In others, again, compression of the recurrent laryngeal nerves leads to paralysis of the abductors of the glottis, which may produce asphyxia. Rarely a growth has eaten into the aorta and caused fatal hæmorrhage. Lastly, there may be deposits in other organs, especially in the liver and lungs. Occasionally these are the cause of death, when the growth in the œsophagus has been too slight to produce any difficulty in swallowing.

**Diagnosis.**—Gradually increasing dysphagia in a person over fifty years of age is, in the great majority of cases, due to carcinoma of the œsophagus. Sometimes the fact of dysphagia may be overlooked: food may be retained sufficiently

long in the œsophagus above the stricture for its regurgitation to be mistaken for vomiting, both by the patient and by a careless inquirer; and so a gastric lesion may be diagnosed. If the patient complains of vomiting immediately after food, he should be asked to drink in the presence of the doctor, when it may be observed that he only takes a mouthful at a time, and waits as if expecting some to come back. The patient can usually tell the exact level at which obstruction occurs.

The presence of an obstruction can be most readily confirmed by the use of X-rays after a bismuth meal (*see* p. 391), when the exact position and extent of the obstruction may be demonstrated. The rays will also show whether the lesion is within the œsophagus or is due to an aneurysm or other tumour pressing upon it (*see* Plate XIV.). When the obstruction is situated in the œsophagus it is still necessary to distinguish between carcinoma, cicatricial or spasmodic stricture, diverticula, and achalasia of the cardia. Direct inspection by the *œsophagoscope* may be employed. In achalasia the œsophagus is greatly dilated, but there is usually not much dilatation with growth, because in the latter case the obstruction comes on more acutely. A closed hollow tube containing mercury, to make it heavy, may be passed down the œsophagus (Hurst). It will not pass in the case of growth but will pass in the case of achalasia. The presence of enlarged and hard cervical glands is also in favour of growth.

**Prognosis.**—This is uniformly bad. Even if the obstruction is overcome, the malignant growth must be fatal by its further extension within a short time. Evidence of pneumonia at one base or marked fœtor of the breath shows that the end is not far off. The duration is generally from six to twelve months.

**Treatment.**—If a bougie, even of small size, can be passed, the passage may be kept even for a time by its use every two or three days. But the maintenance of a channel for the food is best secured by some modification of the method of intubation introduced by Krishaber. A tube is passed through the stricture and retained *in situ* for several days or permanently, and the patient is supplied by that means with fluid nourishment. If these measures are inapplicable, the stomach may be opened by the operation of *gastrostomy*. A diminution of the constriction is sometimes obtained by the use of radium applied locally to the growth of the œsophagus.

### CICATRICIAL STRICTURE

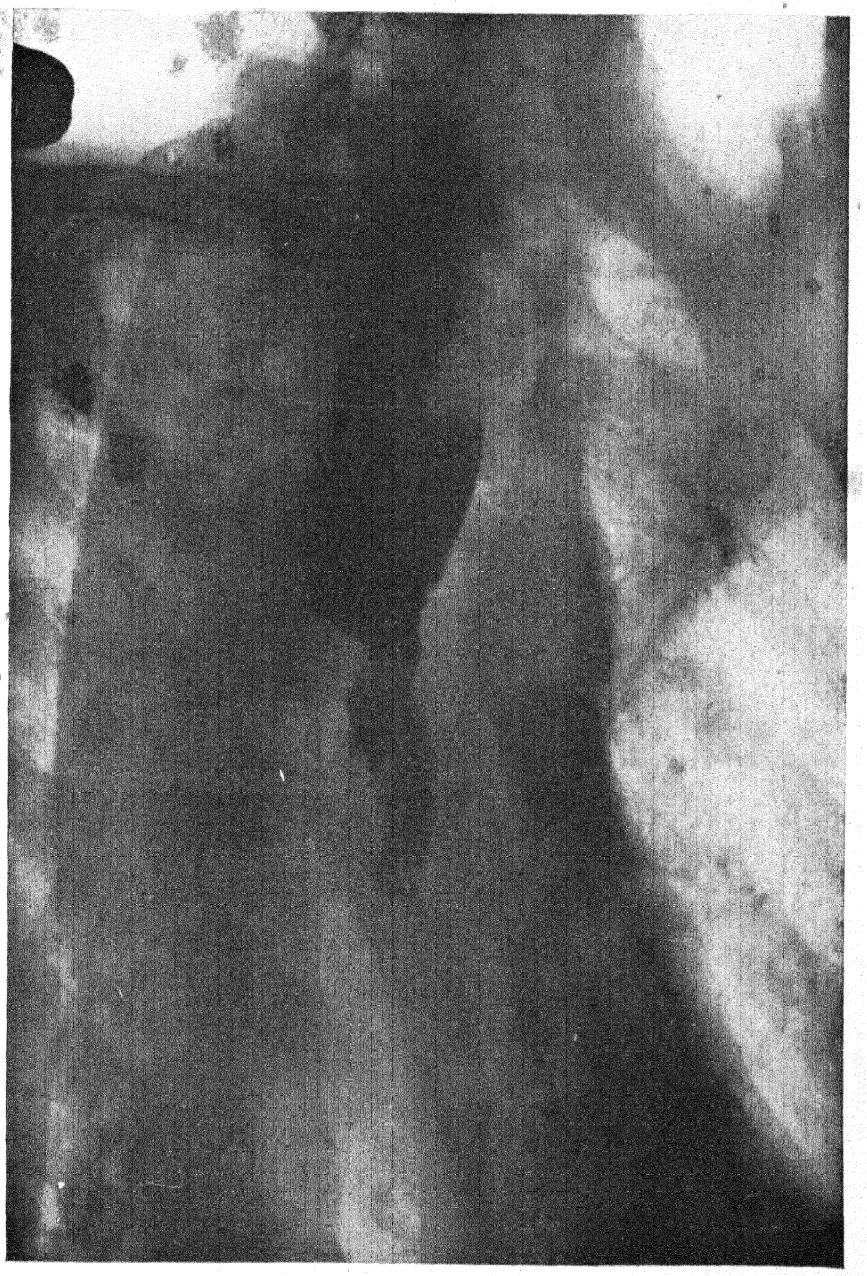
In this, again, dysphagia is the main symptom; but it differs from carcinoma in this, that it may not advance beyond a certain point, and that it does not lead to any secondary effects, except dilatation of the tube above it. In consequence of this dilatation food often accumulates above the stricture, and is regurgitated after a time.

The **Diagnosis** is generally determined by the history and the absence of other symptoms. Carcinoma would be excluded if the patient were young.

**Treatment** offers a fair chance of success if the sound or mercury tube can be passed through the stricture into the stomach. It should be used regularly once or twice daily, and attempts should be made to pass larger and larger instruments. Liquid food may be required always. In unfavourable cases gastrostomy may be advisable.

### SPASMODIC STRICTURE

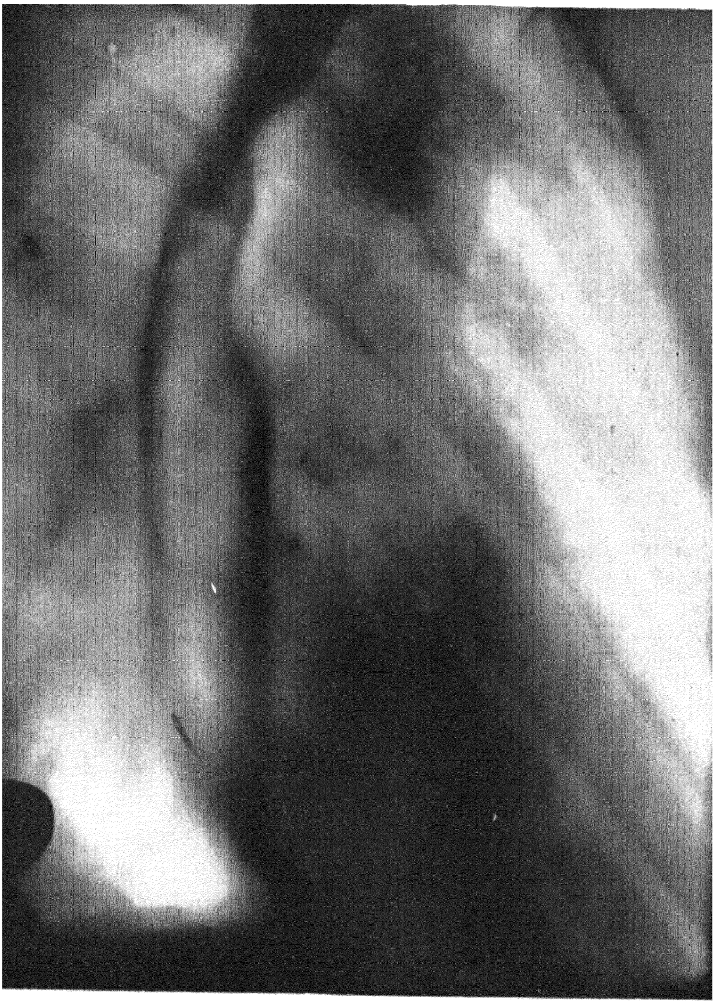
This is really quite a common condition, although it is not generally realised. There is difficulty in swallowing, accompanied by a painful sense of constriction in the throat and chest (heart-burn). The spasm can be recognised by X-rays (*see* Plate XV.). It may be a feature of the dyspeptic symptom complex (*see* p. 409) and so may be associated with aerophagy. The pain is probably due to the associated œsophageal distention. As the radiogram shows, the spasm is not absolute; some of the opaque meal manages to get through.



Radiogram of a Carcinomatous Stricture of the Œsophagus at the level of the Bifurcation of the Trachea, taken in the oblique position. The upper end of the œsophagus is shown full of barium sulphate emulsion, swallowed by the patient. A thin streak of barium is shown passing down the œsophagus from the bottom of the dilated part. The irregularity of lumen due to the growth is well shown. The shadow in front of the barium is the arch of the aorta. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 402.

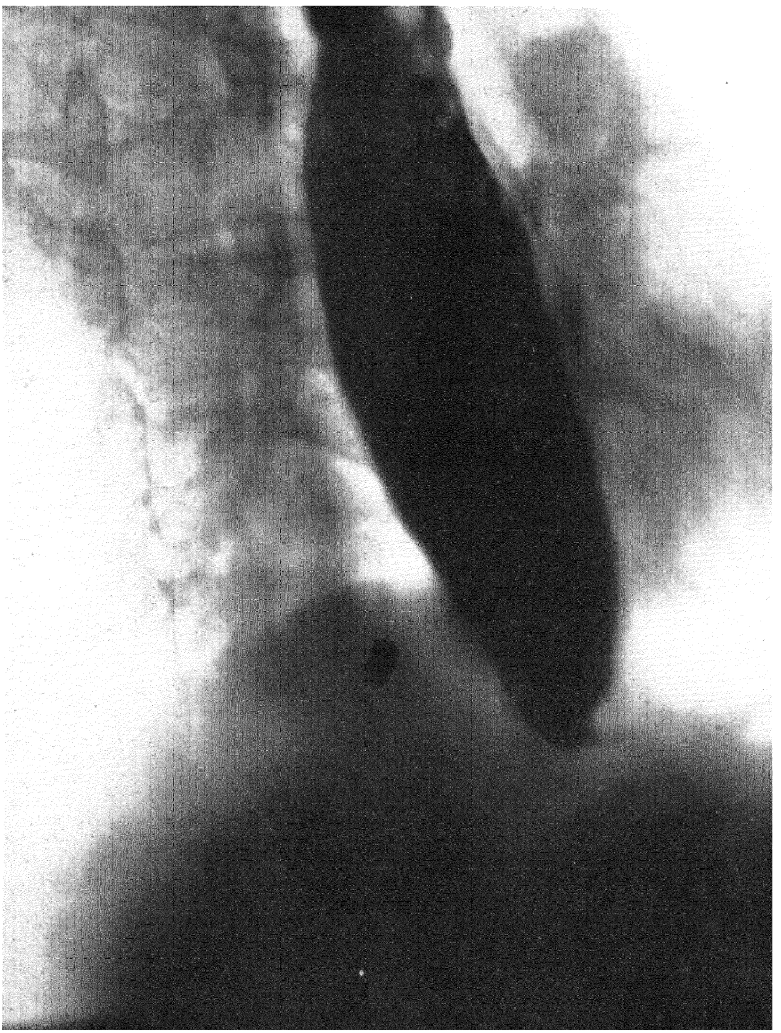
PLATE XV.



Radiogram of Œsophagus in Heartburn viewed obliquely. The œsophagus is distended with air, and there is spasm at both upper and lower ends. (W. W. Payne and E. P. Poulton (**2**).)

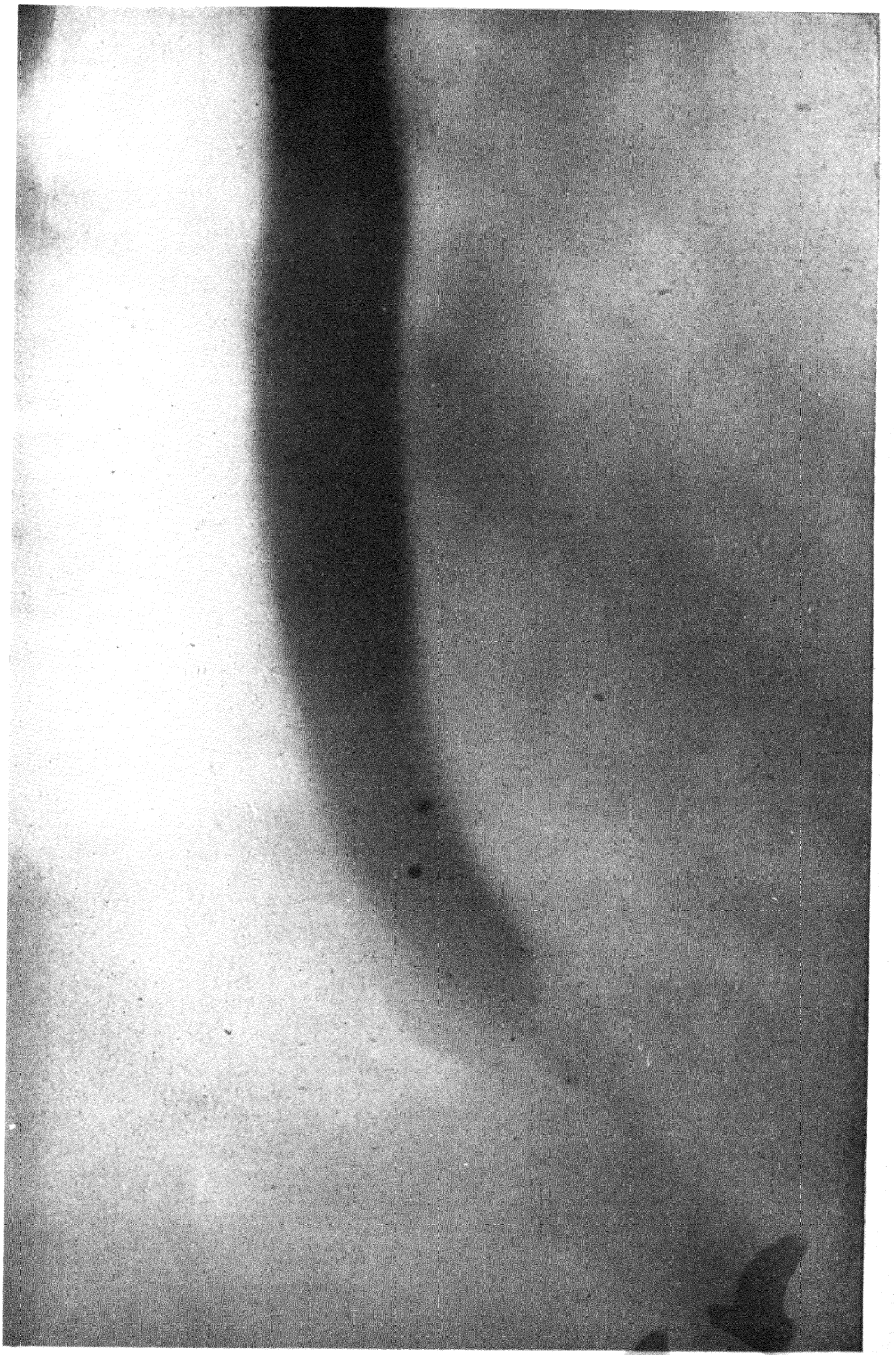
[*To face p.* 402.

# PLATE XVI.



Radiograms of Œsophagus, viewed obliquely, to show dilatations of the œsophagus with achalasia of the cardia. The narrow part at the bottom is at the entrance to the cardia. The shadow in front of the vertebral column is the right diaphragm. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 103



As a contrast to Plate XVI. this Radiogram of a Normal Œsophagus is included. A rapid exposure was made two or three seconds after swallowing a mouthful of barium, and before the cardia had relaxed to allow it to sink into the stomach. The double shadow of the cardia, viz., the narrow streak of barium at the bottom, is due to the alteration of its position during the exposure owing to the weight of the column of barium. (W. W. Payne and E. P. Poulton.)

[*To face p.* 403.

## ACHALASIA OF THE CARDIA

*(Cardio-spasm, Œsophagectasia, Idiopathic Dilatation of the Œsophagus)*

The lower 2 or 3 inches of the œsophageal wall, which may often be seen at *post-mortems* in a relaxed condition a little thicker than the wall above, is the cardial sphincter. In life the cardia is closed by contraction of both its longitudinal and circular muscle fibres; it relaxes in front of each peristaltic wave and subsequently contracts firmly again, probably becoming invaginated a little way into the stomach during the process (**2**). Peristalsis travels rather slowly from the upper end of the œsophagus after swallowing, while a fluid meal is projected straight down. Consequently the bulk of the fluid remains some seconds above the sphincter, before the latter relaxes.

**Pathology.**—In achalasia of the cardia (α priv. and χάλασις, loosening, slackening) relaxation of the cardia never takes place (**8, 9**). This is probably a more accurate term than cardio-spasm, because "spasm" suggests a prolonged tightening of the sphincter. A temporary tightening normally occurs when a peristaltic wave reaches it. Since stimulation of the vagus in the rabbit relaxes the cardia, it is possible that some local lesion in the medulla picking out part of the vagal nucleus may account at any rate for some of the cases. Pressure on the vagus, for instance, by a dilated left auricle in mitral stenosis may be responsible. It may be secondary to gastric ulcer and carcinoma. In most cases there is no obvious cause. The condition is followed by the occurrence of great dilatation of the œsophagus. There is hypertrophy in varying degree of the muscular tissue of the dilated portion, but none at the cardial orifice of the stomach. There may be some chronic inflammation of the œsophageal mucosa.

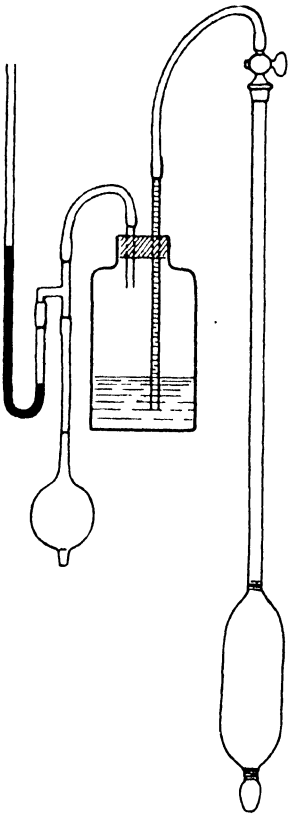


FIG. 48.—Plummer's bag connected with water bottle, mercury manometer, and rubber pump.

**Symptoms.**—The patients suffer sometimes for years from a sense of the food sticking in the throat after it has been swallowed, from actual pain in the epigastrium, from regurgitation of the food, or what is described as vomiting. The condition often comes on gradually, and at first may be spasmodic, recurring from time to time. An X-ray examination after a bismuth meal shows that the œsophagus is dilated into a fusiform body wider at the lower part (*see* Plates XVI. and XVII.). The dilatation may also be seen with the œsophagoscope. In fatal cases the internal circumference at its widest part has reached a measurement of from 10 to 16 cm. (4 to 6 inches).

**Treatment.**—Some cases have been fatal. In others the patients have got over the difficulty by liquid food, or by extremely careful mastication of solid food. It is often found that a column of fluid the length of the œsophagus forces its way through the sphincter by hydrostatic pressure, until the level has fallen by a certain amount, and this fact enables some food to be taken. Cases have been more promptly dealt with by the passage into the stomach before each meal of a rubber tube filled with mercury or by feeding the patient for four days by means

of an œsophageal tube introduced into the stomach and left *in situ* all the time. Another method is to pass a deflated Plummer's bag on the end of an œsophageal catheter right through the cardia. The following is a simple method of using it. The catheter is attached to a tube leading beneath water in a stout bottle (*see* Fig. 48). The bottle is attached to the rubber pump and mercury manometer of a blood pressure apparatus, by which the bag can be filled with water. The bag is now withdrawn until it is felt against the lower end of the sphincter. The water is allowed to run out of it, and the bag further withdrawn by about 2 inches (**3**), so that the bag is in the sphincter. Water is forced in until there is pain. This forcible dilatation may have to be repeated. The pressure from the water-main up to a pressure of 700 mm. Hg. has also been used; but this extreme method of forcible dilatation would not seem to be free from danger.

### DIVERTICULA

These are pouches in the walls of the œsophagus; they have been divided into (1) pressure diverticula and (2) traction diverticula.

1. *Pressure diverticula* arise from the impaction of foreign bodies, or from other local injury. As a consequence, apparently the muscular coat is weakened, and the mucous and submucous coats are bulged out between the muscular fibres, which do not share in the coverings of the diverticulum. When once this has taken place food accumulates in the sac, which gradually enlarges, so that it may attain a diameter of 3 or 4 inches. These diverticula are usually hemispherical in shape; they are most common at the back of the œsophagus in its upper part, and may project on both sides of the neck, sometimes on the left side only.

The **Symptoms** are dysphagia, regurgitation of food, and foul breath from the decomposition of food in the sac. So much food may accumulate as completely to obstruct the œsophagus.

**Treatment.**—The pouch has been removed by operation. Apart from this, the patient should be fed through an œsophageal tube.

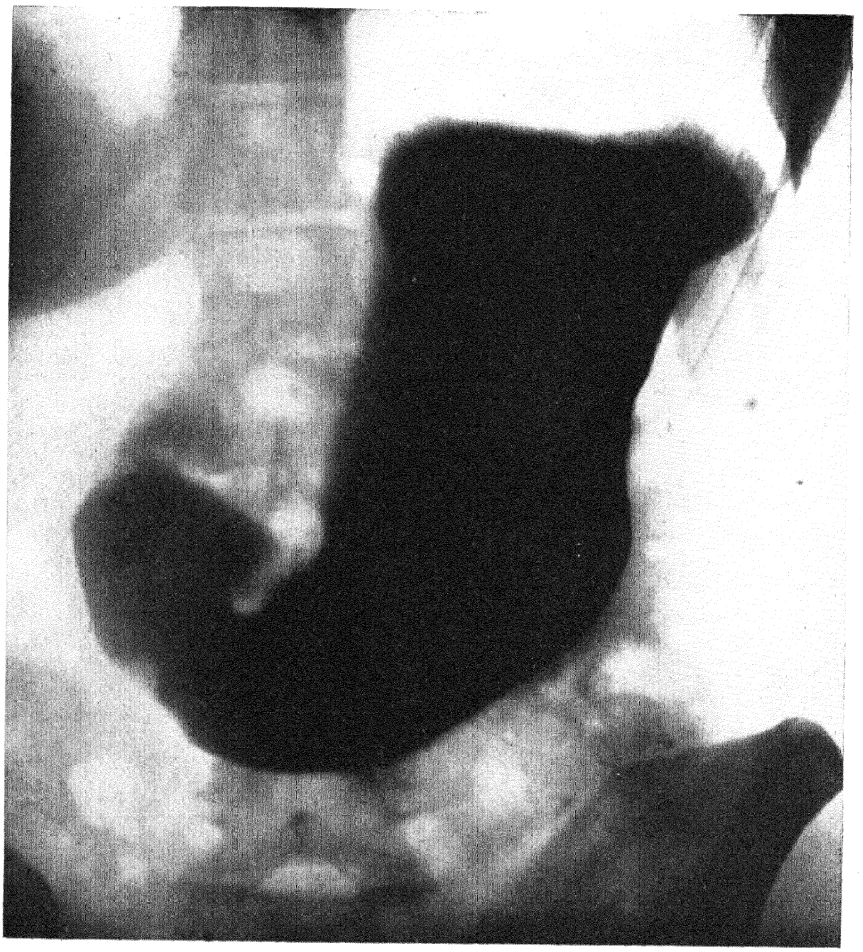
2. *Traction diverticula* are caused by adhesion of the œsophagus to surrounding parts, whereby the coats are pulled out in a funnel-shaped manner. They have occurred in children as a result of suppuration of the bronchial glands. They may give an opportunity for the impaction of foreign bodies, but otherwise have no clinical symptoms.

# DISEASES OF THE STOMACH AND DUODENUM

## EXAMINATION OF THE STOMACH

The position of the stomach varies with the position of the patient. In the erect position the cardiac end lies within the bony thorax, while the body and pylorus lie within the abdomen. In the horizontal position the stomach sinks further back beneath the ribs, and only the pyloric part of the stomach lies in the epigastrium. The stomach always contains some air, and this can be recognised by the full tympanitic note which is yielded on percussion of the lower part of the left thorax in front. This area is limited above by the præcordial dulness, and posteriorly by the splenic dulness and pulmonary resonance. It is impossible to separate the note obtained by percussion of the stomach from the note due to air in the intestines, so that this method is of no value in outlining the stomach.

## PLATE XVIII.



Radiogram showing Normal Stomach. The greater curvature reaches just below the iliac crest (which corresponds to the umbilicus), while the lesser curvature is above this level. The pylorus is patent and the duodenal cap is well filled with barium. The shadow above the cap is the beginning of the second part, and barium in traces can be seen elsewhere scattered through the coils of jejunum. (From a plate taken by Mr. W. Lindsay Locke.)

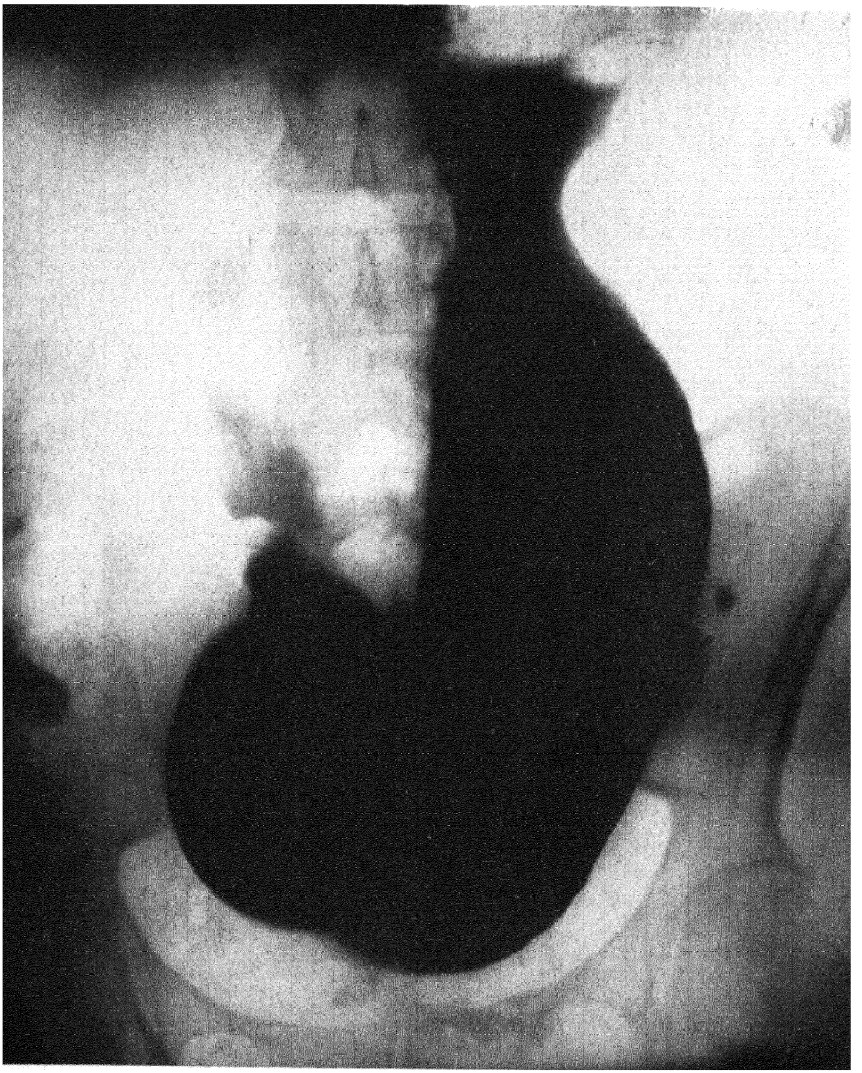
[*To face p.* 405

# PLATE XIX.

**Radiogram of a Hypertonic Stomach.** It is a small stomach, which is seen filled with barium and stretches transversely across the abdomen. It is placed high up in the abdomen far above the iliac crests, which are not shown in the picture. The first part of the duodenum is hidden behind the pyloric end of the stomach. A small portion of the barium has already passed through and is seen below. There is a large air bubble in the fundus of the stomach, seen just below the left diaphragm. The upper level of fluid in the stomach which is horizontal is seen beneath the air. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 405.

# PLATE XX.



**Radiogram of a Dropped Stomach filled with Barium.** The lower part of the greater curvature lies in the pelvis only just above the symphysis. It is a long way below the iliac crest. The upper part of the stomach is not seen; but a narrowing of the lumen about the middle of the stomach is seen. This is due to sagging in of the walls of the elongated stomach. There is no spasm or cicatricial contraction as in hour-glass stomach (see Plate XXV., p. 407). As the stomach is dropped its volume is at the same time larger than normal, so that it forms one type of dilated stomach. The other type, associated with pyloric stenosis, has an entirely different appearance (see Plate XXIV., p. 406). (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 405.

The stomach is remarkable for the great alterations in size and position it may undergo, so as to accommodate variable quantities of food. Like other organs it possesses a system of longitudinal and circular muscle fibres, but it has, in addition, an inner system of oblique fibres which pass down from the circular fibres of the œsophagus along the lesser curvature, spreading out like a fan on the anterior and posterior surfaces. It seems likely that this system is concerned with the downward movement of the greater curvature, which takes place as the stomach fills up.

### EXAMINATION OF STOMACH BY X-RAYS

This gives valuable information about the shape, size, and motility of the stomach. The patient swallows a meal of porridge or bread and milk containing 2 ounces of a solid salt of bismuth, preferably the oxychloride, or 4 ounces of barium sulphate. The rays are then used, and the position and size of the stomach are indicated by the shadow cast on the screen by the contained metallic salt. The X-rays show that the stomach consists of a vertical part and a horizontal part, separated by the *incisura angularis* on the lesser curvature (*see* Plate XVIII.). The vertical part is divided into two by an imaginary horizontal line at the level of the cardiac orifice. The part above, which usually contains air, is called the *fundus*, the part below the *body*. The horizontal part consists of the *pyloric vestibule* and the *pyloric canal*. The first part of the duodenum during the action of the stomach receives and retains for a time the chyme, so that under the X-rays it shows a dark shadow like the stomach, which often has a triangular shape with its base towards the pylorus and has been called the *duodenal cap*. It is separated from the stomach by the transparent line of the pylorus, in the middle of which the pyloric canal may be seen from time to time, wider or narrower according to the amount of chyme going through. Peristaltic waves of muscular contraction from the body to the pylorus are recognised by the X-rays with accompanying changes in the shapes of the body and pyloric vestibule. The average position of the normal stomach in the vertical position is such that the greater curvature lies just below the iliac crests (or umbilicus), while the lesser curvature is above it. However, there are wide variations in the position of the stomach quite compatible with health. The stomach may reach lower down, and this is sometimes known as the *dropped* stomach which is usually also *hypotonic* (*see* Plate XX. and also Fig. 52 on p. 417), or may be entirely above the iliac crests, the *hypertonic* stomach (*see* Plate XIX.). In the horizontal position, with the patient lying on his back, the stomach falls back beneath the diaphragm, and so lies higher than in the vertical position; it is often divided into two by the vertebral column. It has been observed that a stomach may drop several inches, when viewed in the vertical position, as the result of emotion, or just before a fainting attack, or after a nauseating smell, such as asafœtida. Conversely the stomach tends to be hypertonic when the appetite of the subject is aroused (**4**). The mean position of the stomach depends on the physical build of the individual. It is high when the body is broad and short, *i.e.* when the circumference of the chest is 3 cms. greater than the body length (*see* p. 560) and, conversely, low when the circumference of the chest is 7 cms. less than the stem length. The high stomach is usually associated with a high acidity of the gastric juice (**5**).

No X-ray examination of the stomach is complete without determining the rate of emptying. The patient is examined two hours, four hours, and eight hours after taking the meal to see if there is any shadow still remaining in the stomach. Normally the stomach is empty in four hours. The small hypertonic stomach is often empty in two hours. Pyloric stenosis is suggested if most of the contents are still present after eight hours. It will be noticed that these times are rather greater than are obtained with the fractional test-meal method.

## EXAMINATION OF THE CONTENTS OF THE STOMACH

By the examination of the vomit, and of liquids artificially withdrawn from the stomach during the process of digestion, we may try to ascertain the share which a deficiency of the acids, of the pepsin, or of the motor powers of the stomach may have in different forms of disease, especially in the chronic disorders of digestion.

**Vomit.**—If the patient vomits, the quantity, odour, colour, and consistence of the liquid should be noted. The smell is usually acid, but it may be modified by substances recently taken, such as volatile oils or alcohol. The liquid may be colourless, or different shades of brown, or stained yellow or green by bile pigment, or pink or red by blood. Often blood is altered by contact with the gastric juice, and a dark brown, opaque fluid is the result, resembling *coffee grounds*. In consistence vomit may be watery, or more or less viscid from

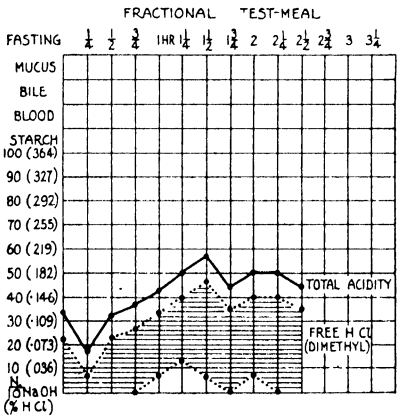



FIG. 49.—The Variations of Free HCl in 80 per cent. of Normal Students (Bennett and Ryle). The other 20 per cent. gave results outside these limits. The total acidity curve is drawn 10 c.c. above upper free HCl curve.

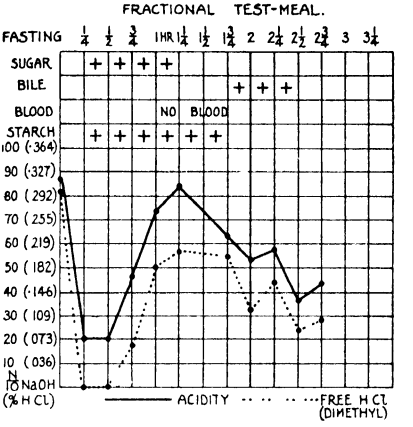



FIG. 50.—Case of Duodenal Ulcer, showing highly acid resting juice, quickly rising curve after the meal, and rapid emptying of the stomach. The fall in acidity at the end is due to regurgitation from the duodenum.

mucus, or frothy. The presence of half-digested or undigested food should be noted.

Microscopically animal and vegetable tissues may be detected, such as muscle fibres, cellulose, starch granules, oil drops, red blood corpuscles, leucocytes, and numerous micro-organisms, especially *torulæ*, *sarcinæ*, and sometimes Oppler-Boas bacilli. For chemical examination the vomited fluid must be strained through fine muslin, and the filtrate may be submitted to the tests presently to be mentioned.

**Test Meal.**—The functions of the stomach can be more accurately determined by the use of a test-meal than by examining vomit. Two methods are used: (1) *Ewald's Test Breakfast.*—The stomach is first washed out, or the meal, consisting of 2 or 2½ ounces of bread or toast and 20 ounces of weak tea, is given in the morning on an empty stomach. The stomach contents are removed after an hour's interval and filtered. Ten cubic centimetres of the filtrate are evaporated to dryness and incinerated. All the HCl, free and combined with protein, is thus driven away, leaving behind that combined with the various metallic bases. This "mineral chloride" is estimated by titrating with silver nitrate. Excess of sodium carbonate is added to another 10 c.c. and the same process repeated. The

sodium carbonate fixes the free HCl and that combined with protein, so that the titration gives the total amount of chloride present. By subtracting the first value from the second the " active HCl " is obtained, *i.e.* the HCl which is both free and combined with protein. The normal value for this is 0·15 per cent., and for the HCl combined as mineral chloride 0·10 per cent. In cases of hyperchlorhydria values up to 0·27 per cent. for the active HCl are obtained ; while in carcinoma ventriculi the active HCl is much diminished and may be absent, and the mineral chloride is considerably increased—to 0·15 per cent. or 0·20 per cent.

(2) *The Fractional Test Meal.*—A small bore rubber tube with holes at the end is passed into the stomach, and the contents removed by means of a syringe. A meal is then taken consisting of two tablespoonfuls of breakfast oatmeal boiled with 1 quart of water until the volume reaches 1 pint and strained through muslin. Specimens of the stomach contents of about 10 c.c. each are withdrawn every quarter of an hour until the stomach is empty. The presence of mucus, bile, and blood is noted. The samples are titrated with decinormal NaOH solution, using dimethyl as an indicator. The titration is then continued until

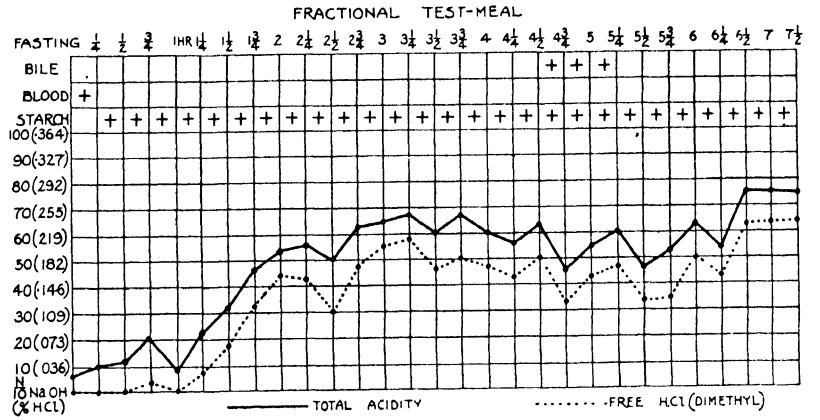



FIG. 51.—Case of Duodenal Ulcer, with prolonged Stasis of the Meal in the Stomach. The HCl showed a gradual rise. A little regurgitation from the duodenum took place after 3¾ hours.

the sample is alkaline to phenolphthalein. The first titration gives the " free HCl " ; but the value thus obtained is sometimes a little too high, as some of the HCl combined with protein is also neutralised. The amount of alkali required to change the phenolphthalein gives the " total acidity." The difference between the total acidity and free HCl is usually pretty constant. It may be greatly increased if other acids, such as lactic acid, are present. More accurate results for the free HCl are obtained if thymol blue is used as the indicator (Cole).

The acidity of the gastric juice depends not only on the secretion of HCl, but on the amount of saliva, on the regurgitation of pancreatic and intestinal juice backwards from the duodenum, and on the rate of emptying of the stomach. The free HCl in 80 per cent. of normal students was found to lie within the limits shown in Fig. 49 (Bennett and Ryle). The total acidity is usually about 10 c.c. higher than the free HCl. Its approximate upper limit for normal individuals is shown in Fig. 49. In 4 per cent. of cases there was complete absence of free HCl (achlorhydria), and in 11 per cent. the HCl was increased. The fall in the curve at one and half hours is not entirely due to a falling off in the secretion of HCl, but to neutralisation from regurgitation of alkali from the duodenum (**6**), and possibly from the pyloric part of the stomach (**7**). This fact illustrates the defects in fractional analysis as usually carried out, since it does not differentiate between

lessened production of acid and neutralisation. Regurgitation increases the "mineral chloride" at the expense of the HCl, and to get a true picture it is necessary to determine the total chloride as well as the free HCl. A simple method of doing this has been described (**7**). "Achylia" and marked hypochlorhydria in normal persons has been found to be due to increased neutralisation (**7**).

Psychical influences may alter the curve considerably. Thus a feeling of nausea which was suggested to a student under hypnosis caused delay in the emptying of the stomach, while a feeling of hunger caused the stomach to empty rapidly, which was doubtless due to increased motor activity. This agrees with the X-ray observations just described. Severe muscular exercise and a hot bath have been found to diminish the secretion of HCl, and to delay the emptying of the stomach. Mild exercise, with pleasurable companionship, produces the reverse effect (**10**). With ulcer in the body of the stomach there is no characteristic curve. However, with duodenal and pyloric ulcer (Fig. 50) there is often a highly acid resting juice in the stomach, and after the fall in free HCl due to dilution with the meal the HCl content rapidly rises until all the food has left the stomach, as determined by the disappearance of starch. After this point the gastric juice still continues to be secreted (hypersecretion). The stomach may empty itself very rapidly ("duodenal hurry"). The high concentration of acid in these cases is explained by the hypersecretion of gastric juice, so that the proportion of gastric juice in the stomach contents rapidly increases, and by the absence of regurgitation backwards from the duodenum which normally occurs (Boldyreff). In cases of pyloric stenosis (Fig. 51) the food remains for a long time in the stomach, as shown by the persistence of starch. The concentration of HCl gradually rises as more and more gastric juice is secreted and mixed with the meal, and there is also not much regurgitation from the duodenum.

**Tests for Blood.**—If blood is not obvious in the vomit or stomach contents, either as bright red blood or as coffee grounds, it may yet be present in sufficient quantity to be detected by chemical tests; but it is even more important to examine for *occult blood* in the stools, because a trace of blood in the stomach contents may be due to trauma.

*Guaiacum Test.*—The fæces are stirred with equal parts of glacial acetic acid and water, and shaken with ether; to the ether extract in a test tube a drop or two of tincture of guaiacum is added, and then 2 c.c. of ozonic alcohol (hydrogen peroxide in alcohol), when a bright blue colour appears. That this may be regarded as a proof of occult hæmorrhage, the patient must have abstained from chlorophyll and blood-containing foods, *e.g.* meat and green vegetables, for at least forty-eight hours beforehand.

It is also desirable as a confirmatory test to examine the ether extract with a spectroscope for the presence of acid hæmatin. Subsequently the ether extract is shaken up with 30 per cent. HCl, and the watery extract is examined spectroscopically for acid hæmatoporphyrin. The presence of this substance means that the blood has been altered in the body, and so has come from high up in the alimentary canal, *i.e.* stomach, small intestine, or upper part of the colon (Ryffel).

## DYSPEPSIA AND FUNCTIONAL DISORDERS

Dyspepsia is the term that indicates that there is disorder of those functions of the upper alimentary tract which are normally concerned with the preparation of the swallowed food and its transmission into the small intestine for absorption. These functional disorders give rise to a variety of symptoms of which pain or discomfort before or after food are the most striking, and since the symptoms and the underlying functional disorders are always associated, *e.g.* the gastric crises of tabes are associated with abnormal movement of the stomach, the term dyspepsia may be applied simply to describe the symptoms.

The stomach is undoubtedly the organ most commonly affected, but, as will be pointed out later, the œsophagus and duodenum may also play a part in the symptomatology. The term "indigestion" is often used in a wider sense to indicate either dyspepsia, or deficient absorption in the small intestine, or a combination of the two, whence the term "undigested stools." The other functional disorders of the stomach dealt with in this section, though sometimes associated with dyspepsia, are often present quite independently.

## ACUTE DYSPEPSIA

Acute dyspepsia may be produced from the ingestion of food insufficiently masticated, or of specially irritating quality, or taken in too great quantity. Any one in perfect health may be tempted to take a larger quantity of food than his stomach can bear; or with an ordinary quantity of food some ingredient, such as ice or coffee, or alcoholic drink in excess, may be taken which retards the process of digestion, and the whole quantity ingested remains for some hours in the stomach. Or the unexpected failure to digest may be due to preceding general exhaustion, in which the stomach shares; for instance, after excessive exercise in walking or climbing during several hours without refreshment the stomach may fail entirely to digest even a moderate meal.

**Symptoms.**—Either at once or within a few hours of the meal there is a sense of distension and discomfort, or actual pain, in the gastric region; if the offending meal has been a late dinner, the patient may go to sleep with a little trouble, but wakes after a few hours with gastric distress, a dry tongue, and perhaps headache, and may lie wakeful for some hours. Sometimes a quick fluttering is felt in the præcordial region from extra systoles of the heart, or isolated extra systoles are felt at longer intervals. In the morning there is little inclination for food, the tongue is dry and furred, and the skin clammy; but in the course of a few hours the symptoms subside. In other cases the illness is more quickly terminated by the occurrence of vomiting, and the stomach is generally emptied of the whole of its contents, which are only partially, if at all, digested, and are mixed with gastric mucus. The pain is often at once relieved. At other times the vomiting may be repeated, and bile, which has been regurgitated from the duodenum, may be discharged with the later efforts. Sometimes in the course of the next twelve hours the bowels are actively moved, from the passage into them of undigested or irritating materials.

**Treatment.**—Where the pain is severe, and the cause is obvious, immediate relief may be obtained from an emetic such as sal volatile or ipecacuanha; if it fails to act, the stomach contents may be removed by a tube. In milder cases it is sufficient to quench thirst with a very little ice, and to abstain from introducing anything further into the stomach until the distressing symptoms have subsided.

## CHRONIC DYSPEPSIA

Sufficient has already been said to indicate that chronic dyspepsia is not a disease, but rather a series of symptoms resulting from disturbance of function in the upper alimentary tract.

**Pathology.**—The following account is based largely on recently published observations (**2, 11**) and on others not yet published. In considering the problem two questions must be kept distinct: (1) What is the organ the functions of which are disturbed in dyspepsia and how does the disturbance produce symptoms? (2) What is the lesion that causes the disturbance?

(1) It is at the present time a relatively easy matter to determine in what organ the symptoms are being produced. Pain is the symptom which has been most fully investigated. When it is felt high up in the epigastrium by the xiphisternum, or behind the sternum, it is caused by the œsophagus (*see* Plate XV., p. 402). Substernal œsophageal pain may be distinguished from cardiac pain by

getting the patient to make a swallowing movement. The resulting peristaltic wave that travels down the œsophagus at about the rate of 1 inch a second momentarily relieves the pain, but may intensify it if the pain is very slight. Swallowing is unlikely to make a difference to cardiac pain. Pain in the stomach is felt in the epigastrium, probably rather lower than the œsophageal pain, and sometimes along the left costal margin, and very commonly round the umbilicus. Pain in the duodenum is felt at about the same level but rather to the right of the middle line. Pain in the jejunum is probably felt below the umbilicus. Sensations of fulness and sinking may both be due to the stomach, while the sensation of nausea, which is felt at the back of the throat and deep in the upper part of the chest, is probably due to reflex disturbances in the upper part of the œsophagus. Visceral pain may be produced experimentally by blowing up an airbag placed inside a viscus, so as to stretch its walls. The presence of such a foreign body in the œsophagus, the organ which has been investigated most completely, causes a series of peristaltic waves to pass down it; but if the foreign body is large, no waves succeed in getting past, and the pain is continuous. If the bag contains rather less air, peristaltic waves succeed in getting past, and each time the wave passes over the bag and compresses it, the pain diminishes or disappears, and increases again or reappears as soon as the wave has passed and the wall becomes stretched again. In this case the pain is discontinuous or "griping." Hollow viscera have a great power of enlarging, *i.e.* altering their "posture," using Sherrington's term, and a foreign body that causes symptoms in the first place, ceases to do so when the organ has become big enough by the lengthening or rearrangement of its fibres, so that it can hold it without being stretched. But this process can only take place rather gradually. Hence the postural lengthening of a fibre must be distinguished from its stretching, which causes pain.

Observations carried out by means of a small water bag on patients, while they were experiencing pain, always showed the presence of a series of contractions. These have been observed in the œsophagus, stomach, duodenum, or jejunum. Further, the mean pressure inside the viscus was increased, though the extent of the increase varied in different cases. The contractions tended to diminish the pain, which was in one case only experienced during the subsequent muscular relaxation, during which stretching of the fibre probably occurred from the increased *mean* pressure in the viscus. Thus, there was very great similarity between the artificially produced pain and that which occurred naturally. Further, a very close connection was observed between the different viscera. Distending the stomach with air produced peristaltic waves down the œsophagus (the gastro-œsophageal anti-regurgitation reflex). Contractions in the stomach in a patient with hunger pains were followed about one second later by similar waves in the duodenum.

Dyspeptic pain is due to stretching of the muscle fibres, and this agrees with other observations that dyspepsia is often the result of unsuccessful attempts at emptying the stomach (**12**). It is quite possible that in some conditions the muscle may be slack or toneless, so that stretching occurs under a pressure that is relatively low; but where the pressure is high there must be simultaneous closure of the sphincters, or otherwise the contents would be driven out, and it is quite likely that the dyspeptic syndrome depends primarily on one of these two factors: tonelessness, *i.e.* undue extensibility, of the muscular wall; or closure, possibly achalasia (*see* p. 403), of the sphincters. Localised contractions (spasms) may occur at other places than at the sphincters—in the œsophagus, or duodenum, or on the greater curvature of the stomach. Pain in the stomach is usually of two kinds. It may come on shortly after a meal, or after some hours, when the viscus is empty of food, the so-called *Hunger Pain*. It has been pointed out that there are two important factors associated with these types of pain, both of which would tend to cause stretching of the wall. These are (1) aerophagy, which may be quite involuntary, and (2) prolonged secretion from the gastric

mucous membrane. In fact, these factors have been regarded as compensatory, the aerophagy leading to a rise of pressure and so to the emptying of the organ, and the prolonged secretion acting as a diluent of any noxious substance, in the same way that irritation in the mouth leads to a copious salivary secretion (**12**).

The nausea, sense of fulness or pain of dyspepsia, may lead reflexly to vomiting, or retching, which is the quickest and safest way for the upper part of the alimentary tract to rid itself of its contents. It is due to strong contractions of various skeletal muscles, by which the contents of the stomach are forced out along the œsophagus and through the mouth. In adults vomiting is usually an extremely painful process; but the pain is possibly due to the stomach contents being pressed out through a closed cardia. Vomiting itself is painless, as the writer himself observed when once attempting to push a bag tied onto the end of a stiff catheter down his own œsophagus. It was simply pushed out again by abdominal contractions. The vomiting of babies, and hysterical vomiting, are painless, and the same is often the case when vomiting is due to intracranial disease. Stimulation of the lower part of the lesser curvature leads to vomiting movements. The abdominal muscles contract, while the stomach is motionless (**4**).

(2) *Lesions responsible for Chronic Dyspepsia;*

(*a*) The stomach itself may be the primary cause, owing to deficiency in the tone of the muscle, which is often accompanied by dropping of the organ (*see* p. 416), or to gross organic disease, such as gastric ulcer and carcinoma, and pyloric stenosis, which are considered later, or to chronic gastritis.

*Chronic Gastritis.*—In very chronic cases, the wall of the stomach is generally thickened, and presents various degrees of vascularity, not always very marked, and there is excess of mucus. Numerous dark or slate-coloured patches of pigmentation give evidence of former congestion or hæmorrhage; and occasionally small ulcers, *hæmorrhagic erosions*, are scattered over the surface. Sometimes the mucous membrane is atrophied entirely, at others there is a fibrous overgrowth of the interglandular, submucous, and intermuscular connective tissue, while the glands disappear or become cystic, and the muscular fibres perhaps waste. The process of thickening sometimes produces, especially in the pyloric region, a rough and wrinkled surface, commonly described as *mammillated.*

Motor defects of the stomach and chronic gastritis may be the result of an acute attack of dyspepsia, but they may arise from the continued ingestion of irritating or indigestible food; and they are constant result of undue indulgence in alcoholic liquors. They may arise from the venous congestion which results from diseases of the liver and heart. Other contributory causes are defective mastication, bolting the food, and repeatedly overloading the stomach. The dyspepsia of phthisis is often due to the over large meals that these patients are encouraged to take. The dyspepsia of gout and obesity may be due to a similar cause.

(*b*) Dyspepsia may be caused reflexly by organic lesions situated at a distance, by duodenal ulcer, chronic appendicitis, gall stones, and other lesions of the gall bladder, lesions of the kidney, particularly movable kidney and calculus. It may be secondary to constipation. In the gastric crises of tabes the primary lesion is in the posterior nerve roots. Angina pectoris may cause a secondary indigestion—*heart burn* in particular. That the origin of the dyspepsia in these cases is reflex, is shown by the following observations. Manipulation over the appendix area in chronic appendicitis has produced a localised spasm of the greater curvature of the stomach. The latter has also been produced by stimulating electrically the peritoneal surface of a duodenal ulcer at operation (**4**). In a case of jaundice from gallstones, the epigastric pain was found to be associated with the gastric movements. Reflex subternal pain has been noted when manipulating a chronically inflamed appendix (**13**).

(*c*) Dyspepsia may be due to more general agencies: pregnancy, sensitiveness

to foreign proteins (*see* p. 224), mental anxiety, overwork, and other debilitating influences, such as prolonged illness, fever, anæmia, Bright's disease, which may act by causing hypotonus of the stomach wall or by interfering with the secretion. Hypoglycæmia may cause a feeling of hunger with pain. The fact that some people may suffer from indigestion all their lives without any obvious cause has led to the suggestion in times past that there is a dyspepsia "sine materia." It is however probable that careful examination in such cases will always disclose, apart from well-marked organic disease, some lesion whether this takes the form of weakness of the stomach wall which has been called atonic dyspepsia, or hæmorrhagic erosions in the mucous membrane (**12**). The present writer has been much impressed with the frequency of slight hæmorrhage in chronic dyspepsia. The majority of his dyspeptic patients show no obvious abnormality in the X-ray appearances of the stomach and duodenum; but the stools contains occult blood and give a hæmatoporphyrin spectrum. The term *acid dyspepsia* only refers to one symptom of variable occurrence, viz. acid regurgitations, which may or may not be accompanied by hyperchlorhydria. The term *nervous dyspepsia* may be used to describe the dyspepsia of organic nervous disease or disturbed mental states, such as the uneasiness and sinking felt in the epigastrium associated with anorexia and nausea, which are characteristic of certain psychasthenic conditions. The patient refuses altogether to take food and becomes extremely emaciated. At the same time the patient may show plenty of bodily activity. The condition has been called *Anorexia Nervosa* by Gull. There is no evidence that dyspepsia is due to a hyperexcitable solar plexus.

Dyspeptic symptoms may persist for some time after the cause has been removed. This is illustrated by an experiment, in which the œsophagus was distended by a bag so as to produce pain. The skin over the sternum was tender and there was pain in the back; both these symptoms continued for some hours after the experiment was ended, and a severe attack of ordinary dyspepsia occurred the next day. The œsophageal muscle fibres were probably slightly injured and produced reflex gastric disturbances.

**Symptoms.**—These vary much in different cases.

*Pain.*—Dyspepsia is shown frequently by pain in the epigastric region, which comes on after taking food, and lasts a certain time, gradually subsiding. It may be localised at the bottom of the sternum, or pass upwards into the chest, especially behind the sternum. This pain is called *cardialgia* or *heartburn*. Often it is felt between the shoulders, going "through to the back." Gastric pains may be referred to the head, producing a brow ache. Sometimes, as digestion slowly proceeds, the pain extends to the umbilicus, and to the right and also to the lower abdomen. In other cases pain begins when the stomach is empty, and is relieved by ingestion of food. Instead of pain there may be only a sense of discomfort, tightness, or fulness, or a sinking feeling. Severe pain may be accompanied or followed by superficial tenderness of the overlying skin, or deep tenderness on pressing the abdomen.

*Flatulence.*—This is a common occurrence in all types of indigestion. There is distension of the stomach with corresponding discomfort in the upper abdomen and pain relieved by eructation. Flatulence is usually regarded as due entirely to aerophagy (*see* p. 415), but it is sometimes partly due to $CO_2$, which is probably given off when the alkaline duodenal contents regurgitate and meet the acid gastric contents in the stomach. Thus the gas in the stomach has sometimes been found to contain 10 per cent. $CO_2$.

*Nausea* is a common symptom of dyspepsia, and *vomiting* less frequent, except in alcoholic dyspepsia. The vomited matter is either the ingested food or merely mucus. With repeated emesis bile may be rejected, and a few streaks of blood. *Pyrosis*, or *water brash*, is a name given to a condition in which a quantity of liquid is brought up into the mouth. The liquid is sometimes neutral or alkaline in reaction, and is then commonly believed to consist chiefly of saliva; but it is

often acid. There is an accompanying burning sensation felt in the throat and behind the sternum. This is not due to burning by the acid, because acid of this strength does not cause any sensation in the œsophagus. The appetite is variable, there may be thirst, especially with vomiting.

*General Symptoms.*—The tongue is variable; it is sometimes furred. The fur may be thin and white, or thick and yellow or brown. The furring so often met with may be due to a slop diet, with absence of mastication, or to a deficient flow of saliva, so that the surface scales are not rubbed off. It is accompanied by offensive breath. Constipation is frequent, but may be interrupted by occasional diarrhœa. The skin eruptions, erythema, rosacea, urticaria, and acne vulgaris, are often associated with indigestion (*see* pp. 936, 967, 974, 975). The effect upon the body generally, or more correctly upon the nervous system, is seen in malaise, indisposition for exertion, giddiness, subjective sensations of sight, drowsiness, irritability, and mental depression; while slight anæmia or sallowness, some loss of nutrition, and in chronic cases a settled expression of discomfort or anxiety upon the face, are not uncommon. But in other instances there is no general indication whatever of the gastric fault.

**Diagnosis.**—The symptoms of indigestion, and their association with food or hunger, are so characteristic that there is no difficulty in diagnosis. However, diagnosis of the cause of the condition is quite another matter, and in long standing cases, in addition to a thorough general clinical examination, X-ray examination of the alimentary tract, examination of the stools for occult blood, a test-meal and analysis of the duodenal contents, may all yield valuable information. Even when gross disease of the stomach has been excluded, the presence of occult blood may indicate a hæmorrhagic lesion in the mucous membrane, which may be due to chronic gastritis.

**Treatment.**—It is necessary to treat any disease or faulty habits of life which may be responsible for the dyspepsia. In particular oral sepsis must be attended to. The actual treatment of the dyspepsia may be classified as follows:

(1) *Diet.*—All foods containing indigestible residues in the form of pips and skins should be avoided, and solid food must be thoroughly masticated, or minced in the absence of teeth, before it is swallowed. Highly flavoured or spiced sauces, fried food, made-up dishes, condiments, acid fruits, and uncooked substances such as radishes, salad, etc., are not allowed. Food must be simple; but it is impossible to give a hard and fast list of what is allowable. So much depends on the patient's own experience. The following principles should be borne in mind. Flesh foods stimulate the stomach to produce a highly acid juice with much pepsin, and are best digested in such a medium. Starchy foods are digested by the enzyme of the saliva, which requires a slightly alkaline medium, while fats diminish the secretion of HCl and delay the emptying of the stomach. Under ordinary circumstances, and especially if the gastric juice is rather deficient, meat should be given at the beginning of a meal so as to stimulate its production, and the meal may be continued with starchy foods and fats in the form of vegetables, cereals, or fruit, with butter or cream. On the other hand, if the secretion of gastric juice is very abundant, the best results may be obtained by giving fats to begin with, in the form of olive oil, ½ ounce, or sardines in oil, with butter, to be followed by meat and cereals, etc., later. It is probably better practice to lessen the secretion by means of fat than to neutralise the HCl in the stomach after it is formed by drugs. Fat should not be given with flesh foods, and the presence of much fat in pork may be the reason that this is usually tolerated badly. Fish, of which many kinds contain no fat, is usually tolerated best of all.

In more severe cases meals of ordinary size cannot be taken at all, and it is certainly the case that the stomach works best when it contains only a small quantity of food, while pain or discomfort are noticed both when the stomach is full up and when it is quite empty. Hence it is advisable to give food in small

quantities at a time and at frequent intervals. Besides a light breakfast, lunch and dinner, something should be taken on waking in the morning, in the middle of the morning, at 4 p.m., and before going to bed at night.

The liquid part of the diet may be regarded from two points of view. There is the volume of fluid that mixed with the solid part of the meal produces the optimum concentration for digestion. This should be taken during, or immediately after the meal, and may amount to a pint in the twenty-four hours. There is the additional fluid, amounting to 1 or 2 pints, that is required in the general metabolism of the body. This should be taken a quarter or half an hour before meals. Solids and liquids should always be taken warm. The most suitable fluids are water, *weak* tea or cocoa, and of course milk, which is also an important food. A digestive fluid containing diastase may be readily made as follows (**12**): A tablespoonful of malted barley is ground up and mixed with a teacupful of cold water in an earthenware pot, which is placed for ten minutes in a saucepan or water-bath of boiling water. It is strained and sweetened before being taken. Alcohol must not be taken.

Some dyspeptic patients are greatly wasted because they refuse to take food owing to the fear of unpleasant consequences. For such patients rest in bed is essential, and they should be compelled to take food in excess of their basal requirement (*see* p. 558) until they regain their weight. It is a good plan to allow a varied breakfast, lunch and dinner, the amount of food being equivalent to the basal requirement, and to prescribe, in addition, 3 pints of milk to be taken on waking, 11 a.m., 4 p.m., and last thing at night.

*Drugs.*—These should only be prescribed when symptoms are complained of in spite of careful regulation of the diet. One of the most valuable is $CO_2$, which may be liberated in the stomach and acts by stimulating gastric contractions and so helps to empty the stomach, or by causing eructations which relieves the intra-gastric pressure. Two mixtures are made: (1) ℞ Sod. Bicarb. gr. xxx.; Infus. Gent. Co. ad ℥i. (2) ℞ Citric acid, gr. xxx.; aq. chloroformi, ad ℥i. To get a maximal effect with eructations the first mixture is followed immediately by the second. A milder effect is obtained by taking repeatedly a teaspoonful of No. 1, followed by a teaspoonful of No. 2, until the pain or discomfort disappears. Instead of the sod. bicarb., a teaspoonful of equal parts of prepared chalk and mag. carb. may be mixed up in a little water and used similarly. The mag. carb. will act beneficially in relieving the constipation so often present. The beneficial effect of sodium bicarbonate in relieving pain when given by itself is almost certainly due to this liberation of $CO_2$ by the acid in the stomach, and not to its alkaline nature. The advantage of giving citric acid is that it makes the action more certain, and it is probably bad practice in most cases to neutralise the HCl of the gastric juice suddenly during digestion. If it is desired to prescribe an alkali independently of the $CO_2$ effect, magnesium oxide may be used. Where it is suspected that there are lesions in the mucous membrane, bismuth or kaolin may be prescribed with the idea of covering the lesions with a plaster and so protecting them; but they must be given in fairly large doses, *i.e.* 2 to 4 drachms, suspended in water with mucilage. In some cases of indigestion acid hydrochlor. dil. in doses up to a drachm, well diluted with water, taken during and after the meal, is of great benefit, especially when the HCl of the gastric juice is low, and there is much flatulence accompanied by flushing of the face, as in cases of rosacea. Other drugs which act by stimulating gastric contractions and so help evacuation, have been taken from time immemorial, such as sal volatile, tr. nucis vomicæ, strychnine, ipecacuanha, the vegetable bitters such as calumba root, and the volatile oils, such as oil of peppermint. These drugs, often called carminatives, are of subsidiary importance.

*Physical Methods.*—Heat externally applied to the upper part of the abdomen is a powerful help to digestion and probably acts, like counter-irritation, by causing reflexly effective visceral movements. Heat may be applied after

meals by means of Gamgee tissue, tied round the abdomen, a small portable hot-water bottle, electric or ordinary poultices, or plasters including antiphlogistin or thermogene wool. Other methods applicable to cases of gastroptosis with deficient motor functions on the part of the stomach are described on p. 417. In cases of very severe acute intractable gastric pain, a tube should be passed into the stomach to relieve the pressure. This is preferable to giving morphia, which may, however, be sometimes necessary.

## AEROPHAGY

The condition of *aerophagy* or excessive air-swallowing gives rise to what is called flatulent dyspepsia. In the past the accumulation of gas in the stomach was considered to be due to fermentation; but normally this does not occur to any appreciable extent, because the stomach contents are too acid and stay there too short a time. Carcinoma of the stomach, producing pyloric stenosis, is the condition chiefly predisposing to fermentation, because here there are both low acidity and stasis of food. Normally some air is swallowed with food, and is observed in the stomach by X-rays as a clear area just below the left diaphragm giving rise to the tympanitic note obtained on percussing over the upper part of the stomach. Its presence in moderate quantity is probably beneficial, since it increases the pressure inside the stomach, and so may stimulate the motor functions of the stomach and facilitate its emptying. It is only when excessive quantities of air are swallowed, and particularly if the process continues between meals, that the condition is known as aerophagy.

**Ætiology.**—There are three groups of cases:—

1. *Dyspeptic.*—This is to be regarded (*see* p. 410) as an exaggeration of the normal mechanism by which air enters the stomach and is a reaction of defence against the dyspeptic state. Air is "pumped" down the œsophagus by repeated peristaltic contractions; the pressure in the stomach is raised, and so the evacuation of the stomach is promoted (**12**).

2. *Bad Habits.*—This is closely related to the dyspeptic variety and may result from it. The patient feels some discomfort, such as a feeling of tightness or fulness in the epigastrium, which he feels can be relieved by a successful eructation. This feeling may arise spontaneously, or may result from an attack of acute indigestion or an acute gastric ulcer, or some other illness. It persists after the original cause has disappeared. It is not due to excess of gas in the stomach, as the latter is often nearly empty. The patient tries to relieve himself by eructation, but the only effect is to force air into the stomach, so that the discomfort increases; this is repeated once or twice until a considerable amount of air has collected. At the next attempt all the gas is eructated with an immediate sense of complete relief. After a short time the sense of discomfort is felt again, and the whole cycle may be repeated indefinitely. Other patients may suffer from noisy eructations repeated every few seconds, so that they become a nuisance to themselves and their friends. This is really a form of "tic" (*see* p. 880), and is often hysterical. In such cases the air does not enter the stomach, but is sucked into the œsophagus and immediately expelled with violence. In yet other cases the air passes into the stomach by suction.

3. *Excess of Saliva.*—The cause of this is obscure; but when it occurs the patients are constantly swallowing it all day long, and air is swallowed at the same time. According to Leven (**14**), this condition, which is very common and troublesome, is indicated by (1) a wet, red, bright tongue, due to the cleansing action of the saliva; (2) irritation of the larynx, due to constant swallowing, which is shown by the patient objecting to wear anything tight round the neck, such as a collar; (3) inability to sleep on the left side; (4) dribbling on the pillow at night; (5) complaints of flatulence; (6) the patient may be observed

to bend his chin down on his chest at frequent intervals. This is preparatory to the act of swallowing.

**Symptoms.**—These include a feeling of fulness with discomfort and sometimes severe pain with distension in the epigastrium, eructations, hiccough, loss of appetite, vomiting. Certain distant reflex disturbances are also attributable to ærophagy, viz. congestions of the face, headache, hot flushes, palpitations, tachycardia, extra-systoles and substernal pains produced in the œsophagus. The experimental distention of the stomach has been found to produce increased œsophageal peristalsis. Much of the air swallowed passes on through the pylorus, and may be observed temporarily by X-rays in the first part of the duodenum, and passing through the small intestine, collects in the colon in large bubbles. This increases the resistance to the passage of the contents, and so causes one form of colonic stasis. The air is finally expelled as flatus through the anus. Its passage through the colon is frequently associated with gurgling noises, known as *borborygmi*.

**Diagnosis.**—The presence of excess of gas in the stomach and intestines can be seen by X-rays. The stomach may be small or large. Aerophagy when the air is sucked into the œsophagus, but does not enter the stomach, can be seen immediately after swallowing. The food is held up, probably owing to spasm, at the cardia for some time, and forms a column. The height of the column suddenly shortens when the œsophagus dilates owing to the entry of air, and lengthens again when the air is expelled (Leven).

**Treatment.**—The dyspeptic variety has already been considered. In the others the full state of affairs must be carefully explained to the patient, and he must be told to swallow as seldom as possible between meals. He can be assisted in this by holding a cigarette-holder in his mouth, or by wearing a tight band round the neck above the thyroid cartilage. By such means he is made aware of the beginning of the act and can stop himself. As long as he is not in public his saliva should be expectorated. Leven advises simple breathing exercises—a deep inspiration through the nose followed by a deep expiration through the mouth ten times every hour. He also advises bismuth carbonate and potassium bromide in small doses. At meal times fluids should not be sipped, but taken through a straw. The **Prognosis** is good. Hysterical cases may require special methods of treatment (*see* p. 907).

## GASTROPTOSIS

The term *ptosis* applied to the viscera means that they occupy a lower place in the abdomen than normal. *Gastroptosis* is met with alone, but is often associated with a dropping of the viscera generally—*visceroptosis* or *Glenard's disease* where there may be ptosis of the liver (*4*) (*hepatoptosis*), spleen (*splenoptosis*), kidney (rt. *19*) (*nephroptosis*), and colon (*47*) (*coloptosis*). All these conditions, but especially dropping of the colon and its flexures, may be met with in quite healthy people. Ptosis of these organs means dropping of the whole organ, but the term gastroptosis is not used in this sense, since the top of the stomach is practically always in contact with the diaphragm. It is the lower part of the stomach that is dropped; *i.e.* the viscus is elongated. Dropping of the diaphragm is rare (*2*).

**Ætiology.**—It has been pointed out on p. 405 that gastroptosis may occur in perfectly healthy people, *i.e.* those with a long, narrow chest and abdomen. The condition is three times as common in women as in men. It is rather commoner in women who have borne several children than in those who have had none, or at most one or two. It occurs chiefly above the age of thirty. Symptoms may be first noticed after acute specific fevers or appendicitis, or an abdominal operation, or a period of mental and physical strain. Over-exertion may cause it.

**Symptoms.**—Constipation is the commonest (*67*), then abdominal discomfort

(*53*), flatulence (*45*), loss of energy (*44*), abdominal pain (*43*), loss of weight (*36*), headache (*31*), nausea (*24*) and depression (*24*). Vomiting (*13*), abdominal distension (*12*) and acid eructations (*12*), diarrhœa (*6*) and heartburn (*3*) are uncommon. There is no reason to believe that gastric ulceration is commoner in gastroptosis than in cases with abdominal symptoms in whom the stomach is normal in position, but the X-ray appearances suggested that duodenal ulceration (*23*) was rather commoner. A coated tongue (*46*), inadequate teeth (*33*), pyorrhœa alveolaris (*19*) were common. Contrary to general belief gastric hyperacidity was frequent (*49*), but there was also hypo-acidity (*21*).[1]

**Diagnosis.**—Gastroptosis can be recognised by X-ray examination after an opaque meal, the patient being in the vertical position. Normally the lesser curvature is above the iliac crests, and the greater curvature below them. When the stomach is dropped the greater curvature may lie as low as the symphysis pubis in extreme cases (*see* Plate XX., p. 405). As already pointed out a low position of the stomach is compatible with health, and there are factors that may produce the condition temporarily (*see* p. 405). Gastroptosis is usually associated

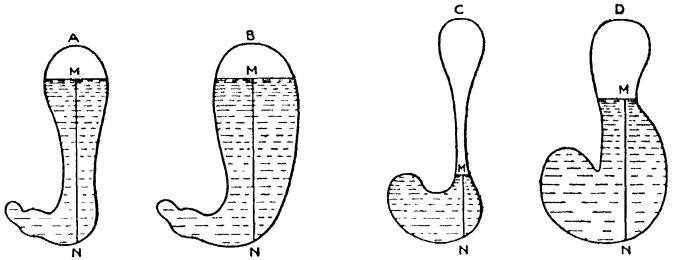



FIG. 52.

with lack of tone (*71*), and this is probably the most important factor in producing symptoms. Lack of tone is demonstrated by giving the opaque meal in successive quantities of 100 c.c. each. Normally the muscle reacts so as to keep the column of food at a constant height (M N in Fig. 52, A and B) whatever the amount given. When the stomach is hypotonic the first portion sinks to the bottom, and subsequent portions fill up the stomach like a bag (**12**) (*see* Fig. 52, C and D). Delay in emptying of the stomach is common (*57*), and there is often X-ray evidence of abnormality of the appendix (*59*), delay in the colon (*66*), coloptosis (*47*), and ileal stasis (*42*). The gastric peristaltic movements were active (*68*) in spite of the low stomach and frequent hypotonus.

**Prevention** consists in regular exercise throughout life, avoiding tight corsets and over-exertion, taking a diet which contains plenty of fruit and vegetables, cultivating regular defæcations, and promptly attending to any irregularity of the bowels or dyspeptic symptoms.

A clinical indication of gastroptosis is that pain is produced by pressing in the middle line above the umbilicus, and this is immediately relieved by simultaneously pressing in just above the symphysis pubis (**14**).

**Treatment.**—Little may be necessary in milder cases, though even here some strengthening of the abdominal muscles by gymnastic exercises, riding or fencing, may be valuable. Abdominal massage and electricity may be useful. In more pronounced cases, a belt should be worn continuously during the daytime: this must be made so as to exert firm pressure just above the symphysis;

[1] The *italic* numbers in brackets are percentages of 100 uncomplicated cases of gastroptosis with abdominal symptoms (**15**).

it is a good plan to have a small projecting cushion on the inner side of the belt for this purpose. Where the stomach is much dilated, small quantities should be eaten at a time, and after meals the patient should lie down on the right side, or walk on hands and feet, which will facilitate the passage of food through the pylorus.

### HYSTERICAL VOMITING

Like other hysterical manifestations, hysterical vomiting usually arises from some complaint of which vomiting is one of the symptoms, and is continued owing to "suggestion" after the original complaint has been cured. The vomiting which is a natural accompaniment of pregnancy may be continued in this way in suggestible individuals. Vomiting of this kind may continue after an attack of appendicitis, even when the appendix has been removed surgically. In the war vomiting was one of the symptoms of "gassing," and in many cases hysterical vomiting resulted.

**Treatment.**—As in other hysterical conditions, psychotherapy must be employed. The exact state of affairs must be explained to the patient. All special drugs and diets which were originally prescribed on account of the vomiting, but which may keep up the condition by suggestion, must be removed, and the patient must be encouraged to take ordinary food. In resistant cases the patient may be fed by a thin rubber tube passed through the stomach into the duodenum.

### CYCLICAL VOMITING

This curious complaint consists of attacks of vomiting occurring at intervals of from two weeks to three or four months, lasting on each occasion from one to three or four days, and especially frequent in children from one to six years of age. There is usually a marked family history.

In the midst of perfect health or with no other warnings than some loss of appetite, offensive breath, white tongue, nausea, and some vague pains in the abdomen, vomiting comes on at any time of the day or night. At first the gastric contents are brought up, then watery, glairy, or bilious fluids, and sometimes "coffee grounds." The vomiting is violent, and everything that is given to the child is rejected.

The vomit as well as the breath of the patient smells of acetone, and the acetone bodies—acetone, aceto-acetic acid, and $\beta$-oxybutyric acid—are found in the urine (ketosis). If the vomiting continues, the child rapidly emaciates, the abdomen is retracted, the face is drawn, and the eyes are sunken. There is generally obstinate constipation; there may be some abdominal pain, and there is slight or more marked pyrexia. The vomiting ends as suddenly as it began, and the child remains well until another attack.

Occasionally the attack is fatal, with headache, delirium, restlessness, convulsions, and collapse or coma from ketosis (*see* p. 568); and in fatal cases the liver has generally been found to be in a state of fatty degeneration. Recently hypo-glycæmia has been found during the attacks, possibly of hepatic origin.

**Prevention and Treatment.**—The attacks may be prevented and treated by giving plenty of sugar. If the child is unconscious 5 per cent. dextrose may be administered through a nasal tube passed as far as the pylorus or duodenum (*see* p. 576), and *per rectum*. The ketosis should be treated by giving plenty of fluids, sodium bicarbonate, etc., as described on p. 576, but insulin must not be given.

### ACHYLIA GASTRICA

This term was employed by Einhorn to describe conditions in which the gastric secretions, both pepsin and hydrochloric acid, are very deficient or absent. The

association of achylia—or, more correctly speaking, achlorhydria—with pernicious anæmia has long been recognised. Unfortunately of recent years this condition has been diagnosed by means of the fractional test-meal, and no account has been taken of the amount of mineral chlorides present, so that it is impossible to say how far the achlorhydria found in severe infections such as typhoid fever, tuberculosis and chronic appendicitis, and various nervous conditions and acne rosacea and visceroptosis may be due to defective secretion or increased neutralisation from duodenal regurgitation. True achylia, *i.e.*, defective secretion of HCl, is probably never present in health.

**Symptoms.**—There may be no gastric symptoms. Dyspeptic symptoms may be present, probably in those cases where excessive duodenal regurgitation has occurred. In another group of cases chronic diarrhœa is the main symptom.

**Treatment.**—Any dyspepsia should be treated on principles already described. In gastrogenous diarrhœa complete relief may be brought about, and in many of the other groups there is definite improvement. From 30 minims to 1 drachm of the acid hydrochlor. dil. may be given in half a tumbler of water at each meal, though smaller doses often achieve the same result. Fats, strong tea and alcohol should be avoided.

## ACUTE DILATATION OF THE STOMACH AND DUODENUM

(*Gastric and Duodenal Ileus.*)

*Stomach.*—Cases of this kind are comparatively rare, though many have now been recorded. Their occurrence is not easily explained; in the majority of cases there is no obvious cause of obstruction, but some have come on after overloading the stomach, especially with vegetables. Excess of gas is rapidly produced, and the stomach becomes distended like the rumen in sheep after eating green wheat. A few cases have occurred after injury, because, presumably, the gastric muscle is paralysed, and more than one-fourth after surgical operations. Here the anæsthetic is supposed to be responsible, especially if it is ether, because patients are apt to swallow air with this anæsthetic.

The onset is generally very sudden; the patient is seized with vomiting, and brings up frequently large quantities of green, brown, or grey fluid. With this are gastric discomfort, pain, and tenderness. The abdomen is generally found to be considerably swollen in its left and lower portions, while the epigastrium is relatively flat. Visible peristalsis is quite exceptional (once in forty-four cases collected by C. Thompson); but varying amounts of resonance, fluctuation, and splashing may be obtained. The patient becomes collapsed, suffers from thirst, the urine is scanty, and the bowels are confined. The symptoms may last a few days.

After death the stomach is found to be enormously distended, stretching down towards the pubes, and there bent on itself with a portion returning up towards the duodenum. The distension sometimes extends some way along the duodenum.

From observations by Box and Wallace it appears that when once the dilatation has begun the distended stomach falling in the abdomen causes a kink in the duodenum, and thereby an obstruction by which the escape of gases from the stomach is prevented, and so the condition is aggravated; and the more the gases accumulate, the more certainly are they prevented from escaping.

*Treatment.*—A tube should be passed so as to remove the contents from the stomach, which are under great pressure. The patient should lie face downwards, and the bottom of the bed should be raised so as to straighten out any kinks.

*Duodenum.*—In this condition there is obstruction due to kinking, possibly by the mesenteric vessels, with or without spasm near the duodeno-jejunal flexure. A chronic condition of dilatation of the duodenum is probably not very uncommon, accompanied by dyspeptic symptoms, distension of the abdomen and bilious

vomiting (**17**). These may be followed by acute dilatation, the symptoms of which resemble acute dilatation of the stomach. The diagnosis would be suggested, if passing a tube into the stomach did not relieve the condition, and laparotomy should then be performed.

## ACUTE GASTRITIS

**Ætiology.**—Acute inflammation of the stomach, or acute gastric catarrh, may be set up by various forms of irritants. The most intense form of gastritis occurs in poisoning by the strong mineral acids, or other corrosives (*toxic gastritis*). The more common cases arise in consequence of the use of indigestible food, such as lobster, crab, or shell-fish, or of unripe fruit, or of flesh, fish, fruit, vegetables, or other food which is in a state of commencing decomposition, and contains ptomaines or the *Bacillus enteritidis* (*see* p. 447). It is thus common in hot weather. Infants frequently suffer from gastritis associated with enteritis (*see* p. 444). Gastritis may also be due to various infections (*infective gastritis*); for instance, pneumococcal, typhoid, syphilitic, and tuberculous forms of gastritis have been recognised. Multiple gastric erosions, described under the heading *acute gastric ulcer*, form one variety of acute gastritis.

**Morbid Anatomy.**—In the majority of cases nothing can be known of the condition of the mucous membrane of the stomach, since recovery takes place; and the changes which have been found in the stomach after death from the infectious diseases must not too readily be assumed as identical with those occurring in ordinary cases, since there is an absence of the symptoms characteristic of these last. But in the well-known case of Alexis St. Martin it was shown that changes quickly followed irritation of the mucous membrane. Red pimples appeared, which were sometimes filled with purulent matter, or there were red patches, or aphthous crusts, or abrasions. The gastric juice was secreted in less quantity, and mucus was poured out freely. Slight hæmorrhage also occurred sometimes. Ziegler states that in gastritis the mucous membrane is dark red and swollen, beset with small hæmorrhages, and covered with a film of mucus, mucoid epithelium, and extravasated leucocytes. The cylindrical epithelial cells of the gland ducts are in an extreme stage of mucoid change, and many desquamate; and the epithelial cells of the peptic glands are detached, and seem more granular than usual. The vessels of the interglandular tissue are distended; and the sub-glandular tissue and even the submucous layer are infiltrated. Abrasion and ulceration may also occur, often perhaps at the seats of previous hæmorrhages; and as later results induration and atrophy of the mucous membrane supervene.

**Symptoms.**—In corrosive poisoning the symptoms are briefly acute pain and tenderness in the epigastrium, vomiting of blood and mucus, and collapse; death is a frequent result. These cases are described in works on toxicology.

In the more familiar cases of acute gastritis there is a feeling of weight or oppression at the epigastrium, and in many cases actual pain, which is increased by pressure, or by the ingestion of food. With this there are nausea and retching, or vomiting is at once produced by the introduction of anything into the stomach. The vomited matters at first consist of particles of food; afterwards they are watery or mucous, or stained with bile. The patient is dull and heavy, with some headache, generally constipation, loss of appetite, thirst, an unpleasant taste in the mouth, a thickly furred tongue, and offensive breath. There is sometimes decided febrile reaction, and the pulse is soft and quick. Examination of the abdomen shows that the epigastric region is hard and tense, and sensitive to pressure. In many cases of food poisoning the intestinal symptoms predominate (*see* p. 447)

In infants the complaint is generally associated with diarrhœa. The little patient is constantly fretting or whining from pain; the legs are drawn up to

relieve it; the abdomen is tense and tender; food is either refused, or, if taken ravenously to quench thirst, is as rapidly rejected; emaciation soon occurs, and death may be the result. Except in the case of infants, the disease generally subsides in the course of a week or two, but repeated attacks may lead to a chronic condition.

**Diagnosis.**—This is generally simple; but acute gastric catarrh may be confounded with the early stages of *enteric fever*, and with *appendicitis*. The former difficulty may not be cleared up until the appearance of rose spots and a typical diarrhœa, or reaction to Widal's test; in appendicitis there is generally acute or rapidly developed pain towards the right side, early spontaneous vomiting, and tenderness in the appendical region.

**Treatment.** In severe cases food should be stopped altogether for a time, and as little as possible of anything should be introduced into the stomach. In milder cases very small quantities of milk and soda water may be allowed, or peptonised milk, or koumiss; and thirst may be quenched by iced soda water or seltzer water, or small pieces of ice. For the pain, hot fomentations or poultices may be used, or, in very severe cases, leeches may be applied to the epigastrium, or morphia injected. The same drug will sometimes allay continued vomiting; bismuth and an effervescing draught (*see* p. 414), or 2 or 3 minims of tincture of iodine in a teaspoonful of water given every half-hour, are also useful. Washing out the stomach by syphonage may often be useful at the beginning of an attack.

As the symptoms subside the food may be gradually increased—milk in larger quantities and more often, then light puddings, dry toast, a little fish, and so on to the normal diet of health.

*Chronic Gastritis* is described on p. 411.

### ACUTE SUPPURATIVE GASTRITIS

Suppuration of the walls of the stomach is a rare event, and occurs either in the form of a circumscribed abscess or as a purulent infiltration. The abscess may be of the size of a walnut, a hen's egg, or larger, and is more often in the submucous than in the subserous layer; it may burst into the stomach or into the peritoneum. The cause may be pyæmic or puerperal infection, but has often been unexplained. There are severe pain in the abdomen, worst in the gastric region, and increased on pressure; vomiting; thirst; intense fever. The abscess has been sometimes felt as a tumour when the diagnosis, otherwise difficult, becomes possible. This might justify an operation.

## PEPTIC ULCER

*(Ulcer of the Stomach and Duodenum.)*

Ulceration is very apt to occur on mucous membranes that habitually come into contact with the acid gastric juice. Such ulcers are conveniently called *peptic*, and there are several varieties. The *acute* ulcer, most commonly multiple, varies in size from a tiny superficial erosion of the mucous membrane up to an inch in diameter. *Chronic* ulcers, usually single, are best classified according to their positions: (1) in the body of the stomach, usually on the lesser curvature, and on the posterior rather than the anterior surface; (2) in the pyloric part of the stomach, also usually on the lesser curvature and posteriorly, but not extending nearer to the pyloric ring than ¼ inch; (3) in the first part of the duodenum, usually posteriorly. In this connection it must be remembered that the first part of the duodenum has a completely different function from the other parts, but resembles more closely the pyloric part of the stomach. Thus the acid chyme is pushed through the pylorus into the first part of the duodenum, and remains

there until the next peristaltic wave pushes some more chyme forward to take its place. Ulcers situated actually at the pyloric ring are uncommon. Ulcers in the body of the stomach often have distinct characteristics, and much confusion has arisen from grouping the pyloric ulcers and these ulcers together under the heading *gastric*, merely because they both happen to be in the stomach, whereas the pyloric ulcers have a much closer affinity with those in the duodenum. Pyloric and duodenal ulcers should be considered together as *juxta-pyloric*.

**Ætiology.**—Acute peptic ulcers occur scattered about irregularly, and are probably quite common in both sexes; but they are commoner in women and tend especially to affect the body of the stomach. Some ulcers in this situation become chronic, though most of them heal readily enough. However, they may come again later and again heal. A continuation of this process, viz. acute relapsing ulceration, tends to the formation of an hour-glass stomach, which is much commoner in women than in men (**18**). While peptic ulceration in women occurs most commonly in the body of the stomach, in men it occurs most commonly in the juxta-pyloric region and does not heal so well. Consequently we find (1) that chronic duodenal ulcer is four to six times commoner in men than in women, whereas (2) chronic gastric ulcer is about equally common in both sexes, since the preponderance of the male ulceration in the pyloric region about neutralises the female preponderance in the body of the stomach, and (3) ulcer scars are more commonly met with in the body of the stomach (**19**). Peptic ulcer has a tendency to run in families. Chlorosis and anæmia are not predisposing causes. Very occasionally gastric ulcer is due to syphilis. Painless hæmatemesis, so common in young women, which has been called *gastrostaxis*, is due to acute ulceration. The lesions are often so small that they are called hæmorrhagic erosions, and so widespread that the mucous membrane may present the appearance of "weeping blood." Such a condition is one form of acute gastritis. Duodenal ulcer has been described in infants.

**Morbid Anatomy.**—*Acute Ulcer.*—In the earliest stage there may be a superficial necrotic lesion with reddened margins situated on a slightly raised and thickened patch of mucosa. Then excavation occurs and tiny vessels are exposed in the floor, which is often covered with a thin black layer of altered blood. This layer separates, leaving a smooth clean floor before healing. The ulcers have a punched-out appearance; the smaller ones are quite shallow, affecting only the mucosa; the larger ones penetrate the muscular coat and have a "terraced" appearance, because the ulcer becomes narrower the more deeply it penetrates. The *chronic ulcer* is generally much larger, and may reach a diameter of 5 or 6 inches. It extends deeply into the wall of the stomach; the edges are thickened and raised, from infiltration with inflammatory fibroid material, and overhang the ulcerated surface; and the thickening extends some little way into the surrounding mucous membrane. When *active*, the chronic ulcer has a narrow zone of sloughing inflammatory exudation in the floor. When *healing*, the floor becomes clean by separation of the slough, the margins become flatter, and epithelium grows inwards over the floor. In a *callous* ulcer the process of healing remains stationary.

When the ulceration reaches the peritoneum this may rupture so that perforation takes place. This is commoner on the anterior than the posterior wall of both stomach and duodenum. The visceral contents escape into the peritoneal cavity and set up intense general peritonitis, or a more localised abscess, *perigastric abscess*, or *subphrenic abscess* (*see* p. 512). This abscess may perforate the diaphragm and set up pneumonia, pleurisy or pericarditis, or it may perforate the colon or duodenum, or open again into the general peritoneal cavity. The term *chronic perforation* is used for an ulcer which leaks into the peritoneal cavity a little at a time over a long period, so that adhesions form and shut off the main peritoneal cavity. These may be so dense as to suggest carcinoma. The stomach becomes connected through the ulcer with a large cavity outside the

stomach, sometimes involving the greater part of the lesser sac. More often the inflammatory process, extending to the serous surface, causes the stomach to adhere to one of the adjacent parts before perforation can occur. This is most frequently the pancreas or the left lobe of the liver, but adhesion also takes place occasionally to the diaphragm, spleen, colon, anterior abdominal wall and even the suprarenal capsule. The ulcerative process then extends into the newly attached organ, and cavities may be formed in the liver and pancreas. Very rarely fistulæ may be produced into the colon or through the skin. An old ulcer may cause so much adhesion and matting together of the parts that a carcinomatous tumour is closely simulated. Hæmorrhage is a common accident, mostly from gastric vessels in the wall of the ulcer, but sometimes from the splenic artery after adhesion to, and ulceration of, the pancreas.

But many ulcers recover completely, and small scars are often found. They are often very difficult to see, and are frequently missed at post-mortem. Larger scars, which are thick and puckered, may themselves give rise to considerable trouble. Thus at and near the pylorus they may by their contraction cause *stenosis*, and consequent *dilatation of the stomach;* if near the cardiac extremity, the stomach may be contracted. Sometimes an *hour-glass contraction* is due to ulcer. Ulcer may set up a chronic gastritis, or rarely a suppurative gastritis; the adhesions to surrounding parts (*perigastric adhesions*) sometimes give rise to pain and dragging sensations. The relation of gastric ulcer to carcinoma is considered on p. 431.

**Pathology.**—Bolton produced ulcers experimentally in animals by preparing a special gastrotoxic serum and injecting it beneath the peritoneal surface of the organ. The cells were devitalised, and the HCl of the stomach took part in the digestion of these cells with the formation of an *acute ulcer*. No ulcer was formed if the stomach contents were kept alkaline. The naturally occurring acute ulcer in man can be explained along these lines. There may be several different factors that play the part of Bolton's gastrotoxic serum, such as embolism, or venous thrombosis of small vessels or hæmorrhage, sometimes due to portal obstruction. The stomach may on rare occasions be injured directly by a blow. Most important of all, micro-organisms, or their toxins, may reach the stomach through the blood stream from septic foci elsewhere, a chronically inflamed appendix, or gall-bladder, septic teeth, extensive and infected burns, or during an acute infection. The HCl of the stomach completes the process forming the ulcer. Bolton's ulcers, experimentally produced, healed readily in a few weeks. The rate of healing was very little altered by trying to infect the ulcers with bacterial cultures, or by bleeding the animal so as to make it anæmic. Healing was, however, definitely delayed by feeding the animals (cats) with meat instead of milk, and by producing some occlusion of the pylorus, so that there was delayed emptying of the stomach. In monkeys administration of HCl caused some delay in healing, and this was greatly accentuated by partially occluding the pylorus. There is no doubt that in man many ulcers heal readily enough, but there is a proportion of them that persist and gradually change into the chronic ulcer for two possible reasons: (1) The hydrochloric acid may play a part in this change; but observations that gastrojejunostomy, in the case of juxta-pyloric ulcer, produces very little alteration in the HCl of the gastric juice suggest that this factor is not of great importance (**22**). (2) Increased movements of the stomach leading to feelings of pain or discomfort are also common, and these movements, which are probably associated with the closure of sphincters or spasms in various parts of the upper alimentary tract, may be an even more important factor, since relatively high pressures have been recorded (**2**). It is possible that a vicious circle becomes established, the ulcer causing the movements and pain in the first place, which then tend to prevent it from healing. The hydrochloric acid may perhaps act by producing reflex closure of the pylorus and so increasing the

mechanical factor. Finally, mention must be made of the fact that strains of streptococci of low virulence have been isolated from tonsils and from various sources, which produce peptic ulcer in animals on intravenous injections, and the same micro-organisms have been found naturally occurring in the ulcers of man, so that they appear to have a specific affinity for the stomach and duodenum, in the same way that typhoid bacilli attack the gut. This phenomenon has been called "elective localisation" (**23**).

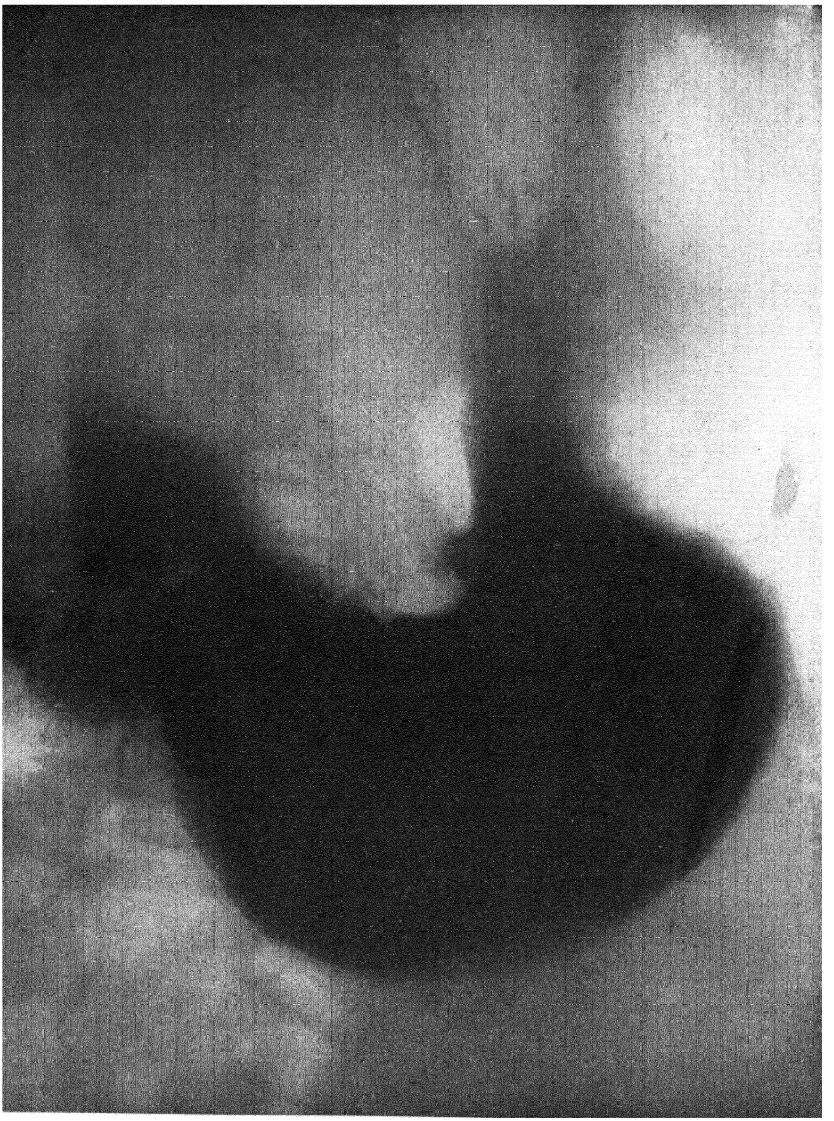
**Symptoms.**—*Acute Ulcer.*—In a large number of cases the first symptom is due to hæmorrhage from the ulcer. In cases of *gastric ulcer* this often leads to *hæmatemesis*, or vomiting of blood, which may be pure or mixed with gastric contents. The patient feels faint, has a sense of oppression in the epigastrium, and in a few minutes vomits the blood, which may amount to 1, 2, or 3 pints. Some of the blood discharged into the stomach finds its way into the intestine; the hæmoglobin is converted into hæmatin and hæmatoporphyrin, and the motions subsequently passed are black, treacly, or tarry, constituting *melæna;* these may appear some hours after the hæmatemesis has ceased. The vomiting of pure blood is rarely fatal; it usually ceases entirely, and may not be repeated. High degrees of anæmia and weakness result from the loss of blood. In *duodenal ulcer* hæmatemesis may also occur; but the tendency is for most of the blood to be discharged *per rectum* as melæna. Apart from hæmorrhage, peptic ulcer may produce dyspeptic symptoms of acute onset, associated perhaps with prolongation of the gastric secretion, as already described (on p. 410). *Perforation* of the ulcer rarely gives the first indication of its presence.

*Chronic ulcer of the body of the stomach* is characterised by *pain* which is felt deeply in the epigastrium, just below the ensiform cartilage, sometimes nearer the umbilicus, or to the right or left of the middle line, the right more often than the left. It is brought on by the ingestion of food, appearing from half an hour to two hours after a meal; it may continue intense until vomiting takes place, by which it is generally relieved, or it subsides as the food leaves the stomach. In character it is piercing, boring, tearing, or burning, and more severe than in any other gastric disorder. Sometimes there is pain in the back, between the eighth dorsal and the second lumbar vertebræ, and even over the forehead. Associated with the pain, and often noticed for some little time after the pain has disappeared, there is cutaneous hyperæsthesia or soreness experienced when the skin is lightly pinched. Heartburn (*see* p. 412) is rather common, but hunger pains are only found in one quarter of the cases; but complete emptying of the stomach is often delayed, though the food passes out readily to begin with (**19**). The secretion of HCl is found to be normal (**22**).

*Vomiting* is nearly always determined by the food, and the matter vomited consists of food. Blood is commonly present in small quantities, and mixed with the contents of the stomach, so that the hæmoglobin is converted into hæmatin by the acid gastric juice; and the vomited matter has the turbid, blackish-brown appearance which has been compared to *coffee grounds*. Hæmorrhage may be severe. The course of the disease is very prolonged; but remissions of symptoms are common.

*Chronic Juxta-pyloric Ulcer.*—The pain resembles in its character that already described; but it is more usually felt to the right of the umbilicus, and it comes on two, three or four hours after food, or wakes up the patient in the early morning. The pain is relieved by taking some food, and so has been called *hunger pain*. There are two types of cases: (1) When the pylorus opens freely, the stomach readily empties itself of food. There is marked hyperchlorhydria of the resting juice, perhaps most marked in duodenal ulcer, and there is a prolonged secretion of highly acid juice after the stomach is empty; (2) When the pylorus does not open freely, which constitutes the early stage of a pyloric stenosis, the hydrochloric acid of the gastric juice shows the climbing curve also with prolonged secretion and hyperchlorhydria. There is marked delay in emptying and

## PLATE XXI.



**Radiogram of a Stomach showing a Niche on the Lesser Curvature due to Ulcer.** The greater curvature above the niche is somewhat convex towards the stomach. This is probably due to slight spasm. There is no cicatricial contraction and no hour-glass stomach as in Plate XXIV. The stomach is dropped; the lowest part reaches a long way below the iliac crest. Some food has left the stomach, and the duodenal cap is well outlined. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 425.

vomiting is common. Hæmorrhage may be severe or fatal when a large artery is eroded. In this case the vomited blood retains its arterial brightness. There may be remission of symptoms, but the history of the disease is not so long as in ulcer of the body of the stomach, because, owing to the beginning pyloric stenosis, the symptoms are usually more severe and radical treatment more necessary.

In chronic peptic ulcer other symptoms described under chronic dyspepsia, such as flatulence and distension, are commonly present, the continued pain, the defective assimilation of food from vomiting, and the loss of blood, naturally impair the general condition of the patient sooner or later; but there is no fever, the tongue is clean, and the appetite is often very good and nausea absent. Constipation, however, is frequent. Examination of the abdomen generally reveals nothing; there may be some hardness or tenseness of the abdominal walls. Only in the case of old ulcers with much thickening, or adhesion to other organs, can anything like a tumour be felt; and if pyloric stenosis results, the dilated stomach may be recognised (*see* Plate XXIV.), and there may be visible peristalsis. After hæmatemesis there is a characteristic pallor, with the usual hæmic murmurs over the præcordia (*see* Anæmia).

**Diagnosis.**—In a case of hæmatemesis where hæmorrhage is the only symptom cirrhosis of the liver and splenic anæmia must be thought of before the diagnosis of acute ulcer of infective origin is made. Hæmatemesis must not be confounded with hæmoptysis or with vomiting of blood after epistaxis. The complaints likely to be confounded with chronic peptic ulcer are the various forms of dyspepsia already described, carcinoma and chronic appendicitis (appendix dyspepsia), or the dyspepsia associated with gall stones.

A test meal which shows a high concentration of "free" or "active" HCl will favour juxta-pyloric ulcer. The presence of a trace of blood in the meal is suggestive, but it may be produced by slight trauma of the mucous membrane caused by the tube. The presence of occult blood in the stools and of a hæmatoporphyrin spectrum in the fæcal extract is, however, valuable evidence in favour of gastric or duodenal ulcer.

The most valuable evidence in favour of chronic gastric ulcer is obtained by X-rays after an opaque meal. A small projection in the shape of a funnel or sac (*niche* or *diverticulum*) is seen on the edge of the stomach near the lesser curvature (*see* Plate XXI.). The projection depends on the presence of the stomach contents in the cavity of the ulcer, and will not be seen if the cavity is closing up in the process of healing. An ulcer cavity on the posterior surface of the stomach will not be seen when the stomach is full. In order to diagnose it, it is necessary, after the patient has taken the first mouthful, to manipulate the abdominal wall so as to spread the opaque material over the stomach wall. Some of it will stick in the ulcer cavity, and show it up. In some, but not in all, cases the projection is associated with a deep indentation of the greater curvature or *notch* at the same level or slightly above or below it, due to spasm, and there may be localised painful spots. The indentation may be seen alone without the projection, and may remain constant in position when from the symptoms it would be judged that the ulcer had healed. In such a case the deformity may become permanent owing to the deposition of fibrous tissue, and a cicatricial *hour-glass stomach* is produced. The indentation may occur with duodenal ulcer or appendicitis, and so by itself it is not diagnostic of gastric ulcer. It is also important to see how long it takes for the stomach to empty, so as to detect pyloric spasm as well as cicatricial pyloric stenosis. A positive Wassermann reaction will suggest that a gastric ulcer is syphilitic, and anti-syphilitic measures (*see* p. 116) should be instituted in addition to other appropriate medical treatment (**21**).

Chronic duodenal ulcer may be diagnosed by the X-ray appearance of the duodenal cap. The latter is shown up best if sodium bicarbonate is given with

the opaque meal. The appearances are very variable, depending partly on the cavity of the ulcer and partly on spasm of the muscular coat (*see* Plates XXII., XXIII.).

**Prognosis.**—Analysis of the cause of death in 150 cases of chronic gastric ulcer and 200 cases of chronic duodenal ulcer considered together showed that in 51 per cent. it was perforation, which was commoner in duodenal than gastric ulcer, 9 per cent. hæmorrhage, 5 per cent. the effects of long obstruction, carcinoma, etc., 17 per cent. the result of operation, particularly pulmonary complications, and in 18 per cent. the cause was not related to the ulcer (**20**). In *acute* ulcer hæmorrhage, though common, is rarely fatal, and perforation is also rare. In *chronic* ulcer hæmorrhage may be fatal and perforation is not uncommon, but these complications will nearly always be preceded by severe pains of some duration, which indicates that efficient treatment is essential. The absence of pain suggests that the ulcer is healing and these complications are then unlikely. Chronic ulcers of the largest size will heal under conditions favouring the absence of symptoms, especially pain, though the process may take months.

**Treatment.**—The most important indication is to give the stomach as much rest as possible. The patient must go to bed and not smoke, and for some weeks afterwards should take but little exercise. The old method of trying to "rest" the stomach by not allowing the patient to take food by the mouth for weeks at a time, but feeding him rectally instead, has now been given up; the well-known "hunger contractions" of the empty stomach show that this treatment was based on a fallacy. In 1901 Lenhartz introduced his dietary in opposition to the fasting treatment. The principle consists in using concentrated protein food, giving small meals at frequent intervals so as not to distend the stomach, and keeping the food iced, an ice bag being also placed on the epigastrium.

The diet consists of eggs beaten up with milk and sugar. On the first day one egg is given with 6 or 8 ounces of milk, and each day one egg and 4 ounces of milk are added until the patient is taking six or eight eggs in twenty-four hours with 2 pints of milk. This amount is continued through the second week. At the end of the first week 1 ounce of raw minced meat is given, and a little more on successive days; on the seventh or eighth day boiled rice is added, and later softened bread, bread and butter, and pounded fish, which gradually in later weeks replace the eggs. In this country it is better to substitute cooked minced chicken or meat for the raw minced meat of the diet. The patient is fed at hourly or two-hourly intervals during the day, but complete rest is given during the night. Subsequent experience has shown that, provided the main principle of small meals at frequent intervals is kept in mind, the details of the diet can be considerably modified. Thus most patients find it difficult to take so many eggs as Lenhartz prescribes, and thin bread and butter may be substituted quite early. Junket, arrowroot, custard, potato or artichoke purée, Benger's food and red currant jelly can also be given quite early in the treatment. Treatment in bed should be carried out for fourteen days after pain, rigidity, occult blood in the stools and X-ray evidence of ulcer have disappeared.

Alkalies are much used in treating peptic ulcer, and in Sippy's diet the attempt is made by frequent doses to keep the gastric contents always alkaline; in fact, in some cases enough alkali has been given to cause nephritis and uræmia (**24**). A doubt has already been expressed as to whether the acid of the gastric juice is really responsible for keeping up a chronic ulcer, and in any case it would seem more rational to counteract a hyperchlorhydria by means of giving fat in the form of cream or olive oil, etc. The alkalis used are 10 to 30 grains bismuth carbonate suspended in mucilage, 30 grains of prepared chalk, 5 to 15 grains of magnesium carbonate or magnesium oxide suspended in milk. These substances often act beneficially by relieving pain, including heartburn; but this may be because $CO_2$ is liberated; and very successful results may be obtained by prescribing a little citric acid at the same time (*see* p. 414). The aim should be to keep the

A.

Radiogram showing Normal Duodenal Cap. It is triangular in shape, and underneath it the pyloric part of the stomach with a peristaltic wave is seen. The upper right border of the cap appears crenated, because the wall of the second part of the duodenum happens to be visible here.

B.

Radiogram showing Duodenal Ulcer. The duodenal cap is deformed owing to the presence of an ulcer cavity, associated with spasm and possibly cicatrisation. The pyloric part of the stomach is also shown. (From plates taken by Mr. W. Lindsay Locke.)

A.

Radiogram showing Deformed Duodenal Cap from Ulceration. A peristaltic wave is traversing the pyloric part of the stomach, and food is passing through the narrow pyloric canal.

B.

Radiogram showing an Extremely Small Deformed Duodenal Cap from Ulceration. Coils of duodenum and jejunums are shown by their crenated borders. The round dark shadow with peristaltic wave is the pyloric part of the stomach. (From plates taken by Mr. W. Lindsay Locke.)

[To face p. 426

patient for a prolonged period free from pain or discomfort, and so the ulcer will slowly heal. Other drugs for treating pain are tinct. belladonnæ ℥v—x. and atropine injection gr. $\frac{1}{100}$, opium in small doses of the extract or tincture, or the liquor morphinæ hydrochloridi in 10 or 15-minim doses. In severe cases the hypodermic injection may be used, but the opiate treatment must always be discontinued as soon as relief is obtained. Local applications to the epigastrium may be used (*see* p. 414). Constipation may be treated by cold water enemata, or Carlsbad salts before breakfast, or by compound liquorice powder, or pills containing rhubarb or aloes. Vomiting may be checked by morphia or bismuth and morphia, by effervescing medicines, or by tincture of iodine (℥ij in water ℥ij every hour.

If a profuse hæmorrhage occurs, the patient must be kept at rest under morphia, and calcium chloride gr. i in 100 minims of water may be injected intramuscularly (*see also* p. 264). A stomach tube should be passed and the contents removed with a Senoran's evacuator and the stomach washed out with small amounts either of ice-cold water or water at 130° F., so as to stimulate it to contract down. Adrenaline (20 to 30 drops of a 1 in 1000 solution) may be left in the stomach. No food must be taken for twenty-four hours after the hæmorrhage has stopped, but water may be drunk in amounts not exceeding 5 ounces at a time. Ice should be applied to the epigastrium. When death is threatened, blood transfusion may be carried out. This does not tend to keep up the hæmorrhage, as might be expected owing to the rise of blood pressure. Surgical measures, such as suturing the bleeding vessel, have also been successful in stopping hæmorrhage from a chronic ulcer.

If a patient known to suffer from gastric ulcer is seized with the symptoms of perforation (*see* Peritonitis), the abdomen should be opened as soon as possible—*i.e.* within five or six hours—the peritoneal cavity washed out, and the ulcer sutured.

In ulceration of old standing, with frequent recurrence of pain and vomiting, or of severe hæmorrhages, or with evidence of much thickening about the ulcer, operation will naturally be considered. This has been most successful in cases where the ulcer is accompanied by stenosis of the pylorus or cicatricial hour-glass stomach. The operations practised for chronic gastric ulcer are gastro-jejunostomy, sometimes with excision of the ulcer, or its destruction by the cautery or with jejunostomy, gastro-gastrostomy, partial gastrectomy (removal of pyloric part of stomach), and the median "sleeve" resection of the stomach. For chronic duodenal ulcer the operations are excision of the ulcer, gastro-duodeno-pyloroplasty and gastro-jejunostomy.

**Prevention.**—When an ulcer is healed, care must be taken to prevent a relapse, which readily occurs. Septic foci in the teeth, tonsils, appendix, gall-bladder, etc., must be dealt with, if necessary by operation. Meals must be light and taken at frequent regular intervals, and the rules of diet on p. 426 should be followed. Alcohol is best avoided, though light wine and very weak whisky may perhaps be allowed at meals. He must eat slowly and masticate thoroughly and only smoke a little. The teeth must be looked after. The patient must be prepared to go to bed on a diet principally of milk at the onset of pain, and olive oil may be taken at meals.

**Sequelæ of Gastro-jejunostomy.**—Statistics show that a cure is by no means invariable after gastro-jejunostomy. Out of 108 cases operated on at Guy's Hospital between 1910 and 1915, 65 per cent. were cured or much improved seven years later, while 35 per cent. were unsatisfactory. The best results were in pyloric stenosis (**25**). In a series of fifty-one cases complaining of symptoms subsequent to gastro-jejunostomy, examination showed a variety of conditions: Gastro-jejunal or jejunal ulcer (*20*), delay in stomach (*18*), recurrent duodenal ulcer (*10*), regurgitation of bile (*8*), dyspepsia due to operation (*24*), etc. The symptoms complained of were pain (*50*), flatulence or distension (*40*), vomiting

(*40*), weakness (*38*), constipation (*38*), diarrhœa (*22*), and headaches (*22*). Patients may show glycosuria and there may be deficient assimilation of fat (**27**).

*Gastro-jejunal and Jejunal Ulcers.*—The gastro-jejunal ulcer occurs at the actual site of the anastomosis, and in most cases it is due to the use of unabsorbable suture material or hæmatoma or bruising by clamps at the operation (**26**). Jejunal ulceration occurs just beyond the junction, and is evidently due to the presence of the same factors as cause a duodenal ulcer, such as infection from focal sepsis that has not been eradicated, hyperchlorhydria which has persisted in spite of operation, and spasms round the stoma or in the descending loop and altered motility. The symptoms are the same as in duodenal ulcer, except that the site of the pain is now rather lower and on the left side of the abdomen. When the ulcer has burrowed down on to the colon, there may be epigastric pain on defæcation. Sometimes jejuno-colic fistulæ occur, with vomiting of fæculent material. These are usually not seen by X-ray examination after an opaque meal.

**Treatment.**—Secondary ulceration must be treated on the same lines as peptic ulcer. Operation may be required and if the pylorus is acting well, the gastro-jejunostomy may be undone. The treatment of the other conditions depends on the X-ray findings. If the stomach empties too rapidly, liquids should not be taken at meals. Massage and gastric lavage may be beneficial. Usually these patients do not tolerate fat well.[1]

## DILATATION OF THE STOMACH

Dilatation of the stomach may take place very gradually (chronic dilatation), or may occur quite suddenly (acute dilatation). The latter has been already described.

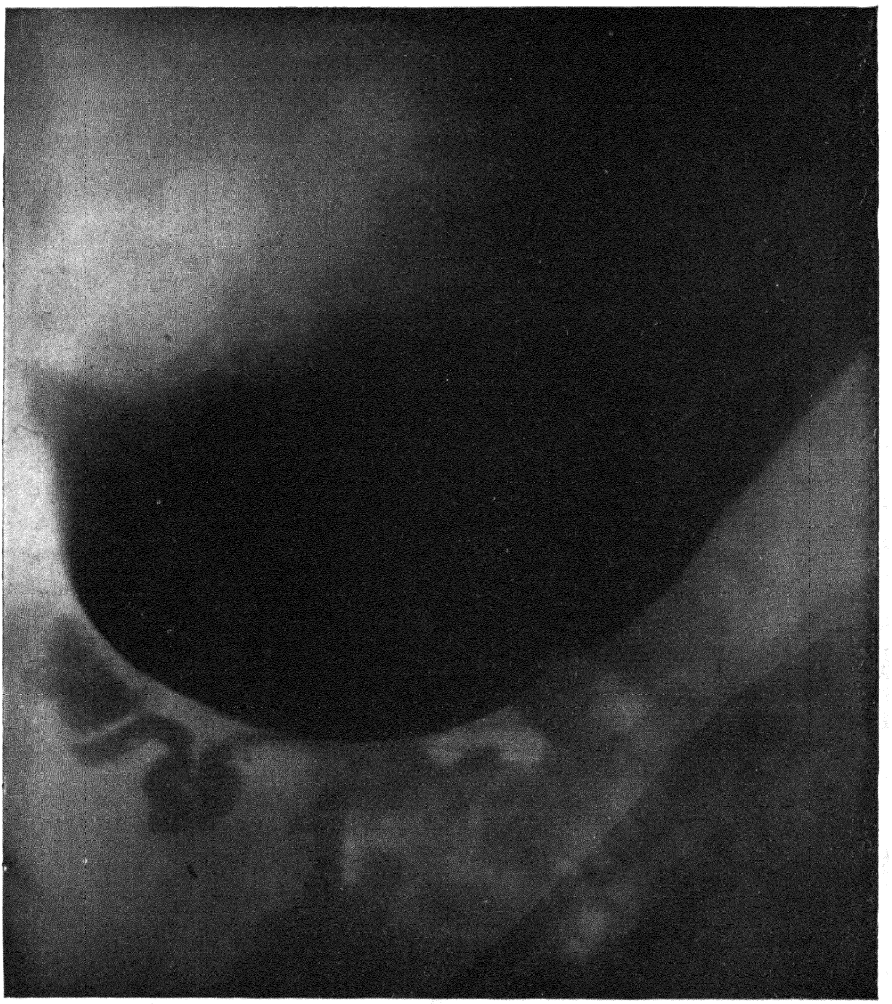
### CHRONIC DILATATION

This results (1) from the various conditions which produce obstruction of the pylorus, so that the stomach wall is also hypertrophied, and (2) from conditions which alter the contractile power of the muscular walls (*see* Gastroptosis). The causes of obstruction are cicatrices of ulcers of the pylorus or duodenum; spasm of the pylorus secondary to neighbouring ulceration; carcinoma of the pyloric part of the stomach; hypertrophic stenosis of the pylorus; pressure from without, binding down by adhesions, or dragging of a prolapsed kidney; and, quite exceptionally, cicatrices from corrosive substances, which, however, generally involve the œsophageal aperture.

**Physical Signs of Dilatation following Pyloric Stenosis.**—In marked cases, when the abdomen is exposed, it is seen to be asymmetrical, presenting a rounded *prominence* in its left half. This prominence extends below the level of the umbilicus, its lower margin having a curve convex downwards and outwards, from the lower part of the costal margin to the right of the middle line. From time to time a wave of peristaltic movement passes from left to right and downwards across the prominent part. A portion at the extreme left, about the size of the palm of the hand, quickly forms a convex prominence, with a decided amount of resistance to pressure; in a few seconds the swelling subsides, and another part, more to the right, swells up for a similar length of time. After each successive portion of the stomach wall has become hard and prominent the whole subsides. This phenomenon occurs spontaneously, or may be set up by manipulating the abdominal wall, or flicking it with the finger sharply, or sometimes on mere exposure of the abdomen. It is called *visible peristalsis*. By sharp movements of the abdomen, as when the patient is shaken, the liquid contents are set in motion, and *splashing* can be heard and felt. This, however, has no significance unless it can be recognised over an abnormal area, as, for

[1] The numbers in *italics* refer to percentages.

PLATE XXIV.



Radiogram of a Dilated Stomach secondary to Pyloric Stenosis following Ulceration. The stomach is large, and reaches transversely across the abdomen, and the greater curvature extends some distance below the iliac crest. It is full of barium, two hours after the meal; but small portions of the meal have passed through the pylorus and are seen scattered about in the coils of small intestine. A slight wave of peristalsis is seen on the greater curvature at the beginning of the pyloric part of the stomach. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p* 429.

instance, as low as an inch from the umbilicus, or at a time when normally the stomach should be empty, namely, six or seven hours after a meal.

A striking feature of many cases of chronic dilatation is the manner in which vomiting takes place. The food is retained for three or four days, and then 2 or 3 pints of fluid are vomited at once. It is generally of a greyish-brown colour, frothing on the surface; and on microscopic examination it shows numerous yeast spores, sarcinæ, and long rod-shaped bacilli, the *Oppler-Boas bacilli*. In other cases the vomiting is more frequent, and the quantity ejected is less at a time.

In addition to the vomiting, the patient suffers from discomfort or actual pain, which is increased as the contents accumulate, and is temporarily relieved after they are evacuated. Great thirst, loss of strength, emaciation, pallor, and constipation are also observed. Much mental depression, and sometimes tetany and convulsions, may also occur. The urine is scanty, and there may be ketosis.

**Diagnosis.**—This depends ultimately on an X-ray examination after an opaque meal (*see* Plate XXIV.). The stomach is enlarged downwards and to the right. At first there may be over-active peristalsis with rapid emptying. Peristalsis then becomes intermittent and the waves of variable depth, so that there is a residue of about half the meal in six hours. In the later stages peristalsis may only be seen occasionally and it may travel in the reverse direction, and the stomach may remain full after twenty-four hours. A fractional test meal (*see* Fig. 51) also shows delay in emptying and a steadily rising free HCl curve.

**Prognosis.**—Dilatation from narrowing of the pylorus must persist as long as the disease which causes obstruction, and treatment other than surgical can only be palliative. When the distension results from weakening of the muscular walls, the outlook is more favourable, and recovery may take place.

**Treatment.**—For dilatation of the stomach the operation of washing it out (*lavage*) is often of great value. The over-distended organ is thus relieved of the accumulation of liquid and undigested food, and washed out with a pint or two of fluid, and any catarrh which may co-exist is at the same time benefited. A rubber tube attached to a funnel is introduced into the stomach; the stomach is filled by raising the funnel above the level of the mouth and pouring in water; it is emptied again by depressing the funnel and inverting it into a suitable vessel, or better by means of a Senoran's evacuator. The water used for washing it is either pure, or contains bicarbonate of sodium (1 or 2 per cent.) or salicylic acid (1 per cent.). The washing should be done once daily, half an hour before the largest meal.

Food should be given in small quantities and at short intervals on the principles already laid down (*see* p. 413). Starchy and saccharine foods should be restricted, or even prohibited, to prevent fermentation.

If the obstruction is organic (cicatrised ulcer or carcinoma), an attempt should be made by surgical means to widen it or remove it, or a gastro-jejunostomy should be performed.

## HOUR-GLASS CONTRACTION OF THE STOMACH

This condition is practically always due to cicatrisation of a chronic gastric ulcer, although occasionally perigastric adhesions may fasten the stomach to the liver, and produce a similar appearance by kinking. Carcinoma may produce narrowing of the stomach, and in gastroptosis the stomach is divided into an upper and lower compartment with a narrow neck intervening; but there is no difficulty in differentiating these conditions from true hour-glass contraction.

The symptoms are those of the associated gastric ulcer; but when the latter has completely healed and the constriction is considerable, the patient will complain of being only able to take small quantities of food at a time, larger

quantities being immediately regurgitated, so that œsophageal obstruction may be suspected.

**Diagnosis.**—This is carried out by means of X-rays after an opaque meal. The stomach is divided into two compartments connected together by a narrow neck, which joins the upper compartment on the right side and not at its most dependent part. This is quite characteristic, and serves to distinguish the condition from the sagging of the stomach in gastroptosis. If the ulcer is not completely healed, there may be some spasm of the musculature in addition to the cicatrisation; this makes the obstruction still greater, so that only the narrowest streak of barium joins the two compartments. When spasm is present it may relax somewhat on cautious manipulation or after a dose of belladonna. An active ulcer may or may not be present, and is shown as a projection to the right of the neck, which is tender on pressure (*see* Plate XXV.).

In carcinoma the growth will be seen projecting into the stomach, as a clear area (*see* Plate XXVI.); but if it arises from the ulcer, the X-ray appearance may be the same as in hour-glass contraction.

**Treatment.**—The only efficient treatment is surgical; the constriction can be widened, or the proximal cavity can be united to the distal cavity, or to the jejunum.

## CONGENITAL HYPERTROPHIC STENOSIS

This is a form of pyloric obstruction of which the symptoms appear as a rule from a few days to six or seven weeks after birth, most commonly during the first four weeks of life (**29**). The disease is about five times as common in boys as girls. The symptoms consist of vomiting and constipation owing to the smallness of the amount of food that reaches the intestine, and emaciation; and the vomited matters are often abundant and thrown up with much force. At first they consist of food, but later there is mucus, and altered blood, as gastritis becomes established; but very occasionally bile. The peristaltic movement from left to right, so characteristic of pyloric stenosis, will be seen; and in nearly all cases—a careful and prolonged examination may be necessary during and after a feed—a tumour or thickening, $\frac{1}{2}$ or $\frac{3}{4}$ inch in diameter, will be found to the right of the middle line, a little below the costal margin, varying in hardness from time to time with the contraction of the pylorus, and so distinguishable from scybala. Visible peristalsis may occur apart from organic disease, so that the presence of a tumour is necessary for certain diagnosis. X-rays may also be helpful.

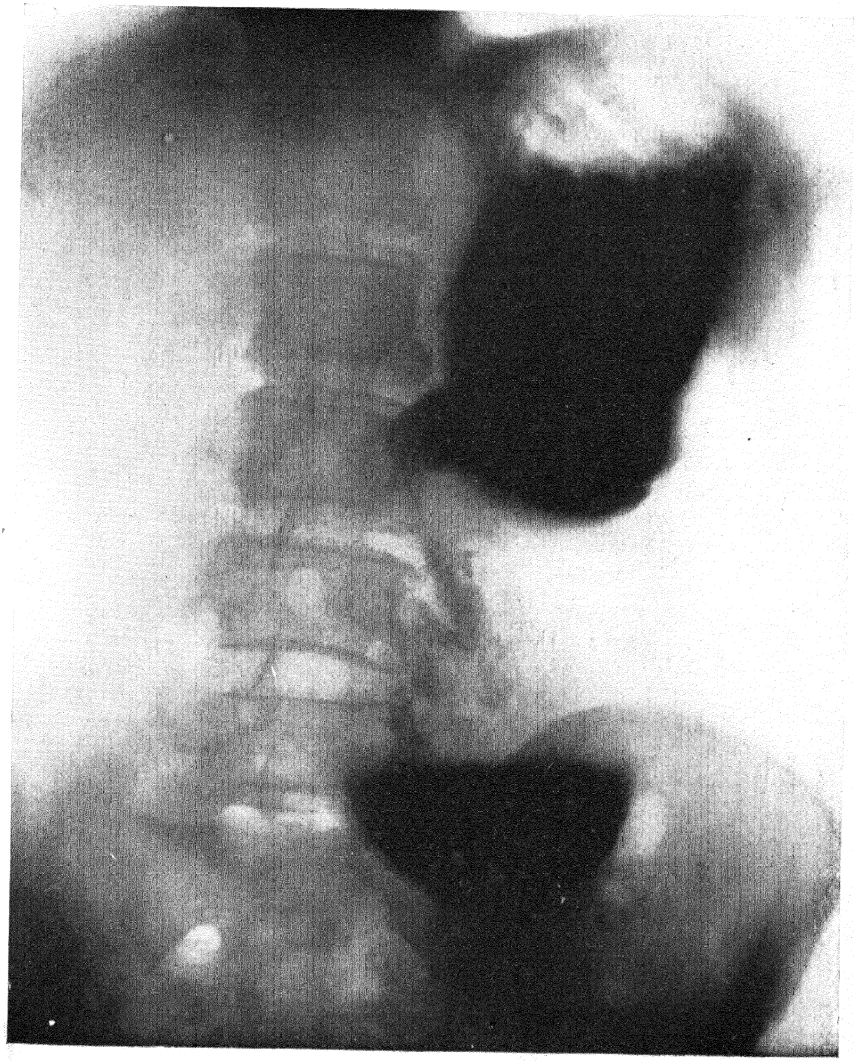
The thickening is a hyperplasia of the muscular fibres of the pylorus, chiefly of the circular coat, and is probably developed during fœtal life; within the thickened mass the mucous membrane is thrown into folds.

**Treatment.**—Medical treatment consists in giving very small quantities (from a teaspoonful to two or three tablespoonfuls) of whey, milk and water, or peptonised milk at intervals of half an hour or longer. In these cases lavage, once or twice daily, has also been of great service. If, after some days of this treatment, the child continues to vomit or to lose weight, Rammstedt's operation should be performed; this consists in cutting through the tumour longitudinally from the peritoneal surface as far as the mucous membrane, so that the obstruction is relieved. The operation has sometimes been followed with diarrhœa with fatty stools (**30**), which suggests that biliary and possibly pancreatic insufficiency play a part in the disease.

## CARCINOMA OF THE STOMACH

**Ætiology.**—Carcinoma of the stomach is rarely seen before the age of thirty, and 60 per cent. of the cases occur between forty and sixty; it is rather more common in males than in females; heredity is not a prominent feature

PLATE XXV.



Radiogram showing "Hour-glass Stomach." The upper compartment, which is filled with barium, has a rounded lower border and the opening into the lower compartment is situated near the lesser curvature and is above the floor of the upper compartment. This is an indication that the hour-glass is due to cicatricial contraction, although there is nearly always some spasm in addition. The ulcer crater is situated just at the opening on the lesser curvature. This is shown by the right-hand edge of the opening being concave towards the stomach. Barium is seen trickling slowly down into the lower compartment, where it settles on the bottom of the stomach and has a horizontal upper border. The stomach is dropped, since the lower border is a long way below the iliac crest. The duodenal cap is not seen, as no barium has yet left the stomach. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 430.

of carcinoma of the stomach. It is equally frequent among the rich and the poor, and is not related to any particular occupation. Much controversy has arisen as to whether carcinoma arises commonly from gastric ulcer, since carcinoma of the duodenum is very rare in spite of the frequency of duodenal ulcer. A short series of gastric cases carefully studied suggests that a carcinomatous ulcer of the stomach may start *sui generis* (**28**). Chronic ulcers removed at operation have shown epithelial proliferation which have suggested carcinoma to some, but others have pointed out that this merely indicates chronic inflammatory reaction. To sum up, gastric carcinoma is probably a rare sequela of gastric ulcer.

**Pathological Anatomy.**—Carcinoma affects all parts of the stomach, but in the majority of cases the pylorus is involved, and the disease extends thence to the adjacent parts of the organs, especially along the lesser curvature. If it affects the cardiac end, the œsophagus is generally also invaded. Sometimes the wall of the stomach is uniformly infiltrated and thickened, and the organ, as a whole, is contracted to a small size (*leather-bottle stomach*). With few exceptions, carcinoma of the stomach is in the form of spheroidal carcinoma or cylindrical carcinoma; and the former is much more common. Either variety may be scirrhous from excess of fibrous tissue, or medullary from deficiency of it; and colloid degeneration may take place in either, but is more common in the spheroidal variety. The scirrhous change is the most common. Sarcoma is rare.

Carcinoma commonly begins as an overgrowth of the epithelial cells of the glands of the mucous membrane; the growths project into the submucous tissue, proliferate further, and gradually involve all the coats. In carcinoma of the pylorus the whole wall of the stomach at this spot is thickened and projects internally so as to narrow considerably the passage from the stomach into the duodenum; this may admit with difficulty the little finger or a large catheter. The projection terminates abruptly towards the duodenum, more gradually towards the stomach. The thickening mostly affects the submucous layer, but also the muscular coat; and the bands of muscular fibre are separated from one another by the new growth. Subsequently the subserous layer is involved, and deposits of carcinoma may occur in the adjacent peritoneal surface; in later stages it often ulcerates upon the inner surface. The adjacent mucous membrane may show nodular growths or villous processes.

Important changes occur in the stomach and adjacent parts as a result of carcinoma, which are in many ways similar to those following simple ulcer. Thus the ulcerative process may erode vessels and lead to hæmorrhage; this is much less often profuse than it is in simple ulcer. Dilatation of the stomach is very frequent as a result of the narrowing or *stenosis* of the pylorus, which the growth of carcinoma necessitates; but in a certain number of cases the stomach is actually smaller, and this generally when carcinoma affects the whole of the organ, as already shown. *Adhesion* of the stomach to other organs commonly takes place, as the growth reaches the peritoneal surface, and invasion of the organ with carcinoma may follow. The liver and pancreas are thus frequently attacked, occasionally the spleen or colon. In the last case a *gastro-colic fistula* may result. When the carcinoma is in front the abdominal wall may become adherent, or, in the absence of adhesion, perforation into the peritoneum may take place; but this is much less likely than in simple ulcer. More often subacute or chronic *peritonitis* takes place without perforation, either spreading from the original lesion or following a general growth of carcinoma in the peritoneum. Carcinoma of the cardiac extremity frequently invades and obstructs the œsophagus.

Secondary deposits occur in various organs, in the peritoneum, as just stated, in the liver, pancreas, lungs, and adjacent lymph glands. These are the mesenteric, retroperitoneal, and portal glands; but, as occurs also in œsophageal carcinoma, the cervical lymph glands are sometimes affected quite early. Other

distant parts of the body are affected more rarely. After death from carcinoma the heart is commonly in the condition known as brown atrophy.

**Symptoms.**—Clinically there are two groups of cases according to whether the pyloric function is disturbed with undue retention of gastric contents or not.

(1) In the earlier stages of this group the symptoms are chiefly those of a painful dyspepsia. There are discomfort, fulness, weight or pain after food, and flatulence. The *pain* may be at the epigastrium, or in the position of heartburn; small quantities of food may be regurgitated. After a time *vomiting* takes place at first only at long intervals, then weekly, or two or three times a week; it is likely to occur earlier in the case, when the carcinoma is situate at the pylorus than if it is remote from that point.

Pain then becomes a more prominent symptom, and though, like the other symptoms, at first related more or less closely to the ingestion of food, it soon becomes more constant, or arises independently of a meal. It commonly radiates from the epigastric region, where it is, in later stages, most intense, and is occasionally felt between the shoulders, or in the lumbar region. It is often stabbing and lancinating, but may be boring, burning, gnawing, or tearing.

The vomited matters consist of food in different stages of digestion, mixed with more or less mucus or streaks of blood. Often the blood mixed with the vomit has the appearance of coffee grounds (*see* p. 406). Abundant hæmorrhages are much less common than in ulcers; but a large vessel, such as the splenic artery, is sometimes eroded, and profuse and fatal bleeding results.

The condition of the abdomen varies. As a result of the small quantity of food that passes the pylorus, it is frequently empty, but presents in many cases the prominence in the upper or left portion which is due to dilatation of the stomach from pyloric stenosis, and visible peristalsis may be seen.

Indications of severe constitutional disturbance set in comparatively early in this type of carcinoma of the stomach. The appetite diminishes, and the patient loses flesh, strength, and colour. Loss of weight may be, indeed, the earliest sign; and in advanced conditions *emaciation* and *anæmia* are extreme. The tongue is often clean, but in the last days may be dry and covered with sordes or deposits of thrush. The bowels are mostly constipated. Melæna is rare as an early sign, but *occult blood* (*see* p. 408) is common.

In a few cases the amount of fibroid change in the pylorus is so great that the lesion has been regarded as purely fibrous, and has been called *hypertrophic stenosis*. But the invasion of the lymphatics by carcinoma shows the nature of the thickening. However, the progress of these cases is slow, and the patients for years suffer only from the resulting dilatation.

(2) When the function of the pylorus is not interfered with, the growth is usually, but not always, in the body of the stomach. There may be no symptoms until the growth is far advanced. Pain may then be felt in the back from infiltration of neighbouring structures. But such a growth may bleed continuously, the altered blood being passed in the motions, and the resulting anæmia may arouse suspicion of pernicious anæmia, when the tumour is impalpable. At the same time there may be progressive cachexia.

In general, it may be said that in the majority of cases of carcinoma a *tumour* is discovered at some time or other, but rarely in the first three or four months, and according to some statistics in only 33 per cent. of the cases within the first six months. The position of course varies with the part of the stomach affected. A pyloric tumour is commonly situated in the middle line, or a little to the right, midway between the xiphisternum and the umbilicus, but it may be close to the umbilicus, or more to the right of the middle line, indeed, almost anywhere in the triangle formed by the right costal margin, the middle line, and a horizontal line running through the umbilicus. When the stomach is much dilated the tumour is even below the umbilicus. It varies in size from that of a walnut to that of a small orange, is generally very hard, sometimes globular, but often

somewhat square, and mostly irregularly nodular. It is at first freely movable, and descends on inspiration, but in later stages it may contract adhesions and become more fixed. It frequently receives an impulse from the underlying aorta. On percussion it is dull or imperfectly resonant; handling it causes pain, which may last for some time afterwards.

The X-rays show characteristic appearances. There is comparatively early an interruption of the shadow due to a bismuth meal by a more or less extensive clear area, which encroaches, as it were, upon the shadow by two or more curved or scalloped outlines; this represents the fungating growth projecting into the lumen of the stomach (*see* Plate XXVI.). It is tender on pressure, and is constant in shape, taking no part in the peristaltic movements which may be seen elsewhere in the stomach. Pyloric obstruction may also be observed. In the case of leather-bottle stomach, the food is seen to fall rapidly through a comparatively narrow tube straight into the duodenum without any peristalsis being visible at all.

Various complications may arise towards the end. Ascites may follow the spread of carcinoma to the peritoneum, or the implication of the liver and portal vein. Occasionally perforation into the peritoneal cavity takes place, and is followed by peritonitis; but the symptoms of this event are often obscure or not distinctive. Carcinoma of the retroperitoneal glands causes œdema of the feet, or the same is brought about by thrombosis of the large veins.

Gastro-colic fistula, which is mostly the result of carcinoma spreading from the stomach to the colon, is marked either by undigested contents of the stomach passing directly into the colon, and hence *per rectum*, or by fæcal vomiting, the contents of the colon passing into the stomach and hence being vomited.

Death commonly takes place from exhaustion as a result of continued pain, vomiting, and deficient assimilation of nutriment, or of a rapid spread of secondary growths in the liver with continued pyrexia. A profuse hæmorrhage or peritonitis, bronchitis, or pneumonia may terminate the scene.

**Duration.**—The illness commonly lasts from six months to two years; two-thirds of the cases last less than eighteen months, and a very small proportion more than two years.

**Diagnosis.**—Carcinoma is usually distinguished from the majority of the diseases of the stomach by the presence of a tumour. This is, however, not generally felt in the earliest stages, and may be imperceptible later from its small size; from the pylorus lying under the liver; from its being concealed by much distension of the bowels, or by ascites; or, finally, because the tumour is situate on the posterior wall of the stomach. Occasionally in such cases the diagnosis has been determined by the discovery of a hard enlarged cervical gland. If no tumour is discoverable, carcinoma may be confounded with pernicious anæmia, as already indicated, or chronic dyspepsia, including gastritis or peptic ulcer, or purely neuralgic pains may be thought to be due to carcinoma. Carcinoma arises in most cases independently of previous gastric troubles, and the pain becomes continuous. The tongue may remain clean, but the patient soon has no appetite for food; and he loses flesh and becomes anæmic. There may be moderate pyrexia.

In dyspepsia and gastric ulcer judicious treatment materially or entirely relieves the symptoms, which in carcinoma are but little, or only for a time, influenced. A cicatrised ulcer may lead to troublesome pains, but there is a long history, and the strength and health are fairly maintained. Dilatation of the stomach occurs very late in ulcer, but much more rapidly in carcinoma. Exceptionally the tumour of carcinoma may be simulated by the matting and adhesion of parts caused by ulcer (*see* p. 422). The age to which carcinoma is almost strictly limited and the short duration of the disease are also important elements in its diagnosis.

X-rays after an opaque meal are of very great value for diagnostic purposes, the shape, position and motility of the stomach being noted, and the relation of

the organ to any palpable lump (*see* Plate XXVI.). The length of time the meal remains in the stomach should be ascertained. Pyloric stenosis will be indicated if the stomach is not empty six or eight hours later.

Examination of the stomach contents in the early morning should be undertaken, after the patient has taken two teaspoonfuls of charcoal in milk the night before (**31**). The presence of visible blood, either fresh or altered, is suggestive of carcinoma, especially if it is found on more than one occasion, while the presence of charcoal indicates delay in emptying. A persistent oozing of blood is characteristic of carcinoma, while an occasional profuse hæmorrhage is more probably the result of ulcer. Coffee-ground vomit is seen in both. After washing out the stomach a test meal may then be given. The presence of lactic acid in the test meal suggests carcinoma of the stomach, and in the latter there is great diminution in the "active HCl" and in the amount of "free HCl"; but this also occurs in some normal people as well as in the group known as "achylia gastrica," and in pernicious anæmia and acne rosacea. The "mineral chloride" is increased, but this may also be brought about by regurgitation through the duodenum.

**Prognosis.**—This is very unfavourable, as death is inevitable unless the growth and all infected glands can be entirely removed.

**Treatment.**—In suspected early cases laparotomy should be advised, as removal of the growth is almost the only possible method of cure. It is usually too late to do this by the time a lump can be felt, owing to spreading of the growth and involvement of the lymphatic glands. Gastro-jejunostomy may be performed as a palliative measure. Deep X-ray therapy might be tried.

If the position or extent of the lesion renders operation inadvisable, a palliative course of treatment must be adopted. The food must be of a light, nutritious kind, and must be given in the natural way—by the mouth. Milk, milk and soda water, or milk peptonised with liquor pancreaticus, may be given in some cases; in others it may be thickened with farinaceous food or eggs; or more solid nutriment may be borne, such as fish, chicken, or oysters. Wine of a light kind may be allowed; champagne is often useful on account of its effervescence, and in late stages brandy may be necessary. The symptoms of pain and vomiting must be dealt with much as in ulcer and chronic dyspepsia. Opium or morphia is again constantly required for the pain, or locally a small blister or chloroform liniment will sometimes give relief. Constipation must be met by enemas, or by saline purgatives, or by pills of aloes and iron.

If dilatation is a prominent symptom, and large quantities of food are vomited every few days, relief may be temporarily afforded by washing out the stomach daily (*see* p. 429), or the patient may be instructed to pass a tube when pain is felt, relief sometimes being obtained by this simple measure alone.

### BENIGN TUMOURS OF THE STOMACH

These include *adenoma*, *myoma*, *accessory pancreas*, *lipoma*, *fibro-lipoma*, *lymph-adenoma*, and *cysts*. They are quite infrequent. The first three are the least uncommon, and they occasionally cause symptoms by obstructing the pylorus. In other cases the symptoms will depend on the size and position of the growth.

# DISEASES OF THE INTESTINE

## CONSTIPATION

The healthy action of the bowels depends on a sufficient supply of food, the waste of which forms the material for the fæces; a natural secretion of intestinal juices; and an intestinal muscular system readily stimulated and strong enough

Radiogram of the Stomach showing Carcinoma of the Pyloric Part. The shadow of the barium meal is broken into by the growth and its outline is very irregular and has a scalloped appearance. Some food is seen in the second part of the duodenum. The stomach is much dropped, the greater curvature lying a long way below the iliac crest. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 434.

to force on the fæces from point to point. This action, however, varies in different individuals, who may still all be healthy. Most persons have an action of the bowels once a day, but others twice a day, and some only every other day.

Constipation is the retention of fæces for longer than the normal period of twenty-four hours, or in some persons two days; and is due to delay (1) in the general movement along the large intestine (*colonic stasis*), or (2) in the evacuation of the pelvic colon and rectum (*dyschezia*), or in both the processes together. If colonic stasis is severe, there will also be delay in the passage of the intestinal contents through the ileo-cæcal valve (*ileal stasis*). Ileal stasis may occur without colonic stasis, especially in cases of sub-acute appendicitis (*controlling appendix*).

1. The general movement of fæces along the intestinal canal is purely involuntary, and is dependent upon the adequate muscular power of the intestinal wall, properly stimulated to action by suitable food. A weak musculature may be a hereditary failing, or it may supervene in later life as a cause of senile constipation. A temporary weakness occurs in fevers and acute illnesses, and may result from anæmia, chlorosis, rickets, and diseases involving nervous depression, such as melancholia, neurasthenia, etc. It is often present in cases of cerebral tumour. Locally it may be due to flatulent distension, and to catarrh of the mucous membrane.

The stimulus to the bowel comes chiefly from the food; and this may be insufficient in quantity, or too dry, or deficient in mechanical stimuli, of which the cellulose of vegetable substances is the most important. It appears also that in some individuals unusual powers of digestion and absorption on the part of the intestines may leave so little residue that evacuations must be infrequent; a condition that is known as "greedy colon." In many gastric disorders, especially where vomiting is frequent, constipation occurs. Further, the reflex action of the bowel may be directly inhibited by painful local affections of an inflammatory or traumatic nature in the pelvis or abdomen.

Colonic stasis can only be diagnosed by examining the intestines after an opaque meal has been given. Under ordinary circumstances, at the end of four hours the stomach is empty and the food is collecting at the end of the ileum. The taking of a second meal causes special activity at the end of the ileum and in the colon, so that the intestinal contents rapidly moved forward (*gastro-ileal and gastro-colic reflexes*) (**32**). The opaque meal has usually left the ileum four hours after it has completely disappeared from the stomach; but ileal stasis can only be diagnosed with safety if no trace of the meal has entered the cæcum six hours after taking it, or if the bulk of the meal is still at the end of the ileum nine hours after the meal is taken if the stomach is known to have emptied itself in three or four hours. Normally the opaque meal reaches the rectum in twenty-four hours. Colonic stasis will exist if at the end of this time it is still entirely in the cæcum and ascending colon, or if the splenic flexure is first reached in twenty-four hours, but the opaque meal still remains in the transverse colon forty-eight hours after the meal.

2. The desire to defæcate is caused by the entry into the rectum of the fæces which have accumulated in the pelvic colon during twenty-four hours, and this is brought about reflexly by the stimulus of eating breakfast, or of getting up, or of some other daily recurring function.

The defect in the final process of defæcation, that is, in the passage of fæces into the rectum, and the final evacuation, is a common cause of so-called habitual constipation. It is called by Hurst *dyschezia* (χέζω, I ease myself). The performance of this function is dependent upon a stimulus conveyed from the rectum, and the response upon the part of the pelvic colon. In well-regulated persons the stimulus arises at a given time every day, and if allowed to operate an evacuation is the result. If the stimulus is disregarded and the desire to go to stool is repressed, the reflex is likely to be less active on a subsequent occasion,

and in course of time the stimulus may fail to be felt. Thus the repression of the desire and the disregard of the sensation are common causes of constipation. Want of time in the case of persons hurrying to business, false modesty in large houses, or in girls' schools, inadequate supply of accommodation in large establishments, mere laziness in many people, contribute to this, and lead to a postponement of the process until the regular habit is entirely given up, the fæces are retained for two or three days or even a week, and then evacuation can only be secured by the use of aperient drugs or an enema.

Another cause of dyschezia is weakness of the voluntary muscles which compress the abdominal contents, and thus assist in the passage of fæces from the colon into the rectum, and from the rectum through the anal passage. These are the abdominal expiratory muscles, the diaphragm, and the levator ani and other muscles of the pelvic floor.

Dyschezia can be diagnosed if the greater part of the opaque meal has reached the pelvic colon and rectum twenty-four hours after it has been taken, and if, in spite of this, there is no desire to defæcate.

In any part of the large intestine delay may be caused by mechanical obstacles to the passage of the fæces, such as compression or kinking of the colon, hard fæcal masses, stricture by growth, a retroverted uterus, spasm of the sphincter ani, and spasm of the colon, a condition described as *enterospasm*. Some of these conditions in a higher degree lead to complete obstruction.

**Symptoms.**—If left to themselves the bowels only act at intervals of two, three, four, or more days; the colon or rectum becomes loaded with hard, round masses of fæcal matter (*scybala*), generally rather pale, which are welded together into masses. The desire to go to stool perhaps at first only results in ineffectual straining efforts; but finally some scybala are passed, and the same may be repeated two or three times within a few hours, till the lower bowel is emptied. After this the bowel is inactive for another period of several days. During the retention the patient may suffer various inconveniences. Locally there may be a sense of fulness in the perineum, or pruritus ani; and the hæmorrhoidal veins may swell, and possibly hæmorrhoids may be caused. Sometimes there is pain down the thigh from pressure of the fæcal masses on the nerves in the pelvis; moderate distension of the abdomen often occurs, with perhaps flatulence and eructations; the tongue is often furred, whitish or dirty brown, and the breath may be foul. Some patients feel languid, confused, wanting in vigour or freshness, and have actual headache, or even a great deal of mental depression. These and many other symptoms are attributed by some to the retarded progress of the bowel contents or *intestinal stasis* (*see* p. 438). But it must be noted that often the more habitual the constipation, the less is the general disturbance; and many are not conscious of having anything the matter with them though their last evacuation was many days before.

Where the fæces are retained in the rectum the latter becomes enormously dilated to accommodate them. The scybalous condition of the fæces is explained by their retention in the colon, during which there is time for the absorption of most of the liquid contained in them. Even when the rectum is distended with scybalous masses, some fæcal fluid may escape from the anus, or a secretion of mucus may be excited, and the discharge of these liquids may stimulate a diarrhœa. A more extensive catarrhal colitis, and stercoral ulcers, may also result from constipation.

The accumulation of fæcal matter in the pelvic colon may be such that it forms a large tumour in the lower part of the abdomen, of which the distinguishing feature is the fact that it can be indented by firm pressure with the finger.

**Treatment.**—In general it may be said that colonic stasis requires treatment by means of diet, and in severer degrees by massage, abdominal exercises and drugs, and dyschezia by persuasion and exercises, and in severer degrees by

graduated enemas. It must be remembered that some patients do well with an evacuation every second or third day.

The patient should make a regular and not a hurried daily visit to the closet whether he feels any desire or not at the time; and this should be continued as a habit for the rest of life, but it may take months before its good effects come into full operation. In dyschezia the squatting position should be adopted.

The diet should be modified so as to include a sufficiency of vegetables, fruit, fresh or preserved, or salad with salad oil; brown bread, wholemeal bread, or oatmeal porridge sometimes supplies the desired stimulus to the bowel. The diet also should be liquid enough, and with some a daily evacuation is ensured by drinking a tumblerful of cold water or eating an apple before breakfast. A teaspoonful or two of the non-absorbable powdered agar-agar added to porridge, potatoes or stewed fruit is valuable in cases of greedy colon.

To those of sedentary habits walking exercise, fencing, horse-riding, or driving is often of benefit, or the abdominal muscles may be specially exercised by Swedish exercises and massage carried out along the line of the colon as indicated by X-rays after an opaque meal. The pelvic floor in women may be exercised by getting the patient to draw in the anus thirty times morning and evening, as if to prevent the escape of flatus.

But with all this it may be still necessary to have recourse to drugs, and a careful selection is requisite. As a rule, very active or drastic purgatives must be avoided; they produce abundant liquid motions, from the effect of which the intestinal muscle is completely exhausted, and consequently no further evacuation takes place for days afterwards. But it has already been shown that constipation depends on deficiency of peristaltic action, and hence over-stimulation and exhaustion are especially to be avoided. From this point of view much advantage is gained by combining with the ordinary laxatives such drugs as have a tonic effect upon the intestinal muscle. These are especially nux vomica and iron. Senna pods, soaked some hours in cold water and drunk at night, are a valuable laxative. Cascara sagrada may be given every night in doses of 30 or 40 mimms of the liquid extract combined with syrup of ginger, or 2 or 3 grains of the solid extract in pill. Pure liquid paraffin may be given in doses of ½ ounce to 1 ounce once or twice daily according to the necessities of the case. An efficient combination is that of aloin with extract of nux vomica, 1 grain or 1½ grains of the former with ¼ or ½ grain of the latter, given in the morning before breakfast; ¼ grain of extract of belladonna or ½ grain of ipecacuanha is sometimes usefully added. Sulphate of iron (1 grain) with aloin and nux vomica is also very useful. If a daily pill is insufficient, two or even three pills may be taken; but in either case the essence of the treatment is that active purgation should be avoided, and directly this seems likely to be produced the three pills each day must be reduced to two, or the two to one; and ultimately the bowels will act without any assistance whatever. In some acute abdominal conditions and after abdominal operations eserine salicylate or pituitrin may be injected subcutaneously. The natural saline waters, when procurable, are of value, such as Rubinat, Püllna, Hunyàdi Janos (containing the sulphates of magnesium and sodium), and Carlsbad (mainly sulphate of sodium); the patient may take from a wineglassful to half a tumblerful before breakfast. Carlsbad salts extracted from the water of the different springs, or sulphate of sodium itself, may also be given—a teaspoonful is dissolved in half a tumblerful of hot water, and drunk before breakfast. Enemas of cold water are often necessary at the beginning of treatment when there is accumulation of fæces in the bowel, and particularly in the rectum, which is below the point of direct operation of aperients. When a large accumulation of fæces has taken place, the enema may have to be assisted by the use of the finger; for some days after this the enema may be used to supply the stimulus until a more natural method is established. In dyschezia not curable by persuasion and exercises graduated glycerine enemas should be

tried, beginning with 1 ounce and gradually replacing more and more of the glycerine by water on successive days.

## ALIMENTARY TOXÆMIA

By alimentary toxæmia is meant the absorption into the blood of toxins or poisons derived from the alimentary canal. The belief has been gaining ground that a great many symptoms, pathological conditions, and even definite diseases are due to this toxæmia. But many difficulties will have to be overcome before this theory can be put on a thoroughly scientific basis. At present one cannot say much more than that treatment based upon the hypothesis has often been successful. The steps between the cause and the result are not always easy to trace.

Firstly, the origins of the toxins may be organisms introduced from without, as in the case of oral sepsis already referred to (*see* p. 392), in which disease may be produced either by streptococci entering the blood stream or by the constant swallowing of these micro-organisms which come from infected dental sockets. These may settle down in various parts of the alimentary tract, and produce infections there Recently some cases of rheumatoid arthritis and osteo-arthritis have been ascribed to this cause (Mutch). Secondly, food may actually contain poisons, or an excessive proportion of proteins which may decompose, or carbohydrates which may undergo fermentation; and, thirdly, food, or rather fæcal matter, may be retained, as the result of habitual constipation, sufficiently long to undergo bacterial or chemical changes with the production of toxins, or poisonous chemical substances (*enterostasis*).

The complicated processes of digestion which take place in the alimentary canal from stomach to rectum inclusive, and the chances of delay and disturbance offered by such an elongated cavity, seem to provide abundant opportunity for the formation of chemical poisons, or toxins, and their passage into the circulation. But at the outset it is possible that it is not only a question of the occurrence of new poisons or toxins, but of the breaking down of the mechanisms by which normally poisons in the alimentary canal or elsewhere are prevented from reaching the blood. These are the digestive secretions, the mucous membranes and their mucus, the antitoxic action of the liver, and possibly the action of the thyroid gland.

But it appears to be uncertain whether at all, or in what circumstances, the bacteria usually found in the intestine do any material harm; and with regard to chemical substances there is still much to be learned, some attaching importance to the formation of indol, skatol, and phenol, and of ethereal sulphates in excess, while others, as Mellanby, see more danger in the amines produced by the splitting off of $CO_2$ from the proteolytic amino-acids by intestinal bacteria.

Cases of food-poisoning, in which decomposing food containing specific bacilli is ingested and symptoms result therefrom, need no comment.

Gout and allied conditions are attributed to the prolonged use of food containing a high proportion of proteins, but it is still open to question whether this is because the proteins decompose readily; whether micro-organisms have any relation to them; or whether bacterial toxins, or other chemical compounds, such as purins, cause the mischief. Similarly an excess of carbohydrates may, in infants, cause a toxic condition in which fever, sickness, diarrhœa with acid green stools, and abdominal distension are present. Some cutaneous eruptions appear to depend upon gastro-intestinal irregularities—for instance, acute urticaria after shell-fish, whether this is due to direct poisoning or is an instance of anaphylaxis, as some believe.

Of late interest has centred around the third factor, namely, chronic constipation or enterostasis, causing fæcal retention, to which Sir Arbuthnot Lane has

attributed so many evil results. His contention is that from improper feeding in early life and from the maintenance of the erect posture the intestines tend to fall, that peritoneal adhesions are formed in various parts to support them, that subsequently with continued retention, and excessive weight of the retained fæcal matter, kinks are formed at the end of the ileum and in the duodenum, which increase retention and lead to dilatation of the parts above, while there is a general proptosis of all the organs. From the fæcal matter thus retained in consequence of this intestinal stasis toxins are formed which act prejudicially both locally and generally. Among the local results are said to be the following: appendicitis, duodenal ulcer, spasm of the pylorus, gastric dilatation, gastric ulcer, gastric carcinoma, and pyorrhœa alveolaris.

In the general condition of the patient the toxæmia caused by this intestinal stasis is recognised by him in every tissue of the body: cold hands, defective circulation, dusky hue of face, pigmentation of the face and body, dull sclerotic and œdema of the conjunctiva, mental dulness, depression, headache, insomnia, incapacity for physical or mental exertion, neuralgia. In the female especially, he states, the effects are pernicious: loss of fat occurs, the kidneys fall, the uterus is retroflexed, cystic disease and carcinoma of the breast occur, and infection of the genito-urinary tract readily takes place.

It must, however, be admitted that other observers do not believe that chronic intestinal stasis does produce such evil results; they believe that the symptoms complained of by such patients are really due to neurasthenia, which has developed from the habit of introspection, and in particular of constantly paying too much attention to the movements of their bowels.

Prof. Arthur Keith holds different views from those of Sir Arbuthnot Lane with regard to the origin of stasis in the bowel. As the result of observations with X-rays on the movements of the stomach and intestine, and of microscopical researches on Auerbach's plexus and associated structures in the bowel, he puts forward the theory that the peristaltic movements start from separate points in the intestinal canal, which are, in relation to such movements, divisible into the following sections:—duodenal, jejuno-ileal, proximal colic, and distal colic. In each of these sections peristalsis has been observed to be more active at the upper end, and less active as it proceeds downwards, so that at the lower end a condition allied to sphincter action is assumed. Keith recognises in the upper end of each of these sections neuro-muscular tissue analogous to the sino-auricular node of the heart (*see* p. 284), which he therefore calls *nodal tissue;* and he looks upon the mesenteric (Auerbach's) plexus as the analogue of the auriculo-ventricular bundle. He thinks that an alteration in the orderly transmission of impulses along either of the sections from its upper nodal tissue downwards will account for the enterostasis in the section concerned without the necessity for the adhesions, bands and kinks described by Lane. He is disposed to believe that similar neuro-muscular tissue in the œsophagus and stomach respectively may function in the same way.

Lane's original remedy was the removal of the colon, but this is seldom carried out now. An alternative operation, viz. the anastomosis of the lower end of the ileum with the pelvic colon, is also now very little used. A very thorough treatment on medical lines by diet, purgatives, and other measures, as described under "Constipation," should be carried out.

## DIARRHŒA

By diarrhœa is meant the passage of motions more often and of looser consistence than is normal. It is the result of excessive peristaltic action, and of excessive intestinal secretion or deficient absorption. Its causes may be classified as follows: (1) *Gastrogenous.* There is often hypochlorhydria, probably always due to regurgitation from the duodenum (*see* p. 419), and the administration of 20 or

30 minims acid. hydrochlor. dil. three times a day may check it. Diarrhœa may follow gastro-jejunostomy. (2) Infection of the *small intestine*, such as occurs in catarrhal enteritis, typhoid fever and tuberculosis, may be responsible. In tuberculosis the looseness and undigested character of the motions may be due to deficient absorption (*see* p. 515). (3) In the *colon and rectum* the causes of diarrhœa are numerous, including the dysenteries, ulcerative colitis, acute colitis often associated with septicæmias, *e.g.* pneumococcal, the colitis of chronic nephritis, malignant disease, the irritation which follows the abuse of purgatives and enemas. (4) *Reflex diarrhœas* result from cholecystitis and appendicitis. (5) The *nervous system* may cause diarrhœa through emotion or hysteria, and there is a rare form of diarrhœa due to tabes. (6) There is a *mixed* group, which includes the diarrhœa of Grave's disease and excess of insulin.

It must be remembered that the frequent discharge of liquids in small quantities does not of itself show that the canal of the bowel is free. Thus intussusception which partly obstructs the gut is accompanied by the passage of mucus and blood; fæcal fluid mixed with mucus may find a way past very large masses of impacted fæces; and, lastly, even a distinctly contracted intestine may allow some of the thin liquid which collects above the obstruction to pass through and simulate a diarrhœa. These are called *spurious diarrhœas*.

**Varieties.**—Diarrhœa has received different names according to the nature of the matters passed. Thus we have *choleraic* diarrhœa, in which the stools are profuse and watery, or like the rice-water stools of cholera; *dysenteric* diarrhœa, in which mucus is largely present, and perhaps blood; *lienteric* diarrhœa, where taking food into the stomach produces a motion: this is probably due to an increase in the normal gastro-colic reflex (**32**); and *bilious* diarrhœa, when the discharges are deeply stained with a brown or greenish-brown colour, which is due not so much to any increase in the quantity of bile secreted as to the fact that the contents of the duodenum and jejunum stained with bile have been hurried through the alimentary canal without giving time for the natural reabsorption of the altered bile pigment (urobilin). *Colliquative* diarrhœa is a term applied to the profuse, exhausting, and intractable discharges which occur in the last stages of phthisis. A *critical diarrhœa* is an old term for a diarrhœa that accompanies the termination of a pneumonia by crisis; it is probably due to an associated entero-colitis.

**Treatment.**—This must depend upon the cause, or the associated condition. In most cases not of a specific nature, the treatment described under Enteritis may be employed. Lienteric diarrhœa, which depends upon an exaggerated intestinal reflex, may be treated with full doses of potassium bromide.

## HÆMORRHAGE FROM THE BOWEL

The passage of blood *per rectum* has already been noticed as occurring in enteric fever, and in ulcer of the stomach and of the duodenum. It also results from other ulcerations, as dysentery and ulcerative colitis, from intussusception, from carcinoma of the sigmoid or rectum, from conditions of intense congestion, from embolism or thrombosis of mesenteric vessels, and from purpura and other conditions of blood disease. The way in which the blood is passed may give a clue as to the point whence it comes. In bleeding from gastric or duodenal ulcers the blood is considerably altered by the secretions, and forms a black, tarry, semi-liquid or treacly mass (*melæna*); in hæmorrhage from typhoid ulcers the blood is equally unmixed with fæces, but brighter red and more fluid than in the former case, from the action of the alkaline contents; the blood in dysentery is in streaks or small clots mixed up with mucus or pus, or thin fæcal matter, though from time to time small quantities of pure blood may be passed. Large quantities of blood may be lost from piles, or from an ulcer of the rectum. Here the bleeding is generally caused by the act of defæcation, the blood either streaking one side

of the solid fæcal mass, or coming more or less pure in drops or streams after the motion is evacuated. In scorbutic, purpuric, and hæmorrhagic conditions (scurvy, purpura hæmorrhagica, acute yellow atrophy of the liver, malignant variola) blood comes from the rectum more or less mixed with fæces, or pure, according to the part of the intestine yielding it, or the freedom with which it escapes. The diagnosis of small quantities of blood has already been considered (*see* p. 408). The **Treatment** of hæmorrhage is described with the various diseases which may cause it.

## INTESTINAL COLIC

The term *colic*, though obviously derived from the word *colon*, means a spasmodic abdominal pain of visceral origin. It is associated with violent contractions and relaxations of the muscle fibres of the alimentary canal which occur reflexly from some irritation and are necessary in order to force some resisting object along the tube. Colic may occur in the ureter (renal colic), the bile ducts (biliary or hepatic colic), or the intestines (intestinal colic).

**Ætiology.**—The most frequent cause of intestinal colic is irritating and unsuitable ingesta, such as pork, cheese, high game, shell-fish, ices, etc. In children, colic is a common result of indigestible food, or even simple excess. With these may be classed the more active purgatives. On the other hand, constipation is often associated with colic, and this is markedly so in the colic due to lead poisoning, whether acute or chronic (*see* Lead Poisoning). Some cases may perhaps be referred to a purely nervous source—for instance, the severe pain of gastric crisis in tabes dorsalis. Lastly, mechanical and acute inflammatory lesions of the bowel, such as strangulation and intussusception, lead to severe pains, which are partly or wholly due to muscular contraction. The term *colic* is, however, generally reserved for conditions in which there is no structural or inflammatory change.

**Symptoms.**—The important symptom is pain, which is situate about the umbilicus, but may move about other parts of the abdomen. This pain is often relieved by pressure, but sometimes there is tenderness. The abdomen is either drawn in, and the abdominal muscles are contracted, or the belly is distended from the presence of flatus. When flatus is present borborygmi are produced by its movements, as it is driven on by the varying intestinal spasm.

The pain may be so severe as to cause much collapse, with profuse clammy sweat and small feeble pulse. Sometimes there is vomiting; often there is constipation; on the other hand, some ingesta, which cause colic, set up active diarrhœa with brown watery stools, and mucus after a time. Here the colic is associated with a definite, though slight, enteritis. The more active purgatives also produce griping and "colicky" pains, which are commonly diminished after each evacuation.

**Diagnosis.**—Gastric and intestinal colic may be confounded with the pains of any acute inflammation in the abdomen, such as appendicitis, peritonitis, pancreatitis, cholecystitis, with the pain of acute strangulation of the intestine, and with hepatic or even renal colic; the special features of these several diseases must always be carefully considered. Lead colic is often mistaken for intestinal obstruction, when a glance at the gums would give the right clue. On the whole, it is the absence of the more positive indications of obstruction, peritonitis, or other acute inflammation, and of the facial signs of abdominal disease, the relation to unsuitable foods, or the fact that the patient has had similar attacks before, which will help most to make the case clear.

**Treatment.**—Obviously, cases of severe abdominal pain must be treated with much caution. If the pain is certainly due to irritating ingesta, relief generally follows the exhibition of purgatives, such as an ounce of castor oil with 15 minims of tr. opii, or ½ ounce of magnesium sulphate with ½ drachm of tr.

hyoscyami, or 5 grains of calomel; and a similar line of treatment is used in lead colic (*see* Lead Poisoning). A warm water or castor oil enema may also help, and hot fomentations or a hot-water bottle should be applied to the abdomen. Papaverine hydrochloride ½ to 1½ grain by mouth may also be tried as an antispasmodic. If there is any likelihood that appendicitis, peritonitis or obstruction may be present, purgatives should be avoided; and the question of treatment by operation must be considered.

## ENTERITIS

There are several conditions, affecting different parts of the alimentary canal, which may properly be termed enteritis, or inflammation of the intestine. For instance, the catarrhal process, of which some forms of diarrhœa are the result; tuberculous and typhoid ulcerations in the ileum; the ulcerative inflammation of the colon, known as dysentery; and the acute changes set up by intussusception and strangulations, are all, in fact, enteritis. But a large number of these have already received distinctive names; others are only secondary conditions, which produce few symptoms beyond those of the primary disorder; and in others, again, inflammation of the coats of the intestine involves a peritonitis, which throws the symptoms due to the mucous inflammation completely into the shade. Thus the number of cases which require separate description as enteritis is but a small one, though it is probable that a fair consideration of the pathological side of many of our intestinal cases, such as diarrhœa, would show that the name might be justly used more often.

The following forms of enteritis will be here described: *catarrhal enteritis, infantile enteritis, food poisoning, sprue, diphtheritic enteritis,* and *phlegmonous enteritis.*

### CATARRHAL ENTERITIS

*(Intestinal Catarrh)*

Anything which irritates the mucous membrane of the intestine may set up catarrh, such as unsuitable food, certain poisons, and purgative drugs. Catarrh is also ascribed sometimes to chill; but a much more potent factor in its production is excessive heat, and it is always more prevalent in the hot weather of summer and autumn than in the remaining part of the year. This frequency in the summer concerns people of all ages, but infants are especially attacked. In very hot weather every kind of food decomposes more readily, and milk quickly "turns"; and these processes are dependent upon micro-organisms. An important factor related to summer heat is the prevalence of flies, which may convey infection to both meat and milk not properly protected from them. Many kinds of organisms have been found in the intestines in health; those most constantly present are the *Bacillus lactis aerogenes,* the *B. coli communis,* and the *Streptococcus brevis,* and these organisms may be concerned in the production of catarrhal conditions. Passive congestion in cardiac and hepatic disease may lead to catarrh of the intestines.

**Anatomy.**—The changes in the mucous membrane of the intestine are similar to those in other mucous surfaces of the body. The tissues are more vascular, and become swollen. The epithelial cells, including those of Lieberkühn's glands, are swollen, cloudy, and become detached, forming mucus, which is present in large quantity, and cellular infiltration takes place in the intertubular tissue. In more advanced cases the solitary follicles are enlarged, and they may become eroded and produce small ulcers (*follicular enteritis*). In some cases also ulcerations occur in other parts of the mucous membrane, and the secretions may consist of muco-pus, or even pus. As a rule, the inflammation subsides, but it may lapse into a *chronic* condition, with more prominent changes in the mucous membrane. Sometimes there is considerable thickening, with

slaty discoloration of the surface; often—especially in the chronic catarrh of infants—there is atrophy of the mucous membrane, involving the glandular layer, but leaving the muscular layer of the mucous membrane, and the submucous tissue, intact.

**Symptoms.**—The chief symptom of enteritis is diarrhœa, or the frequent passage of motions loose or liquid in consistence. This symptom is due not only to the alteration in the secretions poured into the intestinal canal, but also, and largely, to the increased peristaltic movements which the irritation of the mucous membrane calls forth. The condition of the fæces varies much: they are generally at first abundant, liquid, and brownish in colour, with flakes or lumps of more solid matter; but they soon become paler, or, it may be, yellowish or sometimes green. In consistence they are often quite watery, or perhaps slimy, or they contain lumps of stained mucus. Under the microscope there are particles of undigested food, meat fibre, starch granules, and fat, with crystals of ammonium magnesium phosphate, epithelial and pus cells, and bacteria. The bowels may be moved from two or three times a day to ten, twelve, or more.

Pain is often present, but not so much continuously as in the form of colicky or griping attacks, which precede the passage of the motions and subside again afterwards. Actual tenderness is not generally present, nor does examination of the abdomen reveal anything characteristic. Gurgling noises and borborygmi from time to time accompany the more active intestinal movements. The temperature is variable: it may be raised one or two degrees, or remain normal. Frequently the appetite is lost, the patient complains of thirst, the mouth is dry, the tongue is slightly furred, and a considerable degree of bodily weakness results when the diarrhœa is excessive. A very sudden and acute attack may begin with vomiting.

In most cases the symptoms pass off in the course of a few days; the diarrhœa may cease suddenly, leaving a long interval before the bowels are again opened, or the motions may gradually become less frequent, gaining a firmer and firmer consistence. If the complaint becomes chronic, the patient is troubled with three or four evacuations daily of watery mucus, with occasional griping pains. The imperfect digestion and absorption of food may lead to considerable loss of nutrition.

The disturbances of enteritis frequently extend to the large intestine, so that strictly an *entero-colitis* results. When they can be discriminated catarrh of the *small* intestine is more likely to be present if the stomach is at the same time involved; it is less likely to be accompanied by diarrhœa, which must depend finally upon the action of the large intestine. The evacuations often contain bile and undigested food; and mucus, if present, is more intimately mixed with the fæces. In catarrh of the *large* intestine the mucus occurs in separate masses; muco-pus or pus itself may be recognised. As catarrh approaches the *rectum* tenesmus is more likely to be a symptom.

**Treatment.**—The patient should remain in bed and be kept warm. In severe cases it will be advisable to keep the patient without food for the first twenty-four hours; but as much water should be given as the patient wishes to drink. Later gruel, arrowroot, beef tea, or mutton broth, with rusk or toast, milk and soda water, or milk and lime water in small quantities at a time, should take the place of the ordinary meals. They should not be given too hot. Many cases will require drugs in addition. If it is quite certain that irritating matters, such as unripe fruit, are the cause of the attack, and are still in the bowel, a laxative may be given in order to get rid of them and so prevent further irritation. For this purpose a single dose of castor oil may suffice, or one of compound rhubarb powder or of calomel. But generally, by the time the case comes under treatment, there has been a free evacuation, and it is desirable to check the excessive peristalsis and abundant discharge as well as to relieve pain. The tincture of opium may be given in doses of 5 minims every four hours, combined

with an astringent such as hæmatoxylum, catechu, tannigen (5 grains in cachet), the aromatic chalk powder, or dilute sulphuric acid. Bismuth carbonate and bismuth salicylate are also of value, and may be given with opium. If the griping is very severe, morphia may be injected subcutaneously. If the diarrhœa is obstinate and exhausting, an enema of 2 ounces of starch containing 15 minims of laudanum may often be used with success. In children opium must be used as little as possible; aromatic chalk powder and bismuth carbonate or salicylate must first be tried. If opium be necessary, the dose for a child one year old should not exceed 1 minim of the tincture, and it is well to begin with less.

In chronic intestinal catarrh, rest, careful regulation of the diet, with opium, astringents, and antiseptics, are still required. Here also enemas may be found useful—2 or 3 pints of warm water containing 1 per cent. of boric acid, salicylic acid, tannin, or acetate of lead.

### INFANTILE ENTERITIS

Children are subject to a chronic intestinal disorder, which is probably, in part at least, of a catarrhal nature. It is by far most frequent in infants who cannot be brought up at the breast, or must be weaned early and have to depend on artificial feeding. Sometimes immediately after birth, or directly upon weaning, in other cases after a longer or shorter interval, the food disagrees; the child vomits, and the motions are loose; or with not much vomiting, there is constant diarrhœa. If it is taking milk, this is returned in curdled lumps. Occasionally undigested milk curds are passed in the motions. However, the white lumps that are fairly commonly seen in the motions are usually soaps, derived from the fat in the milk. The fæces, which in the healthy infant are yellow, generally become grass-green in colour, are acid, slimy from mucus, and very offensive. Among the poorer people children under twelve or fifteen months are often fed with potato and meat, much the same as their parents, and in such cases undigested particles of these foods are found in the motions. Or if the more saccharine artificial foods are given, or other carbohydrates in excess, fermentation is likely to take place, and acids form, causing increased peristalsis and severe diarrhœa, with loose motions which excoriate the skin round the anus. The children suffer from griping pains, which are brought on by the ingestion of food, or occur just before a loose evacuation. Frequently the abdomen is distended with flatus, but presents nothing on examination with the hand; the condition, though often spoken of by the parents as "consumption of the bowels," has no connection with either tuberculous ulceration, tuberculous peritonitis, or caseation of the mesenteric glands (*tabes mesenterica*), but may be described as fermentative dyspepsia. After a certain time the child emaciates, lies fretful in its mother's arms, and is constantly whining, or screaming from time to time with sudden pain. Finally, collapse and death may terminate the scene.

The *summer diarrhœa* of infants (*epidemic infantile diarrhœa*) is a gastro-enteritis occurring in hot weather, which is the chief predisposing cause. It is commoner in boys than girls. A number of different micro-organisms have been isolated in different epidemics and in different countries, and the disease may be attributed to infection, but not to any specific infection. The disease is much commoner among artificially fed infants, whether on cow's milk or proprietary foods, than among the breast-fed, and this may be due to overfeeding in the hot weather, which upsets the digestive tract, or to the greater possibilities of infection from flies, dirty teats, etc.

There are two types of cases: (1) arising acutely in a well-fed baby; (2) supervening on a case of chronic enteritis with wasting. In the former the liver after death shows great fatty infiltration with some degeneration of liver cells; in the latter there is little fatty change, presumably because there is but little body fat from which the liver can be supplied (*see* p. 563). In both types the intestine

may look normal; but there may be excess of mucus, congestion with petechial hæmorrhages, ulceration with prominence of lymphoid tissue, more marked in the ileal and cæcal regions. Broncho-pneumonia, suppurative peritonitis often presenting no physical signs, sinus thrombosis, and sclerema were characteristic complications (**33**). Sclerema appeared after the patient's chief symptoms had disappeared, and was due to the deposit of a high melting-point fat in the subcutaneous tissues. The symptoms come on acutely with high temperature; vomiting is often absent; the stools are similar to those of infantile enteritis; and death may result in a few days, preceded by collapse, with depressed fontanelle, and unconsciousness, and very often air-hunger due to ketosis (*see* p. 568), or the diarrhœa persists for many days very little amenable to treatment.

**Prevention.**—The management of the child's food is the first consideration. If the child is suckled, it should be seen that the mother's milk is of good quality, and that it is not given too frequently; a baby one or two months old should not be nursed more than once every two or three hours, and the mother must carefully abstain from giving it the breast simply because it cries. If from any cause the mother is unable to suckle it, the most efficient substitute is a wet-nurse, and if this cannot be provided, recourse must be had to the milk of domestic animals—namely, the cow, goat, or ass. Such milks differ from human milk in composition and in the reaction of the casein to the acid of the child's stomach. The average percentage composition of human milk is protein 2·0, fat 3·5, sugar 6·5, mineral matter 0·2, and water 87 or 88. Cows' milk contains twice as much protein, more salts, only slightly more fat, and less lactose, or sugar of milk; the casein coagulates in large lumps. Of the proteins in human milk the caseinogen is only half as much as the whey proteins (chiefly lact-albumin); in cows' milk it is two and a half times as much: thus there is five times as much caseinogen per cent. in cows' milk as in human milk. Goats' milk is very similar to cows' milk, but contains more fat: the casein coagulates in large masses. Asses' milk is deficient in solid constituents, but the casein clots in small particles like that of human milk; it is, therefore, often digested with ease, but it is not sufficiently nutritious to be continued for long. Moreover, the greater facilities for obtaining cows' milk will always be a reason for using it, if it can be so modified as to approximate its composition and properties to those of human milk. The addition of water will reduce the proportion of the proteins and the salts, but the mixture must be sweetened, for instance, by the previous solution of 1 ounce of milk sugar in each pint of water; and 1 drachm of cream (containing 20 per cent. of fat) should be added to each ounce of the milk-and-water mixture, or a drachm each of cream and milk sugar may be added to every 4 ounces of water and milk mixed in the following proportions: for infants under one month old there should be one part of pure milk to two parts of water, for those a little older up to four months equal parts of milk and water, and from four to six months two parts of milk to one of water, and increasing proportions of milk till nine or ten months, when pure milk may be given. The dilution also slightly influences the coagulation of the casein, but this is rendered still more like that of human milk by the addition of lime water or barley water. Lime water, moreover, neutralises the acidity, which distinguishes cows' milk from that of women; it should be in the proportion of one part to two parts of the pure milk employed. Coagulation of the casein is also delayed by the addition of sodium citrate, 1 or 2 grains to each ounce of the milk. Another mode of getting over the difficulty of the digestibility of the casein is to peptonise the milk by means of Benger's liquor pancreaticus or Fairchild's peptogenic powders. This may suffice for a time, but the objection is that it is doing the stomach's work entirely, and depriving it of the exercise of its proper function.

More accurate methods of compounding a suitable milk have been devised, and such an artificial human milk is either a direct mixture of cows' milk, cream,

lactose, and water in certain proportions, or it consists of cows' milk from which some portion of the caseinogen has been removed. Rotch uses a mixture of—milk, 1 ounce; cream (20 per cent.) diluted with one-third part water, 2 ounces; milk sugar, 3ß drachms in water, 3 ounces; lime water diluted with three-fourths part water, 2 ounces. Cheadle altered the milk as follows: The cream was removed by skimming after the milk had stood some time. The remainder was divided into two equal portions, from one of which the caseinogen was removed by rennet. The other half was then added to the whey, and the whole of the cream added. For an older child the caseinogen was removed from a third part only.

But cows' milk, either in transmission or while standing, may be readily contaminated with organic matters of a bacterial nature. Infection by flies should be prevented by keeping it in covered vessels, or in jugs covered with muslin. Contamination, if it is likely to have occurred, must be corrected by *sterilisation*—that is, either by boiling or steaming the milk, or by pasteurisation, or by refrigeration. Boiling has the disadvantage of coagulating the albumin, which, with some fat, is removed in the scum; and the milk is rendered less palatable. But it is a convenient method, and the loss of nutrient value is slight; the boiling, however, must not be prolonged. In the different forms of *sterilisers* the milk is heated just short of boiling, either by exposure in bottles to steam, or by the immersion of a vessel containing the milk in a larger vessel containing water, which is then boiled. The bottles are loosely plugged while exposed to heat, and tightly stopped when the process is complete. Steaming for twenty minutes will suffice for milk to be used immediately, but if it is required to keep for the day a longer time must be given. Rotch directs that his mixture shall be sterilised before the lime water is added.

*Pasteurisation* is the exposure of the milk for fifteen or thirty minutes to temperatures below that of boiling, which are sufficient to check the growth of bacteria, and do not damage the milk as boiling does. These are 65° to 70° C. or 149° to 158° F.

*Refrigeration* answers the same purpose, since a temperature of 40° F. will entirely stop bacterial growth. The milk should be taken direct from the cow to the refrigerator, and when reduced to 40° F. should be bottled and stored at that temperature.

"Humanised" milk can be obtained which is both sterilised and assimilated to the normal composition. A sterilised milk diminishes the risk of infection by typhoid fever, scarlatina, and diphtheria, when those diseases are prevalent. But its unduly prolonged use has sometimes produced infantile scurvy, and this may have to be provided against (*see* Scorbutus).

The quantity of food required by a bottle-fed child depends on the weight and age of the child. Up to three months the child requires about one-seventh of its body weight of milk in each twenty-four hours; between three and six months one-eighth; between six and nine months one-ninth; between nine and twelve months one-tenth. The baby may be fed every two and a half hours in the day-time to start with, being given eight or nine feeds in the twenty-four hours. The number of the feeds is gradually diminished so that when six months old the baby is receiving five or six feeds in the twenty-four hours. But some regard must be paid to the size and vigour of the infant.

If the child is fed with the bottle, it is essential that there should be no rubber tube on it, but that the nipple should fit directly on to the glass neck; that it should be kept scrupulously clean in all its parts, and washed thoroughly and scalded after every feeding.

**Treatment.**—In an acute attack of diarrhœa the child should be removed from the surroundings in which it contracted the disease. All food containing fat, such as milk, must be stopped. Water containing 7½ per cent. glucose in frequent small feeds may be given by mouth, as this combats the ketosis. It is

essential to keep the child warm in bed. If there is much loss of fluid, as indicated by depression of the fontanelle, hollowness of the eyes and wrinkling of the skin, an infusion of normal saline should be made into the axillæ or peritoneal cavity; but hypotonic saline by mouth is the best method of giving fluid, if it can be retained. After twenty-four hours some food with a low content of fat, such as a malted milk, or broth containing lactose, and following these a dried half-cream milk (**34**). It is a good plan to give a purgative, castor oil or calomel, quite early; morphia should not be given. Vomiting may be combated by washing out the stomach, and the large intestine may be emptied by means of an enema at the beginning of treatment.

In more chronic cases the diet should be arranged as described under Prevention. Medicinally very small doses of mercury may be given with advantage —for instance, calomel $\frac{1}{8}$ or $\frac{1}{6}$ grain with sugar or hydrarg. c. cret. $\frac{1}{2}$ grain or 1 grain, with 1 or 2 grains of bicarbonate of sodium, two or three times a day. If diarrhœa is obstinate and associated with much pain, it is desirable to give bismuth (2 to 5 grains of the carbonate or salicylate), creosote ($\frac{1}{2}$ minim, or 1 minim with mucilage), or astringents, such as aromatic chalk powder (5 grains), extract of hæmatoxylum (2 grains), tannigen (2 grains), or tincture of catechu (10 or 15 minims). Either of these may be combined for older children with small doses of opiates in the form of Dover's powder ($\frac{1}{2}$ or 2 grains).

If there is much collapse, brandy must be given in doses of 10 to 30 minims every three or four hours, or liquor strychninæ ($\frac{1}{2}$ or 1 minim) should be injected subcutaneously.

## FOOD POISONING

It has been already stated that simple catarrhal enteritis is probably due in many cases to micro-organisms, but there is a large class of cases in which, either from the obvious fact that decomposing food was ingested, or from the occurrence of the illness simultaneously in a number of persons who have all eaten of the same, perhaps unsuspected, food, it is quite clear that the food is responsible for the symptoms, which are those of an acute gastro-enteritis. Some of these cases were formerly called *English cholera*, or *Cholera nostras*. More recently they have often been described as instances of *ptomaine-poisoning*, but on very insufficient grounds, for, as a fact, ptomaines are rarely, if ever, found either in the food or in the organs of the sufferers, whereas certain bacteria have been constantly found, and the presence of toxins has been demonstrated. The organisms which have been most frequently found are the *B. enteritidis* of Gaertner, *B. suipestifer* (or *ærtryche*), type Mutton, less commonly type Newport, where diseased meat has been eaten. The *B. botulinus*, which does not produce symptoms of enteritis, has been found chiefly in sausages made from liver and blood in Germany, and in tinned fruit and vegetables in the United States. Its spores are widely diffused in nature and the bacillus grows anaerobically, producing a toxin which is a very powerful poison, but which is easily destroyed by a temperature of 80° C. (**35**).

In the case of "Gaertner" and "ærtryche" the symptoms come on as a rule within from six to twelve hours after eating the food, and consist of vomiting, diarrhœa, colicky pains, numbness, and weakness, with perhaps albuminuria, catarrhal pneumonia, and cutaneous lesions, such as herpes, erythema, urticaria, and petechial hæmorrhages. In the less acute and more prolonged cases there may be a close resemblance to typhoid, or paratyphoid, fever. The cases are sometimes fatal, and the autopsies have shown acute gastro-enteritis, sometimes with hæmorrhages, swelling of Peyer's patches, enlarged spleen, and congestion of the liver and kidneys. The bacilli may be isolated from the blood, bowels, or solid organs. The bacillus of Gaertner usually produces more serious symptoms than "ærtryche." Similar symptoms are sometimes caused by eating diseased fish, crabs, mussels, tinned foods, cheese, ice-creams, and even potatoes; and

the cause has often been found to be one of the above-named organisms. Food poisoning by these organisms must be distinguished from typhoid and paratyphoid infections, the usual method being to test the agglutinating power of the patient's serum towards these various organisms.

The symptoms caused by *B. botulinus*, known as *botulism*, concern the nervous system, and consist of paralysis of accommodation, diplopia, ptosis, dysphagia, aphonia, and diminished salivary secretion. Death occurs with great suffering, since the patient retains full consciousness, while he cannot see, speak, or swallow, and his respiratory movements become gradually paralysed (**36**).

**Treatment.**—In cases of gastro-enteritis the stomach should be washed out to prevent further infection; and, except in cases of great collapse, a laxative should be given to clear out the intestines. Stimulants are often required, such as brandy, ether, and ammonia, and if the diarrhœa is a prominent symptom opium in small doses, 5 to 10 minims of the tincture. In cases of great collapse, normal saline solution should be injected into the subcutaneous tissue.

## CŒLIAC DISEASE

This is the name given by Gee to an uncommon disease of children, which presents some similarity to sprue. The child, generally between the ages of one and five, passes abundant pale or almost colourless semi-fluid stools, like gruel or porridge, with a very offensive odour. These stools contain an excess of fat, and with the fatty acid there is a loss of calcium. The abdomen is full, but not tense; there is flatulence, but no vomiting. The child grows pale, thin, and apathetic, but is not febrile; there is marked delay in the growth of the child, although the mental characters are normal: in fact, the disease is sometimes called *cœliac infantilism*. The disease is due to a defect in the power of absorbing fat, although there is also some difficulty in the digestion of carbohydrates. It is possible that pancreatic deficiency may play a part in the disease.

**Treatment.**—Fresh cows' milk must be avoided under all circumstances, although dried milks containing little fat have sometimes been tolerated. The best treatment is to employ a completely fat-free diet—rusks, jellies, chicken, broth, rice boiled in water, potato, and revalenta prepared from lentils. Fat may be introduced by inunction with olive oil. Drugs are not of much use. The prognosis is fairly good with suitable treatment.

## SPRUE

Sprue, or psilosis, is a form of chronic catarrhal enteritis which occurs throughout the tropics, with the exception of Africa, especially in India and Ceylon, Southern China, and the Malay States. The cardinal symptoms are four, namely, soreness of the mouth and pharynx, flatulent distension of the abdomen, a peculiar red or "beefy" tongue, and chronic diarrhœa. The soreness of buccal mucous membranes is commonly associated with formation of minute vesicles or aphthæ. The primary cause of the disease is unknown. The stools are characteristically copious, pale-coloured and frothy, with acid reaction and a disagreeable sour smell from excess of fatty acids, largely in the form of calcium soaps. They are often passed in the morning, the patient being free from symptoms for the rest of the day. Temporary improvement often interrupts the progress of the disease, which is, however, progressive in the absence of suitable treatment, leading to marked wasting, anæmia, and death. When the anæmia is well-marked the blood picture resembles closely that of pernicious anæmia. In fatal cases the stomach and intestines, especially the lower ileum and colon, have been found much thinned and atrophied, with different degrees of erosion and ulceration of the mucous membrane, and degeneration of villi and glands and follicles. The pancreas has been found to have fatty and granular degeneration of its cells,

with increase of connective tissue, and in some cases, besides the general wasting of the tissues, especially the liver, the muscles have shown pronounced atrophy.

**Treatment.**—The majority of cases can be successfully treated by an absolute milk diet, amounting to 3, 4, or 5 pints daily: and this should be continued for many weeks after the last symptom; otherwise a relapse may take place. Strawberries may also be given freely, and often with marked benefit. Other cases have responded to an exclusive diet of meat. In most instances it is necessary for the patient to reside in a temperate climate. The distressing symptoms of sprue may be relieved by administration of alkali, and glycerine and borax mouth washes. Castor oil in small doses is of great benefit, and liquid paraffin should be used in convalescence. Recently successful results have been recorded by oral administration of calcium lactate (10 grains) and parathyroid ($\frac{1}{10}$ grain) two or three times a day (**37**).

### PHLEGMONOUS ENTERITIS

In this form all the coats of the bowel are involved, including the serous coat or peritoneum. There is generally intense redness and vascularity, the mucous and submucous coats are thickened, softer, and more friable than natural, and the peritoneum is vascular, sticky, or covered with lymph. It may arise from mechanical interference with the bowel or its circulation by intussusception or strangulated hernia, or from the spread of inflammation from adjacent parts. In intestinal obstruction also the bowel above becomes distended from retained fæces, and ultimately its walls inflame, numerous ulcers may form, known as *distension ulcers*, and frequently the bowel ruptures. Cases are, however, recorded of localised enteritis involving all the coats of the bowel, in which none of these mechanical causes were discoverable. For several inches the coats of the bowel have been much swollen, infiltrated with pus or lymph, and containing minute hæmorrhages. These cases were no doubt due to infection with pyogenic organisms.

**Symptoms.**—These are often the result of the accompanying peritonitis, and consist of pain, vomiting, local tenderness, collapse, distension of the abdomen, and febrile reaction. The purely infective cases above mentioned have presented the symptoms of intestinal obstruction.

**Treatment** must be directed to the primary cause, and in its absence is practically the same as that of peritonitis.

## COLITIS

Inflammation of the colon presents the same varieties as are seen in other mucous membranes, and may thus be catarrhal or ulcerative. Catarrhal colitis is often a part of a general entero-colitis, arises from the same causes, and has very similar symptoms—namely, pain, distension, tenderness, and frequent motions in which mucus, and even occasionally blood, are present. If the lesion is near the rectum, there may also be tenesmus. Catarrhal colitis may exist in an acute or chronic form, and the treatment is not materially different from that of enteritis.

### MUCO-MEMBRANOUS COLITIS

(*Mucous Colic, Mucous Colitis.*)

Muco-membranous colitis is characterised by the discharge *per rectum* of large pieces of membrane or *casts*. It occurs most frequently in middle-aged neurotic females, but is also not very infrequent in children. There is commonly habitual constipation with abdominal discomfort, and other symptoms of chronic dyspepsia. An attack of the disease is accompanied by severe griping pains, which result in the discharge of membranes.

The disease is associated with sudden spastic contractions of the colon with over-production of mucus. This becomes coagulated by a ferment, mucinase, since, owing to the contractions, it is retained for some time in contact with the mucous membrane before being eventually passed. The casts may be several inches or even feet in length, are quite thin and semi-transparent, looking like skins, and embedded in them are epithelial cells, eosinophil leucocytes, cholesterin, and ammonium magnesium phosphate. *Intestinal sand* may also be passed. There is usually some catarrhal colitis present. The narrowing of the lumen of the colon may be observed by taking a series of radiograms after the colon has been filled with an opaque meal. The sigmoidoscope should also be used, in case carcinoma of the pelvic colon is present, as this may give rise to the disease. According to Lockhart Mummery, the disease may be secondary to many conditions—pericolitis, kinking, visceroptosis, displacement of the uterus, etc.

In some of these cases injection, œdema, and ulceration of the mucous membrane have been seen with the sigmoidoscope.

**Treatment.**—If possible the primary cause should be treated. Careful dieting so as to supply a food free from all mechanically irritating particles, fibres, etc., slow eating and careful mastication, and irrigation of the bowel with from 1 to 1½ pints of warm water or 10 ounces of olive oil, are useful means of treatment. Drastic purgatives should be avoided, because by irritation of the colon they will make the condition worse. Castor oil is the most suitable. Liquid paraffin may be used. Belladonna is useful for the pain. The abdomen should also be kept warm by means of an abdominal binder. Sometimes a diet consisting of fruit and vegetables, mostly uncooked, with coarse pods and skins—the antithesis of that described above—is useful, since it attacks the constipation which the patient suffers from. The mental symptoms should be attended to, and the patient's thoughts distracted from her malady. The disease does not endanger life, but it often lasts for years. Occasionally appendicostomy has been performed, and lavage carried out through the opening.

## ULCERATIVE COLITIS

Both typhoid and tuberculous ulcers occur in the cæcum and ascending colon in association with similar lesions in the ileum; syphilitic ulcers occur in the rectum; and the whole length of the large intestine is involved in ulceration with different forms of tropical dysentery.

But cases of *ulcerative colitis* occur outside the tropics which cannot be traced to any of the above infections, and which are mostly sporadic. They are seen in both sexes, and at all ages, though much more common in adults. Often there is no antecedent condition of ill-health, but some cases have occurred in connection with a chronic septic disorder, and in others colitis has been the final event of a chronic nephritis. The *Bacillus coli*, other pyogenic organisms, and the toxins of antecedent septic conditions, have been suggested as causes; but whether the bacteriology of these sporadic cases is the same in all instances cannot be certainly known. However, similar cases have occurred in public institutions, under the name of *asylum dysentery;* and in them the resemblance to tropical dysentery is perhaps closer, and the *B. dysenteriæ* has frequently been found.

**Morbid Anatomy.**—The colon is the seat of large superficial, irregular, confluent ulcers, with intervening vascular, swollen, or pigmented mucous membrane, here and there undermined by the ulcers. Sloughs consisting of necrotic epithelium may be found adhering to the surface in some places, while in other places healing is taking place, as shown by the spread of rapidly regenerating epithelium over the ulcerated areas. At a later stage smooth patches indicative of scarring are found. The general resemblance to bacillary dysentery is very close. Hepatic abscesses occur, but they are rare.

**Symptoms.**—These come on gradually or more acutely, and consist of

paroxysmal griping pain and diarrhœa, with slimy, offensive fluid motions, containing bright blood not mixed with the fæces, and so easily recognisable, a variable amount of mucus and plenty of pus. Sometimes blood, mucus and pus are passed without fæces. In quiescent periods microscopical examination may be necessary for a diagnosis, since the blood is not visible to the naked eye. Tenesmus is present in a minority of the cases only; the abdomen is often distended. The patients become anæmic, have irregular pyrexia, frequently vomit, and lose strength and flesh.

**Diagnosis.**—Careful microscopical examination, especially for the presence of pus, is necessary. Digital and sigmoidoscopic examination is then required to eliminate carcinoma of the pelvic colon and rectum. The patient is prepared for sigmoidoscopic examination by thoroughly clearing out the bowel. A quarter of a grain of morphia is given in a suppository half an hour beforehand, and the sigmoidoscope passed with the patient in the knee-elbow position without an anæsthetic. When the symptoms are due to carcinoma it may be possible to see the growth if situated low down. In ulcerative colitis the pelvic colon and rectum tend to be invaded early, so that the characteristic ulcers can be seen or felt with the finger.

As seen by the sigmoidoscope, the mucous membrane is red and thick, and bleeds very readily when touched. It may be granular. It is covered with bloodstained purulent mucus. Superficial ulcers are present, but in early cases they may be so small that they are difficult to recognise. More often they are of larger size and may contain small islets of mucous membrane, which may feel like flat polypi on digital examination. The ulcers are always superficial, with irregular edges not undermined, and have a greyish-yellow floor when the blood and mucus have been wiped away (Hurst). If the mucous membrane looks healthy, but blood is seen to be coming from higher up, this in itself is in favour of carcinoma. X-ray examination after an opaque meal shows a mottled appearance of the colon, which is often abnormally narrow from spasm.

**Prognosis.**—This depends on the extent of the ulceration, and may be roughly estimated by the amount of pus discharged. Acute cases may die within a few weeks; but often they get well for the time being, only to relapse later.

**Treatment.**—The diet may be of varied character, but care should be taken to exclude substances containing indigestible residues which will irritate the colon, such as tough meat, porridge or whole-meal bread. Vegetables, if passed through a fine sieve, and fruit juice may be allowed. Plenty of milk should be given. Once a day the colon should be cleared out by a warm water enema, and half an hour later 1½ pints of albargin solution should be run in under a pressure of 12 inches so as to reach the cæcum, through a tube passing just through the anus, with the patient in the knee-elbow position. This fluid should be retained for an hour. Charcoal (½ ounce mixed with milk) three times a day has also been of value. Polyvalent anti-dysenteric serum may also be tried. If much loss of blood has occurred, blood transfusion should be carried out. If the case is intractable, appendicostomy or cæcostomy may be performed and the colon irrigated through the stoma.

## APPENDICITIS

Appendicitis is the name given to inflammation of the vermiform appendix, or appendix cæci.

**Ætiology.**—This disease is much more frequent in early life than in middle or old age, and in the male than in the female sex. The greater prevalence of the complaint in recent years, though generally admitted, is quite unexplained.

**Pathology.**—The appendix normally contains, like the bowel, numerous

micro-organisms, of which the *Bacillus coli communis* is the most important. As a rule, however, they are not virulent, or they are prevented by the normal movements and secretions of the bowel from exerting any influence upon the tissues. If, however, any local injury is done to the appendix, or if the lumen is obstructed by a foreign body, or by kinking of the structure, the cavity is distended, the vascular and lymphatic system is deranged, and bacterial invasion becomes possible. Thus appendicitis may have as immediate determinants, besides the presence of organisms: (1) The spread of catarrh from the cæcum to the orifice of the appendix, obstructing this so that the cavity is distended by retained secretions. (2) A foreign body in the cavity of the appendix, irritating it, or obstructing the lumen. This may be a cherry stone, orange pip, seed, bristle, or similar substance. In many cases a concretion is found of the size of a pea or plum stone, yellow or grey in colour, and consisting of fæcal matter, mixed with mucus, lime salts, and numerous bacteria. This is now regarded as being formed subsequently to the catarrh of the appendix. (3) Torsion of the appendix and strangulation of its vessels by undue distension of the cæcum, or traction by bands and adhesions. Besides the *B. coli communis*, streptococci, staphylococci, *B. pyocyaneus*, and other pyogenic organisms, the tubercle bacillus, the typhoid bacillus, and actinomyces are sometimes concerned in the production of appendicitis. But appendicitis also appears to arise from more remote causes, and then possibly is set up rather by toxins than by micro-organisms themselves. Among other things, it is held by some to be one of the results of intestinal stasis (*see* Alimentary Toxæmia).

The results are very various. In the appendix itself may be noted infiltration, thickening of its coats, distension of its cavity with catarrhal products or pus, suppuration of its substance, and, finally, ulceration and gangrene. Thus an inch or more of the extremity of the appendix may be found of yellow, grey, or greenish colour and bloodless; or at one or two points in the length of the appendix a small slough may be formed. In most of these conditions the trouble spreads almost necessarily to the peritoneal covering and to the sub-peritoneal connective tissue. The peritonitis thus arising is mostly localised and adhesive, matting the appendix to the adjacent bowel, and forming thus more or less definite resistant masses in the right iliac region. If the connective tissue is involved it will be also infiltrated, and assist in the production of the swelling. The inflammatory products may be absorbed, or may suppurate; in the latter case, the abscess, if untouched by the surgeon, may burst externally through the skin, or open into the cæcum, bladder, vagina, rectum, or peritoneal cavity, its course being probably determined to some extent by the anatomical position of the appendix in the subject of the disease.

The appendix sometimes perforates or sloughs before adhesive peritonitis has occurred, and then a general peritonitis, of a very fatal kind, is rapidly determined. Occasionally a peritonitis, at first local, gradually extends with the formation of peritoneal abscesses in different parts of the abdomen. Infection may thus spread along the ascending colon to the upper surface of the liver, forming a right subphrenic abscess, or to the lower surface of the liver and the foramen of Winslow; or if the appendix is on the left side of the cæcum, the infection may reach the descending colon, and spread along it to the spleen and left subphrenic region. Sometimes a pelvic abscess is found, and the neighbouring organs are involved in inflammation, such as the bladder and the pelvis of the kidney. Exceptionally infective organisms are carried to the liver by the portal vein, and suppurative pylephlebitis and hepatic abscesses are the result.

If sloughing or suppuration do not take place, the apparent subsidence of the inflammation is by no means always the end of the disease; the condition remains as one of *chronic appendicitis*, and in many cases from six months to two or three years after the attack acute inflammation is again lighted up, and either terminates in one of the above processes or subsides again,

perhaps to be active after yet another interval. In these intervals, as shown by operation in certain cases, the appendix presents thickening and infiltration of its walls, often with constriction in the middle, and dilatation at the distal end, with perhaps concretions in its cavity, and peritoneal adhesions externally; or the cavity may be obliterated and the organ fibrous.

**Symptoms.**—*Acute Attack.*—The onset is often somewhat sudden. Spontaneously, or after some such indiscretion as has been referred to, the patient is taken with severe abdominal pain, at first diffused over the abdomen, but soon more pronounced in the right iliac fossa, with malaise, nausea, vomiting, and some febrile reaction. The tongue is furred, the appetite fails, there is thirst, and the bowels are constipated. The abdomen may be somewhat distended, but is generally rigid; and there is tenderness in the right iliac fossa. This tenderness is often definitely situate at a point about 3 inches from the right anterior superior spinous process, on a line drawn from this process to the umbilicus—*McBurney's point;* and in this neighbourhood, after a time, a certain amount of resistance can be felt. These symptoms may continue for a few days, the vomiting, pain, and tension may diminish under treatment, and the trouble may subside. The pain at the onset is not always diffused over the abdomen generally or situated over the appendical region: it may be epigastric or on the left side, it is frequently umbilical, and it may begin on the left side and go to the right side.

In some cases, however, beginning like this, and in others of which the onset is more gradual, the localised resistance becomes a definite tumour, bounded externally and below by the crest of the ileum and Poupart's ligament, and extending by a convex border half or two-thirds of the distance from Poupart's ligament to the umbilicus. It is often quite dull to percussion, and sometimes it has a modified tympanitic note, while the rest of the abdomen is supple and resonant. The temperature may rise to 103° or 104° F., the pulse to 100 or 120. The pain may be irregular or paroxysmal, and often shoots down the right leg. In its further progress the tumour may subside, gradually becoming less definite and smaller, so that it disappears in from ten to twenty days from the time it was discovered, while the fever and other unfavourable signs diminish. On the other hand, the swelling may suppurate, with still further enlargement, increasing pain, discomfort, and illness, oscillating temperature, with or without rigors, profuse sweating, and all the other indications of septic absorption. Fluctuation is then generally felt, and occasionally the tumour becomes resonant on percussion, from decomposition and formation of gas in the interior. Spontaneous recovery may even now take place by rupture of the sac into the alimentary canal, when the pain and discomfort are quickly relieved, and a quantity of pus may be noticed to pass by the rectum. Generally, however, the abscess has to be opened by the surgeon; and the convalescence may be slow, as often the sinus is a deep one, and there is much surrounding infiltration. Rupture may take place into the peritoneum, into the bowel, vagina, or other cavity. In the first case the symptoms of general peritonitis will succeed; in the others the swelling in the right iliac fossa will subside, pus is discharged with the stools or *per vulvam*, and the patient gradually recovers.

In other cases beginning like the first, neither is there evidence of a general peritonitis, nor the well-defined mass as above described; but there is fulness or resistance in some part of the right half of the abdomen, local or general pain and distension are present, and vomiting and septic conditions persist. In such a case there is probably a deep-seated abscess, which may indeed be felt *per rectum* or *per vaginam*, or by its proximity to the bladder may cause frequent micturition.

In cases of early *sloughing* of the appendix, the local indications may be entirely absent, or so slight that they are scarcely noticed by the patient, or of such short duration that the case is from the first, or quite early, one of general peritonitis (*see* Peritonitis).

Hyperalgesia, *i.e.* a feeling of soreness, or merely hyperæsthesia, *i.e.* increased

tactile sensibility, may be valuable in diagnosis. They are elicited by drawing a pin over the skin held at a constant angle, or lightly picking up the skin between the finger and thumb. In acute appendicitis these signs are found in about 60 per cent. of cases and nearly always in the right iliac fossa, though there may be additional areas posteriorly and on the left side (**38**). Hyperalgesia is a referred pain, and the fact that it can be produced artificially by stretching the walls of a healthy viscus, and may last an hour or so after the stretching has ceased, shows that it has not necessarily anything to do with inflammation, but is the result of the secondary dilatation and stretching of the appendix walls or possibly a segment of ileum or cæcum.

Extension to the general peritoneal surface is usually indicated by increased extent of tenderness, and by immobility of the whole abdomen on respiration. The general condition of the patient also varies a good deal in different cases. The pulse is generally quickened, and the temperature is raised; but sometimes the temperature is low, though the pulse is rapid, and this is generally regarded as a sign of severe infection. And, generally, no doubt the pulse rate is a more important indication of severity than the degree of temperature.

In the relapses of appendicitis, which have been already mentioned, the symptoms are precisely the same as occur in primary attacks; but the liability to general peritonitis is probably less, because adhesions will have formed around the lesion.

*Chronic appendicitis*, whether in the intervals between acute attacks or independently, may be latent; or deep pressure in the appendical region may cause a little pain, and thickening may be felt. Frequently it gives rise to troublesome symptoms, which are apt to be misleading, because they suggest disease remote from the appendix. Thus the patient suffers from attacks of pain in the epigastric or umbilical region, sometimes even on the left side, or in the rectum, if the appendix is in the pelvis. In the last case there may be frequency of micturition, and in the other cases vomiting. The pain lasts from a few hours to one or two days. An important group of cases is that in which the symptoms are deceptively like those of gastric ulcer or less often of duodenal ulcer. The pain occurs in the epigastric, umbilical, or hypochondriac region, coming on within an hour of taking food, and is accompanied by flatulence, distension, and sometimes vomiting. The pain may radiate to the lower abdomen, or to the appendical region. In some cases this latter region is tender, or pressure over the appendix causes a painful sensation in the epigastrium. The pain is aggravated by exercise or exertion. Attacks occur at intervals over some years, and in the intervals of the severe attacks the patient is in many cases not entirely free from pain. This simulation of gastric and duodenal disease is called *appendix dyspepsia;* and the symptoms are attributable to a dyspepsia produced reflexly from the appendix region. The diagnosis is often very difficult; and many cases have only been cleared up by an operation for appendicitis, after an opening in the upper abdomen has shown that the duodenum and stomach are healthy.

**Diagnosis.**—An apparently spontaneous acute general peritonitis in a boy or girl is nearly always the result of appendicitis; in older patients many lesions may be confounded with it. Nearly all the causes of acute abdominal pain and collapse have been at different times mistaken for it, such as perforation of gastric and duodenal ulcers, acute hæmorrhagic pancreatitis, pyosalpinx, cholecystitis, pyelitis, and renal calculus. The past history, the seat of maximum pain and tenderness, and the local conditions as ascertained by examination externally and *per rectum*, must be carefully considered. Among less severe conditions, neuralgia of the lower abdominal nerves, and the gastric crises of tabes dorsalis, may resemble it; and both in children and adults acute pleurisy and pneumonia at the base of the right chest may cause pain sufficiently low down in the right flank to lead to a diagnosis of appendicitis. Typhoid fever has sometimes been

mistaken for appendicitis, and less often appendicitis for typhoid; the latter is the more dangerous error. A leucocytosis in excess of the normal, especially of the polymorphonuclear variety, would be strongly in favour of appendicitis. It will be remembered that an appendicitis may have its origin in typhoid ulceration of the appendix itself.

At a later stage, when a tumour has formed, this has to be distinguished from fæcal accumulations, malignant growth of the cæcum, movable kidney, inflammation of the pelvic organs in women, and psoas abscess.

The diagnosis of appendix dyspepsia, *i.e.* the belief that in a given patient epigastric pain is due to chronic appendicitis depends on some local signs being elicited in the appendix region, and on the absence of occult blood in the stools, (though it must be remembered that chronic appendicitis and peptic ulcer not uncommonly occur together), and on other negative evidence of peptic ulcer or cholecystitis. A valuable confirmatory test is *Bastedo's sign*. This is the occurrence of pain or tenderness in the right iliac fossa when the colon is inflated with air, slowly pumped in through a rectal tube. In the healthy person this produces some discomfort, but only pain in high degrees of distension, and then on both sides equally. When appendicitis is present, the inflation causes pain in the right iliac fossa; and the appendical region becomes tender to pressure, or, if previously tender, the sensation is aggravated. Sometimes also pressure in this situation will, after inflation, set up the very same pain in the epigastrium, which the patient has spontaneously suffered (Hurst). In a high percentage of cases the appendix can be seen by X-rays from six to eighteen hours after giving a meal of barium sulphate and buttermilk (Spriggs) or barium sulphate and water (Redding). From the shape and mobility of the organ valuable indications can be obtained as to whether it is chronically inflamed. The bowels must first be emptied by purgative and enema.

**Prognosis.**—In recent statistics from the London Hospital there was a 1·2 per cent. mortality of all cases of appendicitis operated on within the first twenty-four hours from the beginning of the attack. This had risen to 3·9 per cent. during the second day and to 8·7 per cent. during the third day. The earlier the operation, the better the prognosis.

**Treatment.**—The facts that appendicitis may progress to the stages of gangrene and suppuration with so few symptoms, and that it is so difficult to ascertain without operation the extent of the danger, have led to the conviction that the removal of the appendix should be undertaken whenever a certain or highly probable diagnosis of appendicitis has been made; and this should be done at the earliest possible moment. If there is doubt about the diagnosis, or the symptoms are less urgent, it may be justifiable to wait; and then the patient should be put to bed, and fed only on milk, Benger's food, and similar articles of diet; hot boric lint or fomentations should be applied, and all aperients should be withheld, except a simple enema if the bowels have not recently acted. In cases where no operation has been performed at the onset, and no localised tumour can be recognised, and yet there is reason to believe from continued pain, from tenderness on deep pressure, fulness or resistance in the right iliac fossa, or right flank, distension of the abdomen generally, fulness or resistance in the rectum, or irritation of the bladder, that there is deep-seated suppuration, laparotomy should be performed. An operation is imperative at the first sign of an extension of inflammation to the peritoneum generally.

Whether every case which has recovered under medical treatment should be operated on some months later to prevent recurrence is an open question; but if a second attack occurs, certainly the opportunity should be taken to operate: and if this attack subsides without operation, what remains of the appendix should be removed some weeks after recovery. Similarly, after a first attack, if recovery is delayed or incomplete, with recurrent local pain, febrile attacks, tenderness and thickening, or if the case can be reasonably diagnosed as one of

appendix dyspepsia, the operation should be performed. If laparotomy is performed the precaution should be taken to examine other parts of the abdomen, and particularly the gall bladder, for associated disease.

## TUBERCLE, NEW GROWTHS, AND SYPHILIS OF THE INTESTINE, AND DIVERTICULITIS

**Tubercle.**—Besides its occurrence in phthisis, tuberculosis of the intestine may arise independently, especially in children, to whom the infection is often conveyed by milk. In either case the lesions occur chiefly in Peyer's patches in the ileum, and in the solitary follicles of the ileum and colon. The process is the same as elsewhere: cell proliferation, caseation, necrosis, and ulceration; and the ulcers in the ileum may be of great extent, being round or oval, and frequently running transversely round the gut rather than along it. The surface is regular, with thickened edges, and the serous surface corresponding to it generally presents small white tubercles in some number. The associated symptoms are pyrexia and diarrhœa. The motions are generally abundant, pultaceous, fatty, and yellow in colour. Sometimes they are more liquid, and still yellow, and if the abdomen is distended there may be a close resemblance to those of typhoid fever. Hæmorrhage and perforation are rare. The treatment has been indicated (*see* p. 264). Fistula *in ano* is no doubt sometimes tuberculous in origin.

**New Growths.**—The new growths of the intestine are chiefly *adenoma* and *carcinoma*, and rarely *lymphosarcoma*. Carcinoma is the most frequent and important.

Carcinoma of the duodenum has been divided by Sherren according to the part affected into that situate above the bile papilla, supra-ampullary, that in the papilla itself, ampullary, and that lower down, infra-ampullary. In any case it is a rare disease; but the ampullary form is of importance, because the tumour blocks the common bile duct, and though not painful, leads to deep jaundice and emaciation. Infra-ampullary carcinoma in the third portion is more common. In these cases the vomit always contains bile and pancreatic juice, the last of which may be shown by the digestion of fibrin in the filtered vomit after the addition of a few grains of sodium bicarbonate. The jejunum and ileum are rarely the seat of tumours. Benign tumours are often pedunculated, and these may lead to intussusception. Bland Sutton points out that some tumours at the ileo-cæcal junction believed to be carcinomatous have really been hyperplastic tuberculous masses, which may be in a position to obstruct the bowel as much as a carcinoma. Excision is the treatment necessary in either case.

Carcinoma is much more common in the large than in the small intestine, and affects especially the cæcum, pelvic colon, and rectum, and less often other parts of the colon. Clinically, carcinoma coli is of two types, depending on whether it is an annular growth which causes obstruction (*see* p. 457) at an early stage, or a fungating growth which produces obstruction late. In the early stages the patient may notice discomfort and colicky pains in the abdomen after meals accompanied by borborygmi. The action of the bowels is irregular, diarrhœa being rather characteristic of cæcal growths and constipation of pelvic and rectal growths. In the latter there may be a *spurious* diarrhœa, *i.e.* the frequent discharge of small fluid motions owing to irritation caused by the growth, accompanied by staining or tenesmus. The stools contain blood, not necessarily in large quantity. The presence of hæmatin and absence of hæmato-porphyrin suggest that the bleeding has taken place in the lower part of the colon (*see* p. 408). Mucus in the membranous or slimy form is commonly found and there may be a little pus. Abdominal palpation may disclose a hard rounded or nodular lump, and with the finger one may be able to recognise a mass of new

## PLATE XXVII.

Radiogram showing Carcinoma of the Iliac Colon. A barium enema has been given, and it has filled up the coils of the pelvic colon. The enema has been arrested in the left iliac fossa. This fact, associated with the irregular upper edge of the shadow, is in favour of the presence of growth at this point. A part of the transverse colon leading up towards the splenic flexone is seen at the top of the picture. This has been filled by means of a barium meal. (From a plate taken by Mr. W. Lindsay Locke.)

[*To face p.* 457.

growth blocking the passage of the rectum not far from the anus. The sigmoidoscope should, however, be used in every suspected case, as it enables a growth of the pelvic colon to be recognised (*see* p. 451). X-rays also may be used for diagnosis. An opaque enema, consisting of barium sulphate suspended in water by mucilage, is run into the rectum. If the funnel is held 2 feet above the anus, the mixture passes round to the cæcum, completely filling the colon, if the patient breathes deeply and at times alters his position. The colon must be emptied before the examination by giving castor oil thirty-six hours previously supplemented by water enemas on the morning of the examination and the evening before. Any narrowing of the lumen or irregularity in its outline is suggestive of carcinoma (*see* Plate XXVII.). An opaque meal traced downwards through the colon is of much less assistance in diagnosis.

In the later stages the patient may present the signs of acute intestinal obstruction, of chronic intestinal obstruction, or he may complain of an abdominal lump, which may be confounded with other lesions. Affecting the cæcum, it may resemble a chronic appendicitis, or enlarged glands; in the colon an enlarged kidney, a diverticulitis, an enlarged gall bladder, or an aneurysm if it overlies the aorta.

The treatment is surgical, and the prognosis good in the early stages. In inoperable cases deep X-ray therapy may be tried.

Polypoid growths, adeno-papillomas of the colon and rectum, occur in bilharziasis.

**Inflammatory Strictures of the Rectum.**—*Syphilis* rarely affects the alimentary canal between the pharynx and the rectum, but it sometimes causes stricture in this last situation. Gummas form in the submucous tissue and slowly lead to cicatricial contraction, owing to secondary inflammatory changes. Stricture may also result from gonorrhœa and more rarely from tubercle (**39**). They occur equally in both sexes, and little may be known of them till symptoms of stricture are observed, and the constriction is recognised by digital examination.

**Diverticulitis.**—This is an inflammatory condition of the pelvic colon associated with diverticula which project out into the appendices epiploicæ through a series of small openings in the mucous membrane. Constipation and flatulent distension probably play an important part in their formation by causing increased pressure in the colon. Diverticula are in themselves quite harmless, but they are apt to undergo changes which are harmful. These changes are: (1) ulceration, which may lead (*a*) to perforation and peritonitis, or (*b*) to fistulæ with neighbouring organs, or (*c*) to peridiverticular fibrous hyperplasia which is secondary to the inflammation (this may form a large mass palpable in the abdomen, and by contraction of the fibrous tissue stenosis of the bowel may result); (2) fæcal concretions in the diverticula; (3) torsion of a diverticulum with strangulation; (4) secondary development of carcinoma.

The symptoms depend on the particular morbid process at work. There may be (1) an acute inflammatory disturbance in the left iliac fossa resembling appendicitis, only on the left side of the abdomen; (2) the development of a lump resembling carcinoma; (3) intestinal obstruction; (4) symptoms from formation of fistulæ. The sigmoidoscope, X-rays, and opaque enema and cystoscope may be useful in diagnosis. The treatment is surgical.

## INTESTINAL OBSTRUCTION

The intestine may be obstructed in several ways: (1) foreign bodies, large gall stones, or collections of fæcal matter in its interior; (2) intussusception or invagination; (3) changes in the intestinal walls, such as strictures caused by healed ulcers, or by malignant growths; (4) volvulus; (5) strangulation by

bands or through apertures; (6) diminution of the calibre due to traction on the intestine, or to compression from outside in various ways.

**Pathology.**—*Foreign Bodies.*—Among the foreign bodies found obstructing the bowels are fruit stones, pebbles, coins, bullets, pins, needles, hooks, and false teeth. Some large masses are formed of vegetable fibre, wool, or husks of oats, matted together. It is especially in lunatics that foreign bodies of these kinds are found. Occasionally a large gall stone is the cause of a fatal obstruction, or it passes *per anum* after more or less difficulty. Such gall stones may be 2 or 3 inches in length by 3 or 4 in circumference; they are formed in the gall bladder, and reach the bowel, not through the bile duct, but by ulceration through the walls of the gall bladder and the duodenum. They commonly obstruct the small intestine, especially the lower part of the ileum or the duodenum. Fæcal masses may accumulate in the same way as has been described under Constipation, and form a serious obstacle in the rectum or colon. They are more common in women than in men, and are mostly met with in adults. Masses of magnesium salts, after the extreme use of carbonate of magnesium, have been known to form in the bowel and give rise to obstruction.

*Intussusception.*—This presents special features which make it desirable to consider it separately (*see* p. 464).

*Strictures.*—These occur both in the small and large intestines; they arise either from contraction of cicatrices of ulcers, or from new growths in the intestinal walls. Of the different forms of ulceration dysenteric and catarrhal ulcers most frequently give rise to stenosis, and typhoid ulcers rarely, if ever, do so. But in many cases of stricture there is no evidence as to what form of ulceration has preceded it. Simple strictures are generally single. Occasionally they follow the reduction of a strangulated hernia or injuries to the abdomen. The new growths causing stricture are, as a rule, of malignant nature, and usually *columnar cell carcinoma.* In the majority of cases it is a primary growth, but occasionally it is secondary or extends from adjacent parts. The form it commonly assumes is that of a band or ring round the bowel, by which the internal circumference is considerably reduced, even to the size of a cedar pencil, but rarely completely obliterated. The longitudinal extent of the growth is often not more than 1 or 2 inches; the inner surface is frequently ulcerated. Simple tumours, such as *adenoma* and *fibroma,* are only occasionally the cause of intestinal obstruction.

Strictures, whether simple or malignant, are more common in the large than in the small intestine; this is especially true of malignant disease. And in the large intestine itself the pelvic colon is most often the seat of stricture; the descending colon comes next in frequency, and the hepatic flexure is somewhat more often attacked than the splenic flexure. In nearly 70 per cent. it is the pelvic or descending colon. Females are somewhat more often affected than males; the patients are generally middle-aged, malignant cases being usually about forty, and cases of simple stricture somewhat younger. Strictures are essentially of slow development, and the gradually increasing obstruction influences the condition of the bowel above. This becomes distended by the accumulation of fæces, and hypertrophied in its efforts to force its contents past the stricture. Fæcal matter naturally accumulates, but is from time to time passed through, sometimes after symptoms of almost fatal intensity; occasionally, however, foreign bodies, such as fruit stones, of larger diameter than the aperture, may collect above it. Increasing distension and pressure from within may ultimately lead to ulceration, gangrene, and perforation of the gut above the stricture. In strictures of the pelvic or descending colon fæcal matter often accumulates in the cæcum.

*Volvulus.*—By this term is meant the twisting of a loop of bowel upon itself, so that the two portions at the ends of the loop cross and strangulate each other. It is most common in the sigmoid flexure, or pelvic colon, which may form a loop sufficiently free, from the length of its meso-colon, for the purpose. More rarely

the cæcum is twisted on its vertical axis so as to cause obstruction, or is bent up in front of itself; and occasionally the small intestine forms a volvulus of the same kind as that described in the sigmoid.

In another form of volvulus two loops help to strangulate one another; so, for instance, the pelvic colon may be doubled round a loop of the ileum, or two loops of the ileum round one another. Volvulus occurs in males more often than in females, and mostly between the ages of forty and sixty.

*Strangulation by Bands and through Apertures.*—This class of cases is precisely analogous to ordinary cases of hernia, and they are often described as internal strangulation and internal hernia; a loop of intestine, commonly the ileum, slips through an aperture, and is strangulated by the margin of the aperture grasping its neck. Some apertures are slits in the omentum or mesentery. Another kind of aperture is the opening into a fossa, or pouch of peritoneum, such as are found in connection with the duodenum and the ileo-cæcal junction; and a hernia may occur into the lesser cavity of the peritoneum through the foramen of Winslow. But much more frequently the constricting ring is formed by a band of adhesion stretching from one part of the abdomen to another, under which the loop of gut passes, or by the same band forming more or less complicated loops in which the gut is involved. Bands of this kind arise from a former peritonitis, probably local in extent, and often in connection with the pelvic viscera or the appendix cæci. They are often solitary, though they may be accompanied by other adhesions not forming bands. The pedicle of an ovarian tumour may strangulate the bowel.

A frequent cause of this form of obstruction is the congenital abnormality known as *Meckel's diverticulum*. This forms a finger-like projection from the unattached side of the ileum, from 2 to 4 inches in length, and ½ to ¾ inch in diameter. It has the same serous, muscular, and mucous coats as the ileum, and is a remnant of the omphalo-mesenteric duct, by which the primitive alimentary canal communicates with the yolk sac. It arises from the ileum, at a point 18 to 24 inches from the cæcum, and its blind termination is generally free; but it may be attached by a fibrous band to the anterior abdominal wall at the umbilicus, or to the mesentery, or to the peritoneal surface at some other point. A ring is thus formed, through which a loop of gut may slip, and then become strangulated.

When once the loop has slipped through, strangulation is favoured by everything which increases the contents of the loop, such as more air or intestinal liquid; and not infrequently the loop itself becomes twisted like a volvulus.

Strangulation by bands and apertures is more frequent in males than in females, and occurs at all ages, but mostly between the ages of twenty and forty. Among cases occurring in early life strangulation by Meckel's diverticulum is the most common.

*Compression and Traction.*—This class includes the following forms of interference with the calibre of the gut: acute kinking due to traction upon an isolated band, or an adherent diverticulum; adhesions retaining the bowel in a bent position; adhesions compressing the gut; matting together of several coils; changes effected in the intestinal coils due to simple traction; and narrowing of the bowel from shrinking of the mesentery after inflammation. Such cases are comparatively rare; they concern the large intestine and small intestine with about equal frequency; and they are likely to be preceded by a history of peritonitis.

**Effects of Obstruction upon the Bowel.**—In a fatal case of acute obstruction of the intestine, the bowel above the seat of obstruction is found enormously distended, while that below is collapsed and empty. The distension begins immediately above the constriction, and affects the bowel for a greater or less distance, according to the severity or duration of the obstruction. Thus in obstruction at the sigmoid the whole colon and much of the small intestine are

affected; in obstruction of the ileum the small intestine is distended and the colon is collapsed. In the upper distended portion is a quantity of fæcal matter, light brown or yellowish brown in colour, and of uniform thick liquid consistence; and this is the same whether the obstruction is in the small or large intestine; there is never sufficient absorption by the intestinal vessels to form the harder and drier fæces of health. In chronic cases the distended bowel becomes gradually hypertrophied from its efforts to overcome the obstruction. If this is unrelieved, ulceration, sloughing, and rupture or perforation take place, with peritonitis as a result. In acute strangulation sloughing may occur at the seat of constriction, from direct interference with the circulation; in the chronic obstruction of strictures the bowel yields in the distended portion above. Where large fæcal accumulations are the cause of obstruction, the scybalous masses irritate the mucous membrane and set up catarrh and ulceration, forming so-called *stercoral ulcers*.

**General Symptoms of Obstruction.**—The symptoms of intestinal obstruction are vomiting, constipation, pain, and distension of the abdomen. The special feature of the *vomiting* is its *stercoraceous* or fæcal character. At first the contents of the stomach are discharged, and subsequently bilious matter; but comparatively early in acute cases, and with the final obstruction in chronic cases, the vomited matter consists of light or dark brown thick liquid, with a distinct or even strong fæcal odour. This rejection of the contents of the bowel has been ascribed to its increased peristaltic movements driving downwards the liquid which is next to the intestinal wall, while a central current is established in a reverse direction—that is, towards the stomach.

The *pain* of obstruction is variable. In acute cases it is very severe, generally paroxysmal at first, and not becoming continuous until the obstruction is complete. Its situation is sometimes determined by the position of the lesion in the abdomen, but often it is referred to the umbilical region though the strangulation may be in quite another part of the abdomen. The pain in chronic obstruction may be very slight, but it is aggravated when obstruction increases to a marked degree Tenderness is not generally present until peritonitis sets in.

*Constipation* is an important feature in obstruction, though not in itself conclusive, as it is present in other conditions. It is generally absolute from the time of obstruction—not only is there no motion, but also no flatus whatever. Occasionally, however, the lower bowel may contain fæces at the time the obstruction occurs, and these may be discharged, or removed by an enema.

For a more complete description of the symptoms and course of intestinal obstruction it is necessary to distinguish between acute cases, of which the strangulation by a band is the most typical example, and chronic cases, of which malignant stricture of the sigmoid is the best instance.

**Symptoms of Acute Obstruction.**—In a case of strangulation by a band the patient is seized with intense pain in the abdomen, generally in the neighbourhood of the umbilicus; he may be walking about, or having a meal, or he may be awakened from sleep. Sometimes the attack is attributed to a strain, or to some unaccustomed or indigestible food taken some hours previously; but it is often impossible to prove the connection. The patient then vomits, either directly or within a short time, the vomited matter being the contents of the stomach. The pain is almost continuous, and vomiting is excited by every attempt to take food. The abdomen generally becomes tense, but the actual distension varies with the position of the obstruction; if this is in the upper part of the small intestine, the abdomen may be flat, or distended only at the upper part, above the umbilicus; if the lower part of the ileum is strangulated, the abdomen is uniformly enlarged. Neither motion nor flatus is passed *per anum;* and the vomiting, at first gastric, then bilious, becomes ultimately stercoraceous.

The effect upon the patient is very grave. Collapse soon sets in; the face is drawn, the eyes are dark and sunken, the pulse small and quick, the temperature normal or subnormal, and much flesh may be lost in a few days. The tongue is

dry, and there is constant thirst. The urine is scanty and high-coloured; its quantity tends to be less the higher the seat of the obstruction—a fact which is to be attributed to the generally more constant vomiting, so that but little food or fluid is absorbed into the system. If the condition is unrelieved, death supervenes, either from exhaustion or from peritonitis, of which a general diffused tenderness may be the chief indication. The duration of the case is from four to six days.

The forms of intestinal obstruction which commonly cause acute obstruction, besides strangulation by bands and apertures, are intussusception, volvulus, impaction by a gall stone, and some forms of acute kinking by adhesions. The attempt to distinguish between these cases is often unsuccessful, as the results of an obstruction must depend much more upon its position in the length of the intestine, upon the rapidity of its occurrence, and upon its completeness than upon the actual tissue changes. And the practical value of the differential diagnosis is not great, as there are few cases, if any, which can be safely left without an operation. Acute kinking produces symptoms like those above described, but the pain is generally less continuous, the case is less rapid, and the symptoms more variable in intensity. In volvulus there is very great distension of the abdomen and embarrassment of respiration; but pain, vomiting, and collapse are often not so severe as in other cases, and the duration may be from four to ten or fifteen days. Intussusception has its special features, described later.

In a case of obstruction from a gall stone the earlier symptoms are probably due to the gall stone passing along and irritating the bowel, and only later when the stone is impacted in the ileum to actual obstruction. There may be in such a case a previous history of gall stone attacks, but often so many years before as to be of doubtful value in diagnosis. Thus the obstruction is at first only partial or incomplete, and the bowels may be open; but the symptoms are, nevertheless, acute, and consist (1) of severe pain, intermittent or colicky, at first in the epigastrium, later lower down, and (2) of vomiting, which is more profuse than in any other form of obstruction, and the result of which is a liquid, black or brown and showing in the early stage perhaps some blood or bile. Often the vomiting becomes less about the third day, and the bowels may then be moved. Later, when the gall stone becomes impacted, as it often is, in the ileum, the obstruction is complete, the vomit is stercoraceous, and exhaustion quickly follows.

Other foreign bodies less often cause acute obstruction.

**Symptoms of Chronic Obstruction.**—In chronic obstruction such as is due to malignant disease of the pelvic colon or of the descending colon, the symptoms are at first only indicative of a moderate interference with the passage of fæces; there are some local pain and occasional vomiting, not particularly related to the ingestion of food. Constipation occurs irregularly, but it can be overcome by aperients. From time to time the constipation is very troublesome, vomiting is more frequent, yet not stercoraceous, the abdomen becomes greatly distended, and the hypertrophied coils become visible in peristaltic movements on the surface of the abdomen. When the distension mainly affects the colon, as in the case of sigmoid carcinoma, the transverse colon bends downwards in the middle and forms two enormous vertical coils. When the small intestine is chiefly distended, and the colon is collapsed, the distended coils often lie transversely across the abdomen. With the peristaltic movement can be heard gurgling sounds, or borborygmi.

After a week or ten days of such symptoms some fluid motions may pass, and then quickly several large evacuations of liquid fæces, by which the abdomen is rapidly reduced to its normal capacity, and all the symptoms are relieved. This sequence of events may recur more than once, but in some such attack the constipation becomes complete, nothing is passed *per anum*, vomiting is more frequent and becomes stercoraceous, the abdomen is enormously distended, with visible moving coils, there is severe pain of griping character, and after some days—

it may be as many as ten or twelve—death takes place from exhaustion, or from rupture of the bowel and peritonitis. If the case is seen early, a tumour can be sometimes detected in the left iliac fossa, or one or other flank, but its recognition may be quite impossible when the abdomen has become much distended. The patient may also present the loss of colour and emaciation so common in cases of malignant disease.

The chronic cases which produce somewhat similar symptoms are other forms of stricture of the large intestine, strictures and growths of the small intestine, most forms of compression, traction, and matting of the gut by adhesion, some cases of volvulus, compression of the gut by tumours outside it, some cases of impaction of a foreign body, and fæcal accumulations.

Sufferers from *fæcal accumulations* have generally had previous attacks of constipation, which have only been relieved by strong aperients; and at length even these are useless. The patient then has indigestion and flatulence; the abdomen swells, it may be to an enormous extent, and causes dyspnœa by its pressure on the diaphragm, while the mass of fæces may press upon the lumbar or sacral plexus, or the abdominal or pelvic veins. Sometimes the fæces excite catarrh of the bowel, and a little thin fluid escapes, which may be mistaken for a genuine relief. Nausea, eructations, and vomiting follow, and the coils may be so distended as to be visible on the surface. Occasionally the vomiting becomes stercoraceous, and death takes place from exhaustion. In many cases a tumour due to the accumulated fæces can be felt; this is especially the case if the obstruction is in the large intestine, when the mass often occupies the cæcum and the ascending colon. The tumour is uneven, round or elongated, and generally painless; often it can be indented by the finger, or is doughy in consistence. The duration of these cases may be several months.

**Position of the Stricture.**—The differences to be noted between strictures of the small intestine and those of the large are that in the former vomiting occurs earlier, and is more determined by the ingestion of food: in the latter distension is greater, and the proximity of the stricture or growth to the anus may lead to alterations in the shape of the motions, which may be ribbon-shaped; and tenesmus is frequently present. The stools, moreover, often contain blood. If the abdomen is distended, the prominence is greatest in the middle line in obstruction of the small intestine or of the ascending colon, but more general if the pelvic colon or the descending colon is the seat of disease.

**Diagnosis of Obstruction.**—Acute abdominal pain and distension with vomiting and constipation are common results of so many abdominal lesions that diagnosis is often very difficult. Those which may be wrongly taken for intestinal obstruction are especially the *perforation* of *gastric* or *duodenal ulcers*, *appendicitis*, *acute perforative peritonitis* from any cause, and *acute hæmorrhagic pancreatitis*, in all of which the resemblance may be so close that only by an operation can the diagnosis be established. The following points may serve as guides: In peritonitis there are diffused tenderness and general distension; the temperature is often raised; the vomiting is perhaps less severe than in strangulation, and is rarely fæcal. The onset may have been preceded by symptoms pointing to the appendix. Fæcal vomiting forms the strongest evidence in favour of obstruction, but the vomited matter must, if possible, be seen, as friends will often represent as fæcal what is merely gastric or bilious. In the event of the vomit having been thrown away without proper examination, it is best to pass a stomach tube, in order to ascertain the exact nature of the stomach contents. The rectum should be examined by the finger; it is often empty and dilated, or "ballooned," in cases of obstruction.

In *Henoch's purpura*, the abdominal symptoms may be like those of obstruction; sometimes these are due to hæmorrhage into the wall of the bowel, or to the intussusceptions which may thereupon ensue.

It is very important to recognise that obstruction may be simulated by some

conditions of nervous or toxic origin in which mechanical or inflammatory lesions have no part. One is the *gastric crisis* of tabes dorsalis, in which pain and vomiting occur; but the abdomen is retracted, and the vomited fluid, though abundant, is dirty green and watery, but not fæcal. A history of similar attacks in the same patient, and the absence of knee jerks and of the pupil light reflex, would speak for tabes. Another condition is the acute pain of commencing *diabetic coma*, which has more than once nearly led to operation; the patient is generally beginning to be drowsy, and sugar and aceto-acetic acid are found in the urine, if looked for.

On the other hand, intestinal obstruction itself has sometimes been mistaken for other diseases—*cholera, lead colic, hepatic colic, renal colic, arsenical poisoning,* and even *meningitis*. The possibility of obstruction from an extra-abdominal hernia, whether inguinal, femoral, or obturator, should not be forgotten. The corresponding opening should be investigated; but if the obstruction can be certainly located within the abdomen, the differential diagnosis as to the exact position is of small importance, as a laparotomy becomes absolutely essential.

**Treatment.**—When the diagnosis of acute intestinal obstruction is established the operation of *laparotomy* or opening the abdomen should be performed without delay; and the cause should be ascertained and an attempt made to remove it, as, for instance, by reducing a strangulated coil, dividing a band, unfolding or resecting a volvulus, or extracting a gall stone. Even if the diagnosis between obstruction and peritonitis cannot be determined, the operation is still desirable, as assisting both diagnosis and treatment. If the obstruction cannot be found, or the parts are matted together so that it cannot be dealt with, the bowel must be opened above the obstruction either in the wound employed, or through a fresh opening in the loin, as seems most expedient. If the patient is already so exhausted, when the lesion is first recognised, as to make it highly probable that he will sink under a peritoneal operation, the bowel should be simply opened at the most distended part, and a fæcal fistula established. Saline infusions may be given with great advantage. In suspected cases previous to operation, and in cases where for any reason an operation is not performed, the patient should be fed by nutrient enemas; and purgatives must not be given, for they can only aggravate the case by exciting the peristalsis of the intestines to fruitless efforts, whereby the congestion and strangulation of the bowel are actually increased, and the pain and vomiting are rendered more severe. It is rarely wise to give opium or morphia for the relief of pain: it has the serious disadvantage that, while easing the pain and checking sickness, it removes two important symptoms, and may lull to a false security while the fatal mischief is progressing. Locally relief may be furthered by the application of turpentine stupes; or of flannels wrung out of hot water, and sprinkled with tincture of belladonna, or opium; or of hot linseed-meal poultices.

In *chronic* obstruction, which is chiefly the result of strictures and growths, whether in the small or large intestine, the diet must be carefully selected, with the object of ensuring regular digestion and the easy passage of the intestinal contents through the constriction. Enemas, and occasionally laxatives, may be used to maintain a periodical evacuation. If an obstinate constipation ensues, and especially if great distension and sickness occur, the treatment must be assimilated to that of an acute obstruction. Opium may be given, with or without belladonna, while food must be given in only small quantities, or *per rectum*, when relief may be shortly obtained. Eventually, if life is to be prolonged, an operation will become necessary.

For stricture of the colon a colostomy should be done in the right or left loin, according to the position of the obstruction; in some cases the removal of the diseased portion of the bowel (*colectomy*) may be desirable. For the small intestine laparotomy will probably be required, and the bowel must be dealt with by intestinal anastomosis or excision.

For fæcal accumulations large and frequently repeated enemas generally suffice, but the case requires to be long under treatment by careful diet, exercise, electricity or massage, to restore the bowel to its former power.

## INTUSSUSCEPTION

If one segment, say a few inches, of the intestine slips into the portion immediately adjacent, it forms an *intussusception* or *invagination*. It will be at once seen that this must present from without inwards to the centre of the bowel three layers of bowel wall, of which the innermost may be called the *entering* layer; the outermost, the *receiving* layer or *sheath;* and the portion joining these two, the *middle* layer. The process of intussusception may continue, so that more and more bowel is involved, and this usually takes place by the entering and middle layer moving in uniformly together, and, as it were, dragging in the outer layer after them. In this way, as more of the entering layer disappears into the intussusception, the middle layer increases at the expense of the outer layer. The inner bend, between the entering and middle layers, remains always the same, the most advanced portion of the intussusception; the outer bend, between the middle and outer layers, is constantly shifting. It is clear that any portion of bowel might slip into a segment above, forming an *ascending* intussusception, or into the bowel below, forming a *descending* intussusception. It is with the latter that we practically always have to do.

Intussusceptions occur at any part of the bowel, and have received names accordingly; thus those of the small intestine are called *enteric*, those of the large intestine *colic* or *rectal*. But at the point of junction with the ileum and the colon two varieties occur—(1) the *ileo-cæcal*, in which the ileum and cæcum pass into the ascending colon, the ileo-cæcal valve forming the most advanced point, the ileum the entering layer, and the cæcum the most advanced part of the middle layer; (2) the *ileo-colic*, in which the lowest part of the ileum is inverted through the ileo-cæcal valve—that is, an enteric intussusception continued into the colon. Of the different forms the ileo-colic is the rarest, and the ileo-cæcal is the most common, forming nearly half of all cases.

Very important changes, dependent on the anatomical relations of the intestines, ensue upon an intussusception. The intussusception, if at all extensive, forms a thick cylindrical swelling, partly from containing three layers of bowel all round instead of one, partly on account of the congestion and œdema to be presently explained. From the mesenteric connections of the bowel this cylinder has a curved shape, since the vessels which supply the inner and middle layers are of the same length as those supplying the receiving layer, and yet have not only to reach the border of the intussusception, but to go into its interior between the inner and middle layers, so that they drag upon the upper end of that part of the bowel. As the intussusception increases it moves farther along the gut, and the internal cylinder of an ileo-cæcal intussusception may even reach the rectum and project from the anus. At the same time the tumour becomes larger. The disposition of the vessels just described leads to their compression and strangulation, and consequently to congestion and œdema of the walls of the intussusception, and even to hæmorrhage from the mucous surface, and the discharge of blood *per rectum*, an occurrence of the greatest value in diagnosis. If the case is not quickly fatal, inflammatory changes ensue in the layers of the bowel, binding them together, and interfering both with the further progress and with the reduction of the intussusception; and, lastly, from the strangulation of the blood supply to the entering and middle layers, these may become gangrenous, slough off, and be discharged *per rectum*. If this has been preceded by the secure adhesive union of the entering layer to the angle between the outer and middle layers, the canal of the bowel is practically restored, and an actual cure may be the result, though this is very rare; if the union is imperfect, the detachment of the inner cylinder is followed by a fatal extravasation.

**Ætiology.**—The cause of intussusception is obscure in the majority of cases. Sometimes it has arisen after strains or direct injuries, or after unsuitable ingesta, or in connection with diarrhœa. Intestinal polypi and carcinomatous tumours have sometimes seemed to favour its occurrence, and in Henoch's purpura it appears to be caused by hæmorrhage into the intestinal wall. It may happen at all ages, but is much more frequent in children; and it affects males more often than females in early life, though the difference between the sexes is not so great in adults. Little that is definite can be said as to the immediate mechanism of intussusception, except that it is due to an irregular peristaltic action.

**Symptoms.**—The onset of an acute intussusception is not unlike that of strangulation by bands—that is, the patient is rather suddenly seized with pain, which is more or less constant, though aggravated from time to time, and griping in character. In the baby, the onset is indicated by screaming. Nausea and vomiting also occur, but constipation is not generally present at first; on the contrary, the bowels are usually moved, and either thin fæces, or (what is especially characteristic of intussusception) *blood* with or without mucus, is passed. Indeed, blood is passed *per rectum* in four-fifths of the acute cases; and often a certain amount of tenesmus is present. The abdomen is not always much swollen, but an examination reveals generally another characteristic feature—the presence of the *tumour* which results from the intussusception. Its position is, of course, related to the site of the lesion; in the more ordinary ileo-cæcal form it is at first situate in the right flank, but as the intussusception increases it is felt in the umbilical region, and is generally oval, cylindrical, or sausage-shaped, lying transversely across the abdomen above the umbilicus. Subsequently it passes into the left flank and left iliac fossa, and ultimately can be felt by the finger in the rectum, or actually projects from the anus. Sometimes there is complete constipation, with much distension, and fæculent vomiting; at others collapse sets in rapidly, and death takes place in twenty-four hours, or from two to five or six days. Death is especially rapid in quite young infants.

But the symptoms are not always so acute; indeed, an intussusception may exist for weeks or even months. In these more chronic cases the extent of bowel involved is generally less, and the canal is not completely obstructed. The bowels may thus be opened, though blood is passed at the same time in about half the cases. The patient suffers from paroxysmal griping pains, not necessarily of great severity. The abdomen is flaccid, and the tumour presents an important characteristic, namely, a varying consistence, so that it hardens simultaneously with the griping pains, but soon becomes soft, and even imperceptible when they subside.

The terminations of the subacute and the chronic cases are various; they may ultimately lead to death by exhaustion, or to complete obstruction with vomiting, constipation, abdominal distension, and visible coils; or they may set up a local peritonitis, followed by the formation of abscess, or by a more general peritonitis; or the intussuscepted portion may separate by sloughing, and so the intestinal canal may be re-established.

**Diagnosis.**—Spasmodic pain, vomiting, the passage of blood *per rectum* and the presence of an oval or elongated tumour which varies in consistence from moment to moment, and lies in the course of the colon, or occupies the rectum, are the chief features of intussusception; but the tumour cannot always be felt, especially in infants with a much-distended abdomen, unless an anæsthetic is given. *Enteritis* and *dysenteric diarrhœa* in children may resemble it, but there is no tumour, and the blood is less often pure or unmixed with mucus.

**Treatment.**—An *acute* intussusception should, as soon as possible, be met by an effort at reduction, and though cases have been successfully treated by injection of fluids into the rectum and colon, it is safer and more certain for the surgeon to perform laparotomy and reduce the intussusception by careful

traction. In the longer-standing cases adhesions may render reduction impossible; and then a portion or the whole of the mass must be resected.

In *chronic* forms when the nature of the lesion is recognised, operation should also be performed.

## HIRSCHSPRUNG'S DISEASE

This is a rare disease, of which the feature is chronic dilatation and hypertrophy of the colon. In many cases the symptoms begin in the first few weeks of life, in others in early childhood, and in others later. There is constipation, obstinate and repeated, the bowels remaining unopened for two or three weeks at a time. The abdomen is distended enormously, so that the pressure on the chest may itself be a danger. Through the thinned abdominal walls the distended coils of the colon may be seen, and generally the front of the abdomen is occupied by a vertical loop, with the angle near the ensiform cartilage, which is formed of the pelvic colon. In addition the transverse colon is often distended, and sometimes the ascending or descending colon. Peristaltic movements are visible in the coils. The child is emaciated, and in the older patients defective nutrition and sallow skin are observed. The disease does not run a rapidly fatal course; but death from intercurrent disease and toxæmia is to be feared. After death the colon is found to be dilated to two or three times its normal diameter, and often much elongated; and in long-standing cases the muscular fibres, especially the circular layer, are much hypertrophied. There are two types of cases, about equally common, according as the dilatation begins immediately above the anus, or above the pelvi-rectal flexure. The most reasonable view of the pathology is to correlate the disease with chronic dilatation of the œsophagus and to regard it as secondary to obstruction (achalasia) of the anus or pelvi-rectal flexure (**40**).

**Treatment.**—Attempts must be made to keep the colon free. Purgative drugs are of little value. In the anal variety the colon may be readily washed out daily through a rectal tube. In the pelvi-rectal variety the treatment is not so easy; but enemas of oil or glycerine may do good. Strychnine in full doses, electricity and abdominal massage may also help. The diet must be of a kind which will leave little residue, such as milk, fish, eggs, and white bread. If all these fail, an operation may be necessary, either a colostomy, or ileocolostomy, or complete resection of the dilated part of the colon.

## INTESTINAL WORMS

The following are the worms more commonly met with in the human alimentary canal:

| | |
|---|---|
| Cestoda, or tapeworms (κεστός, a girdle) | *Tænia solium.*<br>*Tænia saginata* (*mediocanellata*).<br>*Dibothriocephalus latus.* |
| Nematoda, or round worms (νῆμα, a thread) | *Ascaris lumbricoides.*<br>*Oxyuris vermicularis.*<br>*Trichuris trichiura.*<br>*Ancylostoma duodenale.* |

*Trichiella spiralis* also develops in the alimentary canal, but its symptoms mainly result from its infesting the voluntary muscles (*see* p. 912).

It is essential to say something of the life history of each of these worms before dealing with the symptoms which it produces, and the means to be employed for its expulsion.

### TÆNIA SOLIUM

**Anatomy.**—The *Tænia solium* is a flat, ribbon-shaped worm, very narrow at one end, broader at the other, from 10 to 12 feet in length, and divided into a number

of small segments. There is no alimentary tube, but two canals extend the whole length of the animal, constituting the so-called water-vascular system. At the narrow end is a globular swelling, or head, not larger than a pin's head, presenting a central prominence, or proboscis, surrounded by a row of twenty-six hooklets; and four suckers are placed at the sides. Below the head is a narrow portion or neck, where the segments are quite small and thin, but they gradually get broader and larger towards the other extremity. As they become larger these segments acquire sexual characters, and are then called *proglottides;* each one bears male and female organs, the apertures for which are on one edge of each segment, alternately on the one side and the other of the tapeworm. A fully developed *T. solium* may contain about 850 segments, of which only the last 80 to 100 are mature. A mature proglottis measures ½ inch in length by ¼ inch in breadth. The uterus is an elongated cavity running the whole length of the segment, and giving off from seven to ten branches on each side, which again branch freely. The ova measure 0·03 mm., are slightly oval in shape, and have a thick shell, presenting fine, radiating lines, visible under the microscope. The embryo develops while the ovum is still in the uterus.

The tapeworm inhabits the small intestine, being attached firmly to the mucous membrane by its head, while the chain of segments lies partly coiled, along the bowel, as far down as the lower end of the ileum. As the lowest segments become mature they are detached, and are passed with the fæces. The ova may escape during this transit, or subsequently by the decomposition or rupture of the segments, and they thus become scattered on the ground, or on leaves, grass, or elsewhere.

For the further development of the ovum it is essential that it shall be taken into the stomach of an animal; and in the case of the *T. solium* it is the pig that performs this service, swallowing the ova with vegetables, or with the refuse upon which it feeds. Arrived in the stomach of the pig, the shell of the ovum is dissolved by the gastric juice, and the embryo, or *onchosphere*, provided with six hooks, escapes to bore its way into the gastric or intestinal vessels, and thus is carried to the liver, muscles, or other part of the body. In some such situation the embryo remains, and develops into a little globular bladder about the size of a pea, with which is connected, by a narrow, segmented neck, a head with hooklets, suckers, and proboscis, precisely like that of the complete tænia. The head and neck, however, are usually retracted or inverted into the centre of the little cyst. These cysts occur in great numbers in the muscles of the pig, and the flesh so affected is described as "measly pork"; and they are seen occasionally in man, in the connective tissue, in the eye, and elsewhere, and are known as the *Cysticercus telæ cellulosæ*. In those situations they can develop no further, and in course of time perish; but when the flesh containing them is eaten by man or a carnivorous animal, the head and neck are extended from the globular cyst, the cyst is dissolved in the stomach of the host, the head attaches itself by its suckers to the alimentary mucous membrane, and the segments of the tænia successively grow upon it until a complete tapeworm (*strobila*) is formed.

The **Symptoms** and **Treatment** are the same as those of *Tænia saginata*.

### TÆNIA SAGINATA

This is the tapeworm most commonly met with in England. Its head is provided with four suckers, but is without proboscis or hooklets. In length the animals may attain 5 or 6 yards, and the segments number from 1,200 to 1,300. The complete development of the sexual organs occurs about the 600th segment; the last 150 to 200 are ripe proglottides. The mature segment measures ¾ inch in length by ⅓ inch in width, and the uterus extends the whole length of the segment, giving off on each side from twenty-five to thirty lateral branches, which divide at their extremities. The eggs are only a little larger than those of

*T. solium*, and have the same shape. The cysticercus is commonly found in beef or veal, and not in pork. The mature segments frequently find their way out of the anus, independently of the act of defæcation.

**Symptoms.**—The presence of the worm may cause no symptoms at all, and it may only be recognised by the discovery of segments in the motions. Sometimes disagreeable sensations in the abdomen are described, or gnawing or colicky pains, irregularity of the bowels, and deficient or voracious appetite; itching at the nose or at the anus, salivation, and vomiting also occur. More remote symptoms are giddiness, faintness, and languor; headache, mental disturbance, depression, and even fits, either hysterical or epileptic in character. These are more likely to be aggravated in persons of hypochondriacal or hysterical tendencies. It is obvious that there is nothing pathognomonic in these symptoms; they can only give rise to a suspicion, which must be confirmed by the appearance of the segments. These the physician should himself see, since they may be simulated by fragments of mucus or half-digested food. It may be desirable in some cases to give a purgative to bring away segments.

**Treatment.**—This should never be undertaken unless the presence of a tapeworm is absolutely certain. The method of treatment is the administration of a drug, which is fatal to the worm, and the subsequent removal of the worm by a purgative. In order that the former drug, or anthelmintic, may come into full contact with the worm, it is desirable to have the stomach and intestines as empty as possible. In most cases it is sufficient for the patient to have no food after six or seven in the evening, and to take the anthelmintic before breakfast the next morning. If the bowels have been previously confined, they may be cleared by a dose of magnesium or sodium sulphate the day before the remedy is given. In either case, the anthelmintic should be followed in three or four hours by a dose of calomel, compound rhubarb powder, or castor oil. Several drugs will destroy the tapeworm; the most commonly used in England is the rhizome of filix mas, or male fern, of which the liquid extract may be given in a dose of 1 to 1½ drachms suspended in mucilage. It should be noted that fatal results have occurred when the castor oil has been given at the same time as the male fern; apparently the oil dissolves some toxic constituent of the fern, and thus allows its absorption by the alimentary tract. Alkalies, for a similar reason, are undesirable during anthelmintic treatment.

Other remedies are kousso in the form of infusion; oil of turpentine, from ½ to 2 ounces, which should be followed by a purgative to ensure its not being absorbed; decoction of pomegranate root bark in three or four doses of 1 to 2 ounces each every half-hour; tannate of pelletierine, 8 to 12 grains in capsule; and kamala powder, 1 to 3 drachms in wine or water. Carbon tetrachloride 1 drachm, followed by a saline purgative, is very useful.

The dead worm must be looked for in the motions which follow this treatment by mixing them with water, stirring, and pouring off the upper portions from time to time. The cure cannot be considered complete unless the head of the worm is found, for the worm may break at the neck, and if the head remains attached to the bowel, it will give rise to fresh segments, and ultimately to a complete tapeworm. In this case, a period of almost exactly three months elapses before the mature segments again appear in the fæces; and as it is obvious that the head may elude even a very careful search, it is quite as well not to repeat the treatment forthwith, but to wait until the reappearance of segments conclusively shows that there is still a worm in the bowel.

### DIBOTHRIOCEPHALUS LATUS

This worm is much larger and longer than either of the preceding, measuring from 17 to 26 feet in length. The head is elongated, and presents only two suckers in the form of long grooves (*βόθρος*, a trench). The segments are about

3,000 in number; they measure in the middle ½ inch broad, and only $\frac{1}{7}$ inch in length; but lower down they become more square in shape. The uterus is unbranched, but is bent several times upon itself. The eggs measure 0·07 mm. in length, and have a lid at one end. The portions of the tapeworm that are detached are often several feet in length. The life history is different from that of the tænia, since two intermediate hosts are necessary, firstly, a certain species of fresh-water crustacea, and, afterwards, either a perch, trout or pike. The onchospheres are provided with six hooklets and numerous cilia; by means of the last they swim about freely. There is no cystic stage. *Dibothriocephalus latus* is found especially in Switzerland and Central Europe.

**Symptoms.**—In contrast with the tæniæ, *D. latus* may produce an *anæmia*, which may present all the blood characteristics of pernicious anæmia. Thus there is a great diminution of red blood corpuscles, a colour index above unity, and the presence of poikilocytes, megalocytes, megaloblasts, normoblasts, and corpuscles showing polychromasia and basophil stippling. The anæmia is the result of absorption of the products of decomposition of the parasite, producing hæmolysis, and acting upon the bone marrow.

**Treatment.**—This is the same as is employed for the tæniæ.

### ASCARIS LUMBRICOIDES

**Anatomy.**—In shape and size this resembles the ordinary garden worm (*lumbricus*), but the structure is entirely different; it is pink, cylindrical, and tapering at each end. The mouth is at one extremity, and is surrounded by three tubercles or lips provided with fine teeth; and it communicates with an intestine running the whole length of the animal. The male ascaris is about 10 inches in length; it is seldom met with. The female is from 12 to 16 inches in length, and it has been estimated that the organs of generation can contain at one time sixty millions of eggs. These measure 70$\mu$ in length by 60$\mu$ in breadth. They have a dirty brown colour and are nodulated on the surface from the presence of an albuminous substance deposited outside the shell. They are found in the fæces of the human host. The egg develops within a month into a larva, which remains inside the egg-shell. The latter is thick, so that it withstands partial drying. The eggs may be deposited on vegetables manured with human excrement, and so they may gain entrance to the body. There is no intermediate host. The digestive juices set the larvæ free, and they pass to the liver and from there by the blood stream to the heart and lungs. They pass into the air-sacs, causing hæmorrhage, and then up the bronchi, when they are swallowed a second time, and they gradually attain adult form.

They have a curious tendency to insert themselves into apertures, or ring-like bodies, that may have been swallowed, such as the shanks of buttons; they have been found blocking the common bile duct, the glottis, or the nasal passages; and occasionally they occur in abscesses in the groin or in some part of the abdomen, about which it is not always easy to say whether the inflammation has been set up by the worm or by some other cause. The number of ascarides which may be present in the same individual is very variable; there may be only one, often there are only two or three, but sometimes they exist in hundreds.

**Symptoms.**—These are not very different from those set up by tapeworms. On the other hand, there may be none. Nausea, foul breath, irregular appetite, itching of the nose, or abdominal pain may be present. In other cases there may be reflex symptoms, such as fits, choreic or convulsive movements, or mental disturbance. But the parasite may lead to more serious troubles, such as jaundice, by obstructing the bile duct, or suffocation, by entering into the larynx; and occasionally the worms have formed a convoluted mass large enough to cause intestinal obstruction.

**Diagnosis.**—Here also the diagnosis depends on the appearance of the

worm or the discovery of its ova in the fæces. If a worm is discharged through the anus, or by vomiting, it is as well to treat the case as if others existed. Even when several have been expelled by treatment there may be others left behind; and this may be shown by the detection of the ova in the fæces. As specimens of the garden worm may be brought to the physician and passed off as ascarides, the following differences should be noted: the earthworm is redder in colour, it is less tapering at its extremities, it is provided with bristles along the sides to aid its progression, and its mouth is a short transverse fissure on the under-surface of the rounded head, and the worm is segmented.

**Treatment.**—The best treatment for the *Ascaris lumbricoides* is the administration internally of santonin, the active principle of worm seed (santonica). It is tasteless, and can be taken as a powder mixed with sugar, or simply placed on bread-and-butter, or suspended in milk. The dose for a child is 2 or 3 grains, for an adult from 4 to 6 grains; it should be taken on three or four successive mornings, and followed by a calomel purge, or a dose of compound rhubarb powder. Santonin sometimes affects the vision, so that objects appear green, yellow, or blue; or it may cause tenesmus, or hæmorrhage from the bowels. Severe nervous symptoms, convulsions, and collapse have followed large doses. The urine is always coloured bright yellow, and is then turned red by the addition of an alkali. Carbon tetrachloride (1 drachm), followed by a saline purge, is extraordinarily efficacious. As many as eighty worms are recorded as having been expelled after a single dose.

### OXYURIS VERMICULARIS

This, the threadworm, is very much smaller than the preceding. The female is about $\frac{1}{2}$ inch long, the male only $\frac{1}{6}$ inch. It has a mouth and a complete alimentary canal, and the uterus of the female develops an enormous quantity of ova, which measure 50$\mu$ by 23$\mu$, are elongated, curved, provided with an operculum, and contain the embryo already formed. The adult threadworms occur only in the large intestine, to a great extent in the rectum, where they are often matted together in balls, but also in the cæcum. There is, however, no new generation *in situ*, but the eggs must be first taken into the stomach of the host, whether it be the same individual or another; and infection probably takes place from child to child, the eggs drying on the clothes or on the skin and hair about the anus, and being conveyed by the fingers in scratching or otherwise. And as the eggs must also be present in the fæces of those affected, they may sometimes, from imperfect sanitary arrangements, get into drinking water, and be carried thereby to other people. The embryos are then set free in the upper part of the alimentary canal, and reach their full development in the cæcum, whence they generally move down into the rectum.

**Symptoms.**—These are mainly local, and due to the presence of the parasite in the rectum. The chief symptom is heat or itching at the anus, and this is worse at night, when the patient gets into bed, or shortly before this. There may be at the same time irritability of the bladder, with frequent micturition; or tenesmus, prolapsus ani, or excessive secretion of mucus; and in girls the worms may creep into the vagina, and set up irritation at the vulva, or cause a vaginal discharge.

**Treatment.**—The use of purgatives, such as calomel, scammony, and jalap, will, of course, bring away some worms; but the object of treatment should be to kill them *in situ*. So far as the rectum and lower bowel are concerned, this may be effected by astringent enemas, such as infusion of quassia (1 : 100), with or without some solution of perchloride of iron, solution of alum (7 or 10 grains to the ounce), of tannin, of common salt, or of lime. The rectum should be cleared by a warm-water enema, and 5 or 6 ounces of the astringent should be injected and kept in for some time. This should be repeated two or three times

a week for two or three weeks. To destroy the worms resident in the cæcum it has been recommended to give saline purges frequently, or large doses of infusion of gentian or quassia internally. The itching at the anus is relieved by the application of unguentum hydrargyri. The constant application of a mercurial ointment (ung. hyd. nit.) for six weeks has also been recommended to prevent reinfection by the freshly deposited ova.

### TRICHURIS TRICHIURA (*Trichocephalus dispar, Whipworm*)

This nematode worm measures 1½ to 2 inches; in its anterior two-thirds it is extremely fine, like a thread or hair, but the posterior third is thicker. It inhabits the cæcum, but rarely gives rise to clinical symptoms. The eggs, which may be found in the fæces, have a long, oval shape, and measure 50$\mu$ in length by 23$\mu$ in breadth. Infection is direct from food contaminated with human fæces. The egg contains the embryo, which develops slowly and has no free stage.

### ANCYLOSTOMA DUODENALE (*Hookworm*)

This parasite is a small nematode worm, which attaches itself in great numbers to the mucous membrane of the duodenum and jejunum, and causes, among other symptoms, a high degree of anæmia. The disease occurs throughout the tropical and subtropical belts, extending into Japan and Europe. The so-called "Egyptian chlorosis" is due to this parasite. In 1902 it was discovered in England, in a Cornish tin mine, by Boycott and Haldane, though it is probable that the disease had existed there since 1894.

In South America, India and Ceylon an allied species of hookworm—*Necator americanus*—occurs. There are certain distinctive points in the structure of these two nematodes, but in their mode of life and in the symptoms they evoke they are identical.

The female of the *A. duodenale* is ½ inch in length, and the male about ⅓ inch. The eggs, which can be detected in the fæces, are oval in shape and measure from 50$\mu$ to 60$\mu$ in length by 30$\mu$ to 40$\mu$ in breadth. The egg-shell is smooth, thin, and apparent as a single line only, transparent, and showing from four to sixteen cells in the interior. The eggs are hatched outside the human body and produce larvæ, which, while still of microscopic size, are surrounded by a hard coating, and are said to be encapsuled. In this condition they can neither grow larger nor reproduce themselves, until they enter the body of their human host. They do so, as was first shown by Looss, of Cairo, by penetrating the skin, and rarely by being introduced into the mouth. They are found in the subcutaneous connective tissue, and Looss traced them into the lymphatics, thence into the lungs, whence it appears they reach the bronchi, are coughed up through the larynx, and hence by the pharynx reach the stomach and intestines. The heat and moisture of mines, and other places where they are prevalent, are favourable to their growth, and the naked bodies of the workers give ample opportunities for direct invasion. In Porto Rico, Malay States, India and Ceylon, the hookworms infest the coffee, tea and rubber plantations, where the men work in a moist infected earth with naked feet and legs. The absence of proper arrangements for the disposal of sewage contributes in all these instances to the multiplication of the worms.

**Symptoms.**—A few worms may be present in the bowel, and eggs may be found in the fæces, without any other sign or symptom to indicate their presence. But in larger numbers they produce a severe *anæmia*, and where the larvæ are constantly being brought into contact with the skin they produce a characteristic *eruption*. The eruption is called *bunches* by the Cornish miners: it occurs commonly on the forearms and hands, and consists of papules, pustules or furuncles, and urticarial wheals. The anæmia is shown by a gradu-

ally increasing pallor of the face, lips, conjunctivæ, and body generally; puffiness of the face and feet; feebleness and lassitude, with quick, small pulse, palpitation, dyspnœa, and deranged digestion. The anæmia is of a severe secondary type (*see* Anæmia), with a diminution of the hæmoglobin to 40 per cent., a low colour index, a varying increase in the leucocytes, and a marked relative and absolute increase in the eosinophil cells, which may reach from 30 to 50 per cent. of the leucocytes (Boycott and Haldane). Death may occur from intercurrent infections.

**Prognosis.**—The anæmia can be cured, the patient restored to practical health, and the majority of the worms can be expelled from the intestine; but it is extremely difficult to get rid of the last few worms, and eggs may be found in the fæces months and years after recovery.

**Diagnosis.**—When the disease occurs as an epidemic disease among groups of workers underground its recognition is of course easy. The cutaneous eruption, or the dyspnœa, but especially the anæmia, brings the patient under observation; but in Egypt the diagnosis is complicated by the prevalence of another parasitic disease, namely, bilharziasis, which may also cause anæmia. In any case, however, the character of the blood count will be a guide to the kind of anæmia, and the presence of the worm can be determined by finding the eggs in the fæces. A small piece of fæces may be mixed with water on a slide and examined with the microscope; or it may be shaken up with water in a test-tube, when the eggs will fall to the bottom, and the supernatant fluid may be decanted. If the eggs are in small number, they may be separated by Clayton Lane's flotation method. The fæces are disintegrated by vigorous shaking in a special closed centrifuge tube. The supernatant fluid is removed after centrifuging, and then salt solution of specific gravity 1,150 is added. On again centrifuging the eggs float upwards and adhere to a cover-glass which has been fixed on the tube, where they may be examined and counted.

**Treatment.**—Chemically pure carbon tetrachloride is the most effective drug for destroying the worms. One or two administrations by mouth are enough in doses of 10 minims for a child of one year, increasing by 2 minims for each year of life, so that an adult receives from 50 to 80 minims (**41**). Thymol (60 grains), naphthol and oil of chenopodium (maximum dose 25 minims) have also been used and are effective. The dietary must be carefully controlled during treatment and no fatty or alcoholic substance given. Hæmatinics are of little value until the worms have been cleared out; then inorganic forms of iron, together with arsenic, perhaps best given hypodermically, will rapidly improve the condition of the blood.

# DISEASES OF THE LIVER

The liver occupies the right hypochondriac region, under the ribs, and stretches across the upper part of the epigastrium. Normally it can scarcely be felt even in the latter situation, and there only when the abdominal parietes are very thin. Percussion gives dulness (*hepatic dulness*) in the mammary line, from the upper border of the sixth rib to the costal margin; in the middle line there is very slight loss of resonance for 1½ or 2 inches from the base of the ensiform appendix, where the thin left lobe lies over the stomach. When the abdominal parietes are thin the edge of the liver may be perceptible to sight during deep inspiration, as the organ descends for 1½ inches, and the percussion dulness shifts to a corresponding extent. In the axillary line the hepatic dulness begins at the eighth rib, and in the line of the scapular angle at the tenth or eleventh rib.

In disease the organ is often enlarged. It then, as a rule, projects below the costal margin, and can be felt as a definitely resisting mass, different from the

supple part of the abdomen below it, and different from the left hypochondrium. It may reach to the level of the umbilicus, or much lower, its lower margin extending across the abdomen from the right flank to the left costal margin. It presents different degrees of consistence, and alterations of surface, according to the disease affecting it; and the dulness extends to a corresponding degree down into the abdomen, since the organ always lies in front of the hollow viscera, unless there is liquid in the peritoneal cavity, which will enable the liver to fall away from the anterior parietes. From the presence of the intestines behind it, the dulness is not so complete near the free edge as it is higher up, and the percussion must be lightly performed in order to localise it exactly. In the presence of ascites, percussion is useless as a test of the size of the liver.

The liver only encroaches on the chest when the enlargement is (1) localised rather than general, such as that due to carcinoma, hydatid, or abscess—and in these cases the ribs may be bulged outwards so as to enlarge the right costal angle; or (2) when the liver is itself pushed up by something below.

Apparent enlargement of the liver arises from tight-lacing, and from tumours or pleuritic fluid in the chest. The former elongates the organ vertically (*see* p. 488); in the latter the whole liver is displaced. Displacement of the liver downwards, or ptosis, occurs also as a part of *Glénard's disease* (*see* p. 416).

In atrophy of the liver there is a diminution of the percussion dulness, but a similar diminution may be caused by the encroachment of intestines distended with air.

A distended gall bladder may be felt as a globular prominence at the lower border of the hepatic dulness, in the mammary line.

## TEST OF HEPATIC EFFICIENCY

*Lævulose Test.*—This test depends on the fact that when lævulose is absorbed from the alimentary canal and reaches the portal circulation, it is almost entirely taken up by the liver, and so does not reach the systemic circulation. If the liver is diseased, the lævulose is not retained. It passes through into the systemic circulation and causes a rise in blood sugar (*see* p. 563). In performing the test, a dose of 30 to 50 grammes of lævulose is taken by the mouth. The blood sugar is estimated first beforehand, and half an hour, one hour, and two hours later. Normally there may be a very slight rise, which is less than 0·01 per cent. Thus, if the blood sugar is 0·10 per cent., it will not be higher than 0·11 per cent. after an hour. If the liver function is deranged, as may occur in catarrhal jaundice, or in arseno-benzol poisoning, the blood sugar may rise to 0·15 per cent. in half an hour and 0·19 per cent. at the end of an hour, and it then slowly returns to normal (**42**). Simultaneously with the rise in blood sugar the lævulose often appears in the urine, as the kidney very readily excretes it. This test should be compared with that for mild diabetes mellitus (*see* p. 564).

*Glycuronic Acid Test.*—Another test for hepatic efficiency depends on the power of the liver to form glycuronic acid with certain drugs. Five grains of aspirin or sodium salicylate are taken by mouth, or 5 grains of camphor in olive oil are injected subcutaneously. The urine is then collected for twenty-four hours, and of this 20 c.c. are treated with 5 c c. 10 per cent. basic lead acetate solution and filtered. Ten cubic centimetres of the filtrate are heated with 5 c.c. conc. HCl and 1 c.c. of 1 per cent. solution of naphtho-resorcin for fifteen minutes on a water-bath. After cooling it is shaken with 2 c.c. ether. The presence of glycuronic acid, indicating a normal hepatic function, is shown by the ether having a purple tinge. If red the reaction is negative.

Gross inefficiency of hepatic function may be shown by the occurrence of jaundice or ascites, which will now be described. In this connection certain special tests are available, viz. Van den Bergh's tests, Hay's test for bile salts, and the examination of the urine for urobilin (*see* pp. 475, 608).

## JAUNDICE

The term *jaundice* (from *jaune*, yellow), or *icterus*, denotes the circulation in the blood of constituents of bile. In the past the term has been used solely for the yellow staining of the skin and mucous membranes by bile pigment, but it is now used in a wider sense and includes those cases where bile salts appear in the blood without bile pigment—one form of *dissociated icterus*, and cases where the bile pigment is present in such small quantity that no colour is produced (*latent icterus*).

**Symptoms.**—Owing to the colour of bile pigment the skin has a more or less deep yellow tinge, the conjunctivæ are yellow, and the visible mucous membranes have their natural colour obviously modified by the yellow tint. In long-standing cases the colour of the skin becomes deeper, and finally of a greenish or olive-brown tint. This, formerly distinguished by the name of *green* or *black jaundice*, is due to the gradual conversion in chronic cases of bilirubin, the yellow pigment of the bile, into biliverdin by oxidation in the skin. The yellow colour must be distinguished from other changes of colour in disease, such as the lemon yellow tinge of cases of pernicious anæmia, the sallow tint of malarial cachexia, the brown colour of Addison's disease, and the yellow colour due to the pigment *carrotin*, which appears in the skin after eating large amounts of green vegetables and carrots. The colour can be generally well recognised in the conjunctiva, but in some people small masses of subconjunctival fat give a tint which is not very unlike it.

The colour of the *urine* is at the same time altered, from the presence of the biliary pigment. In small quantity this gives it a bright saffron colour, which is best seen in any froth which may form on the surface; if there is more the urine becomes brownish yellow, or yellowish brown, or even dark brown like porter. If linen or paper is dipped in the urine, it is stained bright yellow; but the presence of bile pigment can be more certainly proved by the application of chemical tests which will be mentioned presently. Of the other secretions of the body the sweat is sometimes tinged yellow. The milk of nursing women, tears, and saliva are rarely stained. The cerebro-spinal fluid is not usually stained. At the commencement and at the end of an attack of jaundice the urine often contains urobilin, a derivative of bile pigment, but none of the bile pigment itself, and there is a group of cases in which bile pigment is absent from the urine (*see* Acholuric Jaundice).

In most cases of jaundice the *fæces* are altered in colour, becoming whitish or clay-coloured; this is due to the presence of an excess of fat and to absence of urobilin in those cases of jaundice where the bile is unable to find its way into the duodenum. Bile is known to have some share in the digestion of fat, especially in its absorption, and is thought to have a power of preventing putrefaction in the intestinal contents, and to stimulate the muscular fibres of the intestinal wall. Accordingly constipation is frequent, though by no means invariable. When diarrhœa occurs, it has been attributed to the irritation of the putrescent fæces.

Other symptoms are often present in jaundice, which are due to the circulation in the blood of bile salts. These are: (1) *bradycardia;* the *pulse* becomes slowed to fifty or forty per minute; (2) *itching;* it may be so intense that sleep is rendered impossible, and blood crusts, papules, or wheals of urticaria are produced by the incessant scratching.

Some patients have a bitter taste in the mouth, and digestive disturbances are frequent. Hæmorrhages take place under the skin or from the mucous surfaces, and the bleeding from wounds is not readily checked; the coagulation time of the blood is prolonged. In some cases, serious cerebral symptoms arise, such as delirium, convulsions, and coma; but these are probably always due to the presence in the blood of other poisons than those contained in the bile. Yellow vision (*xanthopsia*) is sometimes observed. A disease of the skin named

*xanthelasma* or *xanthoma* occurs in some cases of long-standing chronic jaundice (*see* Diseases of the Skin).

**Clinical Tests for Jaundice.**—*Van den Bergh's Test for Bile Pigment in Serum.*—Besides indicating the presence of bile pigment in serum, this test differentiates between the pigment retained in the blood from obstruction of the bile passages and that due to hæmolysis. Ehrlich's diazo reagent is used. This consists of (*a*) sulphanilic acid 1 gramme, conc. HCl 15 c.c., distilled water 1,000 c.c.; and (*b*) sodium nitrite 0·5 gramme, distilled water 100 c.c. The two are mixed just before use in the proportion of 25 c.c. of (*a*) to 0·75 c.c. of (*b*). *Direct Reaction.*—One cubic centimetre of serum obtained from blood, allowed to clot on standing, and 1 c.c. of the reagent are mixed. A bluish-violet colour reaction begins immediately, and is at its maximum in ten to thirty seconds. This indicates obstructive hepatic jaundice. If there is no direct reaction, a delayed reaction may occur, beginning after one minute. This is due to hæmolytic or toxic and infective jaundice. A biphasic reaction, which begins at once, but only slowly develops, indicates the presence of a mixed type of jaundice. *Indirect Reaction.*—If no direct reaction is given, 2 c.c. of 96 per cent. alcohol are added to 1 c.c. serum and centrifugalised. To 1 c.c. of the clear liquid 0·5 c.c. of alcohol and 0·25 c.c. of the reagent are added. A violet-red colour, which is of maximal intensity almost at once, is obtained, and this indicates hæmolytic or toxic and infective jaundice. The tests may be carried out quantitatively (**43**).

*Tests for Bile Pigment in the Urine.*—The essential feature of these tests is the production of a green colour by the oxidation of yellow bilirubin into green biliverdin; in some processes other tints are temporarily developed. *Gmelin's test* may be carried out as follows: A few drops of urine are placed upon a white plate, and a little strong nitric acid is dropped close by, and then the two fluids are gently run into one another. At the line of contact the colour of the urine changes, becoming green, blue, violet, red, and lastly yellow or brown. Ryffel has devised a modification of this test capable of detecting very small quantities of bile pigment. The urine is saturated with ammonium chloride and made alkaline with ammonia. Ammonium urate is thus precipitated and carries down any bile pigment with it. The urate is filtered off, and nitric acid is added to the residue on the filter paper. A green colour indicates bile pigment.

*Hay's Test for Bile Salts in Urine* is carried out by sprinkling powdered sulphur on to the surface of fresh urine. If bile salts are present the surface tension is lowered and the sulphur sinks. This test, which has been much discredited in the past, is now regarded as reliable; but the test is also given if large amounts of sandal-wood oil, copaiba, cubebs and turpentine have been taken by mouth. A test for the presence of bile salts in the intestine, depending on the fact that they facilitate absorption of fat, has also been described (**44**).

**Pathology of Jaundice.**—Modern views on the pathology of jaundice have been originated as the result of Van den Bergh's test (**45**) and of work by French observers (**44**). Bilirubin exists in two forms: (1) as finally elaborated by the liver cells and excreted into the bile passages. This gives Van den Bergh's direct reaction; (2) before final elaboration in some closely related form, which gives Van den Bergh's indirect reaction. That the liver is not necessary for the production of bilirubin is shown by the fact that this pigment is formed locally from old blood clots anywhere in the body. However, the preliminary stage in the formation of the bilirubin which exists in bile probably takes place by means of the *reticulo-endothelial system*, which consists of certain cells in the sinuses of the spleen and similar cells, known as Kupffer cells, in the portal capillaries of the liver. This bilirubin is subsequently taken up by the liver cells, and after further modification is excreted into the bile capillaries, where it becomes the bilirubin of the bile. If something goes wrong with the formation by the reticulo-endothelial system, or blood destruction in the body is excessive so that the manufacture of bilirubin by these cells is greatly increased, pigment will not all be taken up

by the liver and excreted as bile, but will gain entrance to the circulation. This result may also occur if the liver cells are damaged so that the final elaboration of the pigment into the bilirubin of the bile is interfered with. There are thus three types of jaundice:

1. *Hæmolytic Jaundice,* a group which should also include pernicious anæmia, since in this disease, owing to excessive blood destruction, bilirubin is present in the serum and is responsible for the characteristic yellow colour of many patients.

2. *Toxic and Infective Hepatic Jaundice* from destruction of liver cells. The agents producing this variety of jaundice are—(*a*) *chemical poisons,* such as tetrachlorethane (*i.e.,* dope for aeroplanes), T.N.T. chloroform, phosphorus, toluylene-diamine, nitrobenzene, arsenobenzol derivatives, arseniuretted hydrogen, mushrooms, snake venom, etc.; (*b*) *bacterial poisons,* such as occur in relapsing fever, malaria, the enteric group, typhus, pneumonia, influenza, syphilis, yellow fever, septicæmia, spirochætosis ictero-hæmorrhagica, catarrhal jaundice, etc.

3. *Obstructive Hepatic Jaundice.*—In this group there is some obvious obstruction of the bile ducts: (1) gall stones and inspissated bile, very rarely hydatids, liver flukes, and foreign bodies from the intestinal canal, including *Ascaris lumbricoides;* (2) stricture or obliteration of the duct from congenital defect or atresia, or from former ulceration of the duodenum or of the bile duct itself: catarrhal or inflammatory swelling or carcinoma of the wall of the bile duct: spasm of the duct; (3) compression from the outside by glands in the portal fissure: by tumours of the head of the pancreas, stomach, colon, kidneys, omentum, ovaries, or uterus: by abscess or hydatid of the liver: by an abdominal aneurysm, accumulated fæces, or pregnant uterus. In many cases of obstructive hepatic jaundice the bile distends the gall bladder and the bile ducts, and then passes into the lymphatics and blood vessels, circulates in the latter, and gives the characteristic tinge to the skin and other parts. An interesting fact in the secretion of the bile makes it likely that a complete obstruction is not necessary—that is, that the bile is secreted under very low pressure, such that in guinea-pigs a pressure of 20 cm. of water will force the secreted bile back into the circulation. Where there is a complete obstruction, as from a gall stone in the common duct, or a tumour pressing upon it, the bile is unable to reach the intestines, and the fæces, as already stated, are white or clay-coloured. When the obstruction is relieved the symptoms disappear.

While the first and third of these types of jaundice are clearly differentiated, since they give respectively the indirect and direct Van den Bergh reactions, members of the second group commonly give a biphasic reaction. In the past the jaundice in these cases has been looked upon as due to a cholangitis affecting the smaller bile ducts and therefore obstructive; but this view has been attacked by Brulé, who points out that sometimes the bile salts alone are retained in the circulation and at other times the bile pigments alone—these are the two types of *dissociated jaundice*—so that the liver cells themselves must be implicated (**44**). He regards the jaundice due to cirrhosis, concussion or congestion in heart disease as belonging to the same group.

*Latent Jaundice* means that there is bilirubin in the blood, but not enough to produce a typical yellow colour or to cause the kidney to excrete bilirubin. In such cases the urine commonly contains plenty of urobilin, which is probably a transformation product of bilirubin by the tissues. However, it must be noted that urobilinuria is commonly absent when bilirubinuria is marked. Latent jaundice may be present in heart disease and may suggest the onset of failure. Its presence will differentiate pernicious anæmia from secondary anæmia due to hæmorrhage. It may indicate damage of the liver after treatment with arseno-benzol, and in various infective diseases. It is very commonly present in cirrhosis.

**Icterus Neonatorum.**—Jaundice is not infrequent in new-born children. In the majority of cases it lasts only a few days or a week or two, and is unaccompanied by any symptoms. It is probably due to hæmolysis, since the blood of the new-born infant contains a high proportion of red cells, the blood count diminishing after birth, and the serum gives Van den Bergh's indirect test (**53**). The yellow colour affects first the face and trunk, and later the limbs; and is recognised by pressing the reddened skin, so as to exclude the blood colour. The fæces are generally normal, and the urine is untinted by bile pigment, except in the severer cases. In cases in which death has occurred from accident or otherwise, the organs and tissues are found to be stained with bile, including the central parts of the brain (corpus striatum and thalamus), but not the cortex of the brain, nor the liver, spleen or kidneys. The patients recover, and no treatment is required.

Less frequently occur cases of catarrhal jaundice, infective and epidemic infective jaundice, and the more serious cases of familial jaundice, cases due to congenital obstruction of the bile duct, septic infection through the umbilical cord, and syphilitic disease of the liver.

## ASCITES

By this term is meant the presence of serous fluid in the peritoneal cavity. Like other effusions into the serous cavities, it is commonly alkaline, of a pale straw colour, of specific gravity 1,015 to 1,018, highly albuminous, and containing chlorides. It arises (1) from obstruction of the portal circulation, either in the trunk of the portal vein, or in its distribution in the liver; (2) as a result of diseases of the peritoneum; and (3) as a part of the general dropsy of renal disease, or cardiac disease.

The portal vein trunk may be obstructed by the pressure of tumours and enlarged glands in the portal fissure, by carcinoma, abscess, or hydatid in the liver itself, and by coagulation of blood in its interior (*thrombosis, pylephlebitis*). In the liver the chief cause of portal obstruction is the compression of the interlobular veins by the fibrous overgrowth of cirrhosis. It is thought by some that portal obstruction is not an adequate cause of ascites, which they attribute to toxins produced in the diseased liver, or absorbed from the intestine and undestroyed by the liver. Another cause of portal obstruction is perihepatitis. A third kind of obstruction is formed by the different forms of cardiac and lung disease, in which the right side of the heart is dilated and the passage of the blood through the chest is impeded (*see* pp. 232, 337).

The peritoneal diseases causing ascites are—acute and chronic peritonitis, tuberculous peritonitis, and carcinoma of the peritoneum.

In Bright's disease the peritoneum is the seat of effusion, in common with the other serous cavities.

The **Physical Signs** of ascites must be carefully considered, as it is not impossible to confound it with other conditions. The abdomen, of course, enlarges, and in the early stages of a considerable ascites it is generally tense, and the form tends to be globular, with a decided prominence in a forward direction. Later the walls of the abdomen become stretched, and as the patient lies in bed the fluid gravitates backwards in each flank, and gives a broader and flatter shape to the belly. The liquid then poured out may amount to 3, 4, or 5 gallons, and the abdomen becomes proportionately enlarged so that it may measure from 40 to 42 inches or more in circumference. The presence of fluid is detected by three methods of examination—*percussion, fluctuation,* and *displacement.*

*Percussion.*—Normally the surface of the abdomen is resonant from the air contained in the stomach and intestines; but when fluid is poured out this collects at first in the flanks and hypogastric region, so as to give a dull note to percussion in these parts, while the centre of the abdomen remains resonant.

As the fluid increases the dulness encroaches more and more from the sides and hypogastrium upon the centre, and at length only a limited area remains resonant—namely, that which includes the umbilical and the left hypochondriac regions. If in either of these stages the patient be turned upon one side and again percussed, it will be found that the anterior and central regions have become dull, and the flank, which is now uppermost, gives a resonant note. This is due to the gravitation of the fluid to the lowest part and the floating of the air-containing bowel to the highest, and this occurrence is the most conclusive proof of the presence of fluid in the peritoneum. Occasionally, however, the abdomen is entirely dull, when the mesentery is so short, or the fluid so abundant, or the viscera are matted down by chronic peritonitis in such a way, that the intestines cannot float to the uppermost part. Then also this test by change of position fails to give the desired information.

*Fluctuation* is obtained by laying one hand on one side of the abdomen and sharply tapping or flipping the other side with the finger. The applied hand then feels the transmission of a wave across the abdomen. This is a less certain sign than the former. Very fat abdominal walls may transmit a wave without the presence of fluid, and to prevent this the edge of the hand, or of a book or card, should be pressed on the centre of the abdomen while fluctuation is tried.

The method of *displacement* has only a limited application, but it provides in some cases earlier evidence of ascites than either percussion or fluctuation. If in a case of ascites the liver is enlarged, it sinks in the fluid, and a small quantity of fluid lies between its anterior surface and the abdominal wall. By placing the fingers on the abdomen at this spot, and suddenly pressing them in, the fluid is displaced, and the surface of the liver may be felt. This is a proof of the presence of fluid, since, if there were none, the liver would be in close apposition with the anterior abdominal wall.

Ascites is, however, sometimes simulated by one or other of the different kinds of cyst which may occur in connection with the abdominal or pelvic viscera, by a pregnant uterus, or by a distended urinary bladder. These cysts are ovarian, parovarian, hydatid, and renal cysts. They are excluded if the percussion test is successful; on the other hand, they may give the fluctuation test; and if the whole surface is dull there may be some difficulty in distinguishing between one of these and an ascites in which the intestines are bound down. An operation for ovariotomy has several times been attempted, at which the case has been proved to be one of ascites. An ovarian cyst is chiefly distinguished by the abdomen being dull in front and resonant in the flanks, into which position the intestines are pressed by the cyst, and by the swelling due to the cyst, which begins on one side, though later on it is central. Not infrequently also the outline of the cyst can be recognised at the uppermost part, especially if looked for during the movements of respiration.

**Chylous and Chyliform Ascites.**—In exceptional cases the fluid contained within the peritoneal cavity is opalescent and milky instead of being a clear serum.

Sometimes this is due to the extravasation of chyle from the thoracic duct or lacteal vessels into the peritoneum, either from rupture or from obstruction of the vessels by disease or by the presence of parasites (*see* Filariasis). This is true *chylous ascites*. The fluid is then of a yellowish-white colour, has a specific gravity of 1,012 or more, and an odour dependent upon the food which is being taken. On standing fat separates and forms a creamy layer on the surface; fatty globules are seen under the microscope, but few cellular elements. A clot of fibrin may form in it after removal from the body.

In another group of cases, *chyliform ascites* or *pseudo-chylous ascites*, the fluid is pure milky white, of a specific gravity less than 1,012. The amount of fat is variable: it may form a creamy layer on the surface, or there may be only traces of it; but in any case the opalescence is not due to the fat, but to minute

granules of a compound of lecithin and globulin, held in suspension by inorganic salts. Microscopically cellular elements containing fat may be present. The presence of lecithin enables this fluid to resist putrefaction for a long time.

These chyliform effusions are not distinctive of any one pathological condition; but in a large majority of the cases there has been found either carcinoma, or tubercle, or cirrhosis of the liver, or chronic nephritis; and generally the prognosis is bad.

Both chylous and chyliform liquid may occur simultaneously in other serous cavities; and there is no means of knowing, until paracentesis has been performed on one or other cavity, whether an effusion is of the kind under discussion.

## CIRCULATORY CHANGES IN THE LIVER

### CONGESTION

The *nutmeg liver*, which has been already described (*see* p. 337) as one of the results of valvular disease of the heart, is an extreme form of passive congestion. In earlier stages of the same change the organ is simply engorged, being larger than normal, and dark red in colour, with obvious distension of the intralobular veins. It causes fulness and discomfort in the right hypochondrium, and ultimately ascites and slight jaundice. Hepatic venous pulsation is occasionally associated with it.

The term *congestion of the liver* has been applied in the case of people who habitually over-eat and drink freely and take no exercise. They complain of feeling out of sorts; there is a sense of fulness and weight in the lower part of the chest, a furred tongue, constipation, and perhaps slight jaundice. Such people improve with restriction of diet, purgatives and regular exercise. There is no evidence in what way, if any, the liver is affected in such cases.

Acute inflammation of the liver is the result of various intoxications and infections, which lead to enlargement of the organs and some of them to suppuration, such as occurs in tropical abscess (*see* p. 169), and in the abscesses connected with pylephlebitis, suppurative cholangitis, and general pyæmia.

## ABSCESS OF THE LIVER

**Pathology.**—Abscesses of the liver arise under a variety of conditions, but what is common to nearly all of them, except injury, is the introduction of some septic agent by one of three channels: the hepatic artery, the portal vein, or the bile ducts.

In the first case they form part of a general pyæmia, such as results from wound or injury in any part of the body, but especially injuries to the head; they are small in size, and numerous, or at least multiple. They are known as *pyæmic abscesses*.

The portal vein is responsible for a still larger number of cases, the septic agents being carried from lesions within the portal vein area, such as gastric ulcer, appendicitis, pelvic suppurations, and especially ulcerative lesions of the intestine, including tropical dysentery. The abscesses may be single, few, or multiple; and when they are multiple the condition may be spoken of as *portal pyæmia*. Sometimes the portal vein and its branches are filled with broken-down purulent clot, and the walls of the veins are inflamed, constituting *suppurative pylephlebitis*. *Tropical abscess* which is due to dysentery has been already described (*see* p. 169).

Invasion by the bile ducts is chiefly effected in consequence of gall stones ulcerating into them (*suppurative cholangitis*).

Abscesses of the liver vary in size from a pin's head up to that of a hazel-nut; they may contain well-formed pus, or sanious liquid and *débris*, or more bulky

sloughs that have only just been separated. In cases originating in pylephlebitis it may be easy to show that much of the suppuration is in the course of the distribution of the portal vein. The capsule of the liver is frequently inflamed where abscesses approach the surface.

**Symptoms.**—Cases of multiple abscesses in the liver are often very obscure, especially when they form a part of a general illness like pyæmia. There is severe constitutional disturbance, with fever of hectic type, rapid pulse, dry brown or furred tongue, and early prostration. Vomiting is often present, but the action of the bowels is variable: sometimes there is constipation, at others diarrhœa. The liver is mostly enlarged, and in some cases may reach to the level of the umbilicus; it is painful and tender. Jaundice is sometimes, but not necessarily, present; it probably requires the compression by an abscess, or the obstruction by gall stones, of some larger bile duct. The condition of the urine and of the fæces as to the bile pigment will, of course, vary with it. The duration of the illness is from one to several weeks, but the end is certainly fatal.

**Diagnosis.**—This must depend on the fact that the liver is involved in an acute process, with severe general toxæmia, especially if these symptoms are associated with some lesion which can be recognised as the primary cause. The presence of jaundice very much facilitates the diagnosis. Where jaundice is absent, it may have to be distinguished from tropical abscess by the uniform enlargement of the liver.

The **Treatment** must be mainly symptomatic. An attempt must be made to improve the general condition by nourishment, quinine, and stimulants. Opium and local applications, poultices, fomentations, etc., will be required to relieve pain.

## CATARRHAL JAUNDICE

(*Catarrh of the Bile Ducts; Catarrhal Cholangitis*)

This is one of the commonest forms in which jaundice occurs. It is now thought that the disease is most commonly due to an acute hepatitis from some infection carried to the liver by the blood stream, and rarely to infection spreading up the bile ducts from which its name is derived.

**Ætiology.**—Catarrhal jaundice is especially frequent in early life. It may be associated with evidences of gastro-duodenal catarrh. It is usual to associate with catarrhal jaundice the well-known instances of jaundice from fright, the main features of which are, at any rate, similar.

Cases indistinguishable in general characters from catarrhal jaundice have often been observed to occur in epidemics, and have been recorded as *epidemic jaundice*. In many of these children are alone or chiefly attacked; in a smaller number of instances adults suffer most. Neither to the sporadic cases, nor to the epidemic cases, has a definite bacteriology been assigned (*see also* Spirochætosis Ictero-hæmorrhagica).

**Symptoms.**—The patient may have indigestion, weight, pain, or distension of the stomach after food, with, perhaps, occasional sickness for three or four weeks before the jaundice; and in other cases it may occur after unusual indulgence in particular kinds of food; but in very numerous instances the patient knows absolutely nothing of his illness until he himself sees in the looking-glass, or is told by his friends, that his skin is acquiring a yellow tinge. Occasionally the jaundice is preceded by severe pains in the limbs. In catarrhal jaundice the skin and conjunctivæ are of a bright yellow colour; the urine is yellowish brown from bile pigment and contains ketone bodies; the fæces are pale or clay-coloured. The serum usually gives Van den Bergh's direct test (**53**). The temperature is generally normal, and there may be no constitutional disturbance, the patient being able to do his work as usual; but often he is languid, indisposed for exertion, with a bad appetite, and some nausea. There is mostly no pain in

the hepatic region, and not even tenderness ; but both may be present in moderate degree. The liver also is often not at all enlarged, but sometimes its dulness reaches one or two finger-breadths below the margin of the ribs, and the edge may then be felt, as well as the distended gall bladder. The bowels are variable, most often constipated, occasionally loose. The pulse may be unaffected, but it is especially in this form of jaundice that abnormally slow pulses have been recorded.

The illness lasts from two to five or six or more weeks, and the jaundice gradually disappears, the urine becoming normal in colour first, and the skin more slowly recovering.

**Diagnosis.**—It is occasionally possible to make the diagnosis before the onset of jaundice, since enlargement of the liver, which is an early sign, may suggest that functional efficiency tests for the liver should be carried out, and a functional deficiency may be found. The painless, or almost painless, onset of jaundice in a young person previously healthy, or at most suffering some gastric disturbance, as a rule, distinguishes it from the jaundice of *gall stones*, of *carcinoma*, and of *cirrhosis*, the other most common causes. If the jaundice lasts more than five or six weeks, the possibility of one of the above three diseases or of a more general cholangitis should be considered. *Acute yellow atrophy* may begin with a jaundice which is in no respects different from catarrhal jaundice, and which lasts from three to five weeks before the onset of the serious symptoms.

**Prognosis.**—With the above exception, it is entirely favourable.

**Treatment.**—The patient need not be confined to bed, nor even to the house, but should take a light, simple diet, and avoid fats and stimulants ; and a saline laxative should be given if the bowels are actually confined. Sometimes the jaundice passes off quickly with little else, but in many cases recovery appears to be hastened by the internal use of alkaline remedies, especially the bicarbonate of sodium in combination with rhubarb, taraxacum, or calumba. Sodium salicylate and urotropin may also be useful. Carlsbad and Vichy waters are often recommended, the former especially for its laxative action. The flow of bile from the gall bladder may be facilitated by giving magnesium sulphate (*see* p. 500).

## ACUTE YELLOW ATROPHY

In this remarkable disease the liver undergoes a rapid degeneration of its tissues, and diminishes in size to two-thirds, or even one-half, of its normal bulk.

**Etiology.**—It is more common in females than in males, and the majority of patients are under thirty years of age, though it is very rare in children. Indeed, at any age it is a disease of extreme rarity. Its onset is often preceded by severe mental disturbances, and many of the cases have occurred in people who have led a dissipated life, in the subjects of syphilis, and in women who are pregnant. It has also occurred within twenty-four or forty-eight hours of a surgical operation ; this has mostly been an abdominal operation, performed under chloroform. It has recently been found that the toxic jaundice that occurs in poisoning with trinitrotoluene (T.N.T.), tetrachlorethane (aeroplane dope), arseno-benzol derivatives (*see* p. 123), and in cases of spirochætosis ictero-hæmorrhagica, is associated with morbid appearances in the liver that are indistinguishable from acute yellow atrophy, and they must be regarded as some of the causes of this disease. In fact, the present view is that acute yellow atrophy on the one hand and cirrhosis of the liver on the other are different phases of essentially the same process, viz., poisoning of liver cells, in the one case acute, in the other extremely chronic. Further, there is a whole series of cases of *subacute atrophy*, or *multiple nodular hyperplasia*, that bridge the gap between the two. It is possible that some cases called *catarrhal jaundice* are really cases of acute yellow atrophy that recover.

**Morbid Anatomy.**—1. *The Acute Type.*—The liver is very much diminished in size ; it may be only 30 or 28 ounces in weight. It is soft, flaccid, almost like

a bag of fluid, and its capsule, which is wrinkled, appears too large for its contents. On section the liver is of a yellow colour, with patches of rather bright red; or in some parts it is entirely red, in others all yellow. The essential change is a granular and fatty degeneration, by which the hepatic cells are more or less completely destroyed. The necrosis begins in the central zones of the lobule. In the yellow parts of the liver the destruction is less advanced, and some bile-stained cells may still perhaps be found. In the red parts the colour is due to the more complete necrosis of the tissue, by which the vessels are left alone to represent the substance of the liver. Under the microscope one can often see nothing but granules of albuminous matter, fat and pigment, and larger globules of fat. Leucin and tyrosin are also found in the liver, and will spontaneously crystallise on the surface of sections some hours after death. The bile ducts are empty, and not stained by bile pigment; the gall bladder is also empty, or contains a small quantity of viscid grey mucus.

Other organs undergo fatty degeneration, especially the kidneys, in which the secreting cells are granular and fatty, and the heart and muscles. Petechiæ are found under the skin, in the mucous membranes, under the serous membranes, in the kidneys, and other parts.

2. *Subacute Atrophy, or Multiple Nodular Hyperplasia.*—The liver is not so much reduced in size, and regeneration of liver tissue is the chief feature. Adenomatous-looking nodules, yellow in colour, are seen set in a stroma of fibrous tissue. They may be small or large, depending on the amount of regeneration, and microscopically they contain newly formed liver cells and bile ducts, though necrotic cells are also present in abundance. To the naked eye the liver has a curious appearance, the arrangement of the fibrous tissue being similar to cirrhosis, and the yellow nodules resembling in appearance the acute form of the disease.

**Symptoms.**—The symptoms are at first obscure. Often it begins with a jaundice indistinguishable from catarrhal jaundice, or with gastro-intestinal symptoms, such as nausea, vomiting, and irregularity of the bowels; and pains in the hepatic region may occur comparatively early. These symptoms may last two or three weeks, or much longer when the more characteristic features develop. These consist of marked cerebral disturbances—at first headache and restlessness, then delirium and gradually developing coma, with convulsive twitchings, or more rarely epileptiform fits, towards the end. Jaundice then appears, or if it has been present early it becomes deeper. The blood serum may give either a direct, indirect or biphasic reaction with Van den Bergh's test. The temperature is rarely high, but may be from 101° to 102°. The pulse, which may have been slow with the early jaundice, now becomes quick. The tongue is dry and brown, and as the symptoms progress sordes collect about the lips and teeth. There is, besides, pain in the hepatic region, and decided tenderness, which may be recognised even during the stage of coma, if pressure be made there. The extent of dulness diminishes with great rapidity, so that finally its vertical measurement is only an inch or less.

The abdomen is natural, or towards the end it is retracted. The spleen is mostly enlarged. The urine contains bile and ketone bodies. Leucin and tyrosin which have been described as the most characteristic feature in the urine of acute yellow atrophy, are, as a matter of fact, very rarely observed. On the other hand, the urine not infrequently contains albumin, especially towards the end, and casts; and there may be also blood, the indication of a general hæmorrhagic condition which may be further shown by coffee-ground vomit; by the fæces, which appear to be mostly pale, and deficient in bile, containing blood; and by epistaxis, metrorrhagia, or petechial hæmorrhages under the skin. With increasing coma death finally takes place, the severer symptoms lasting only from two to four days. Pregnant women, as a rule, abort.

Sometimes a case lasts a much longer time, several months or two years, and these cases have been called *subacute atrophy*. In other cases the attack is quite

mild, and the patient recovers. In fact, in cases of T.N.T. and arseno-benzol poisoning it is only the worst cases that die. It was formerly believed that acute yellow atrophy was almost invariably fatal; but these cases of recovery prove that a mild form of the disease does exist. There is often a remarkable latency in the T.N.T. poisoning cases, since jaundice may only be noticeable some years after exposure to the poison has ceased.

**Diagnosis.**—This generally depends upon the occurrence of cerebral symptoms and rapid diminution of hepatic dulness in a jaundiced patient. Occasionally, however the jaundice has been absent. The examination of the liver by X-rays, as described in the diagnosis of poisoning by arseno-benzol (*see* p. 123), might be used for any case of acute yellow atrophy. The disease is closely simulated by phosphorus poisoning, in which the liver undergoes fatty degeneration, and there are jaundice, petechiæ, and cerebral symptoms. But in phosphorus poisoning the liver is usually enlarged and is more fatty.

**Prognosis.**—Acute yellow atrophy when the symptoms are developed is exceedingly fatal; but cases have temporarily improved, to relapse and die later. These are the subacute cases. The milder cases may recover completely.

**Treatment.**—In the final stage little can be done; but in the earlier stages attempts may be made to remove or neutralise the toxic factor, and so possibly prevent the further progress of the disease. Rest in bed, a diet of milk and carbohydrates, abundance of fluid, sodium bicarbonate in large doses (1 ounce in twenty-four hours), and moderate or free purgation, should be tried.

## CIRRHOSIS OF THE LIVER

The name *cirrhosis* is given to diseases of the liver where there is infiltration with fibrous tissue. The name *cirrhosis* (κιρρός, yellow) was used owing to the general yellow colour of the liver in portal cirrhosis, and not in reference to the presence of excess of fibrous tissue. Nevertheless the name has been often applied to chronic fibrous changes in other organs of the body—*e.g.* cirrhosis of the lung and cirrhosis of the kidney; but the term "fibrosis" is much to be preferred for these organs.

There are *three* chief types of cirrhosis: (1) *portal cirrhosis*, in which chronic irritants reach the liver through the portal vein and in which circulatory disturbances causing gastric hæmorrhage and ascites are the prominent clinical features; (2) *biliary cirrhosis*, in which jaundice is the prominent feature, and ascites occurs only as a terminal condition; (3) *pericellular cirrhosis*, occurring in congenital syphilis (*see* p. 490).

### PORTAL CIRRHOSIS

(*Multilobular, Alcoholic Cirrhosis, Hobnailed Liver*)

**Ætiology.**—In the great majority of cases portal cirrhosis is dependent, wholly or in part, upon the excessive use of alcohol, in the form of beer, wine or spirits. Little is known as to the amount that is required to produce cirrhosis; there are the widest individual differences. Some people may drink freely all their lives without acquiring it, whereas in others a few months' indulgence seems sufficient for the purpose. In some children that have been the subjects of it the fact of alcoholism has been proved. But there are cases of undoubted cirrhosis of the liver in which alcohol as a cause can be certainly excluded. The following views have also been advanced: that the real irritant is some toxin produced in the mucus which results from the accompanying gastro-enteritis; that the irritant is not alcohol, but some other constituent in the liquid drunk. The possibility of the former explanation is supported by the fact that cirrhosis in one or other form may be produced by other poisons, or bacterial toxins. It

is said that infectious diseases, such as scarlet fever, measles, pneumonia, or syphilis supply the requisite toxin for the occurrence of the common forms of cirrhosis, and much less pronounced degrees of fibrous overgrowth appear to result sometimes from heart disease, rickets, malaria, dysentery, and rarely tuberculosis. It is conceivable also that intestinal toxins may cause some forms of the disease. Adami combines the action of alcohol with that of bacterial toxins. He suggests that alcohol has a deleterious action upon the hepatic parenchyma and at the same time causes inflammation of the intestinal mucosa; that colon bacilli entering the portal vessels are carried to the liver and there are destroyed, their toxins causing simultaneous degeneration of the hepatic cells, and overgrowth of the connective tissues.

Cirrhosis of the liver occurs as a late result in some cases of splenic anæmia (*see* Anæmia), and the cases are then described as *Banti's disease*. A large-livered cirrhosis is also associated with pigmentation in cases described, some as *hæmochromatosis*, and others as *diabète bronzé*. Of these it is believed that the deposit of pigment from the blood is the first change; cirrhosis results from the irritation of the pigment in the liver; and in bronzed diabetes glycosuria follows upon its deposition in the pancreas.

The tropical infectious disease, Kala-azar, is accompanied by a moderate degree of cirrhosis; a somewhat similar combination of cirrhosis, enlarged spleen and bone marrow changes, but without the Leishman-Donovan bodies, is endemic in Egypt (Day and Ferguson); and a curiously localised portal cirrhosis is found as a result of bilharzia infection (*see* Bilharziasis).

**Morbid Anatomy.**—The cirrhotic liver varies considerably in size. It may be so large as to reach during life 2 inches below the level of the umbilicus, and to weigh after death as much as 8 or 10 lbs.; it may be so small as to be inaccessible to touch, and to weigh only 28 or 30 ounces; and it may be of any intermediate size. The larger livers are often smooth, or only slightly granular on the surface; the smaller livers are coarsely granular or nodular, or present large round bosses, or are distorted into curious shapes. In all cases the organ is very much tougher and harder than normal, from the development of fibrous tissue, which runs in all directions through it. The liver presents on section a number of yellow, brownish-yellow, or brown areas surrounded and separated from one another by broad tracts of grey translucent fibrous tissue. If it can be examined in the earliest stages of cirrhosis, there are found large numbers of round cells infiltrating the tissue about the portal canals (Glisson's capsule), and in some cases penetrating more or less between the lobules, or even within them. Later on white fibrous tissue is developed, which forms a large part of the section in an advanced case. The bands of fibrous tissue running through the organ break it up into islands of hepatic tissue, each of which may consist of several lobules (*multilobular cirrhosis*); but the fibrous tissue not infrequently breaks right through a lobule, and sometimes single lobules are surrounded by it. There is great variety. The cells are atrophied and mostly stained yellow or brown by pigment granules. In the fibrous tissue are numerous newly formed blood vessels, which can be injected from the hepatic artery, and regenerating liver cells are also seen. In fact, the production of cirrhosis may be looked upon as the attempt of the body at repair, which follows necrosis of the middle and outer zones of the lobule. The newly formed, highly vascular, fibrous tissue is an advantage since it supplies nutriment to the regenerating liver cells (**46**). Later on it becomes useless when it contracts, and thus compresses more and more the hepatic cells, the branches of the portal vein, and perhaps the bile ducts. The organ is at first enlarged by the overgrowth of connective tissue, and some large cirrhotic livers also contain a quantity of fat. The liver cells and fat may eventually disappear, and the organ may be reduced much below its normal weight. The varying size of the liver is thus, in part at least, dependent on the stage of the process.

The organ varies much in size; it may be very large, or about the normal size, or very much smaller: in the former case, sometimes called *hypertrophic* cirrhosis, the surface may be fairly smooth or present rather fine granulations; in the latter case, sometimes called *atrophic* cirrhosis, the shape is often much altered from the extensive and irregular contraction of the fibrous tissue, which forms coarse knobs on the surface (hobnailed liver). When ascites has constantly reappeared during life, after tapping, the capsule of the liver is found to be thickened (*see* Perihepatitis).

*Cirrhosis carcinomatosa* is the name given to cases in which a primary carcinoma develops in a cirrhotic liver (*see* p. 491).

**Symptoms.**—The symptoms of portal cirrhosis are mainly due to increasing obstruction of the portal circulation. Jaundice is often slight, and comes on late in the course of the disease. Still, the frequency of the occurrence of latent jaundice (*see* p. 476) shows that the liver parenchyma is usually affected to some extent, though there may be little in the way of subjective symptoms, such as fulness or pain in the hepatic region. The serum may give Van den Bergh's direct test (**53**). On the other hand, there is frequently, as a result of free drinking, a gastritis which produces loss of appetite, furred tongue, and vomiting, especially in the morning. An examination of the abdomen at this stage may, however, reveal a considerable enlargement of the liver, of which the patient is entirely ignorant. The next symptom is not infrequently *hæmatemesis*, or vomiting of blood; this is due to the commencing obstruction in the portal circulation: as the blood in the portal vein finds a difficulty in passing through the liver, the radicles of this system, viz. the mesenteric, gastric, and splenic veins, are, of course, congested, and tend to bleed on to the mucous surfaces. But sometimes the blood proceeds from a rupture of the veins at the lower end of the œsophagus, which have become varicose in the course of establishing a free communication between branches of the portal vein and branches of the inferior vena cava or azygos vein. The quantity thrown up is often as much as 1 or 2 pints, and occasionally the hæmorrhage is directly fatal. The vomiting may be followed or accompanied by melæna. Piles are not infrequently present at the same time, and hæmorrhage from other parts (gums, nose, and lungs) is liable to occur in the course of cirrhosis.

The most important and constant result of the portal obstruction is the effusion of fluid from the distended veins into the peritoneal cavity, constituting the form of dropsy already described as *ascites* (*see* p. 477). In many cases, when ascites has developed, the liver is still enlarged, and can be felt 1 or more inches below the ribs; if the fluid be displaced by the hand (*see* p. 478), the surface is felt to be rough, granular, or nodular, and the edge is rounded. If the organ has already contracted below the normal, it may be impossible to feel it; but the results of percussing the lower ribs to estimate the vertical extent of hepatic dulness are always uncertain in such a case, since the presence of fluid allows considerable change in the relations of the liver, intestine, and abdominal walls to one another. The spleen is often enlarged and may be felt; it is frequently from 20 to 30 ounces in weight. The surface of the abdomen is covered by large veins, running between the iliac and thoracic trunks. This collateral circulation is partly, perhaps, due to the compression of the vena cava inferior by the fluid in the abdomen, for the feet and legs are often œdematous at the same time; but it is also a means by which the portal circulation is relieved. This is an important point, for it must be remembered that the portal system is not completely shut off from the general circulation, but that there are, even in health, means of communication which in cirrhosis become greatly enlarged, and allow of some of the blood in the portal vein radicles reaching the right side of the heart without passing through the liver itself. Those which have been described are communications (1) between the gastric and œsophageal veins at the opening in the diaphragm; (2) between the inferior mesenteric and the hæmorrhoidal branches

of the internal iliac vein; (3) between the coronary veins of the stomach and branches of the phrenic veins; (4) between branches of the mesenteric vein and the spermatic vein, or others in the abdominal wall. Frerichs described (5) vessels forming in the adhesions between the liver and the diaphragm; and (6) a large vein (accessory portal of Sappey) has sometimes been found running along the round ligament of the liver, by which the portal vein communicates directly with branches of the epigastric and internal mammary.

The bases of the lungs are often seriously compressed by a large ascites, and the heart is displaced upward. Hydrothorax and general anasarca often occur. The urine is generally scanty, high-coloured, with abundant deposits of red urates, and not infrequently a trace of albumin. The last may be due to pressure on the renal veins; it is unsafe to diagnose co-existing granular kidneys from that fact alone.

By the time that ascites is well developed the patient is in other respects often seriously ill. He is thin, weak, with sunken eyes, a slight tinge of jaundice, and small stellate venules on the face. The temperature is mostly normal, but fever is sometimes present.

The symptoms remain much the same, but the prognosis is very unfavourable. Sometimes recovery follows the use of diuretics and purgatives and the removal of the fluid by tapping, and is no doubt largely due to the development of the venous anastomosis above noted. And in some cases in children ascites has occurred as the first symptom, and yet the patient has lived for eight or ten years. But death often results within a few months of the appearance of ascites, with cardiac failure, or with cerebral symptoms (delirium and coma), which may ensue at the very time that the fluid is becoming absorbed, and may carry off the patient in a few days. This mode of death is probably due to a toxæmia resulting from the imperfect performance of the hepatic functions. Occasionally hæmatemesis, or hæmorrhage from the gums or nose, or peritonitis after tapping, is fatal. Secondary infections also commonly cause death.

**Diagnosis.**—Cirrhosis is often latent until hæmatemesis, ascites, or slight jaundice discloses the secret; it has been already stated that examination may discover an enlarged rough liver in a tippler who has no decided trouble. Most commonly the diagnosis has to be made when ascites has already appeared, and then the history of drinking and of hæmatemesis, the presence of an enlarged liver, enlarged spleen, and slight jaundice, are sufficient to determine the case. Functional hepatic tests are of value in diagnosis. Of the other conditions of the liver and peritoneum causing ascites the most important are *carcinoma*, which may obstruct the portal vein, or its largest branches, and the association of *perihepatitis* with *chronic thickening of the peritoneum* (*see* Perihepatitis). *Carcinoma* and *tubercle*, apart from the liver, also cause a peritonitis, which results in ascites. The former may be recognised by the occurrence of nodules of growth in different parts of the abdomen. The latter often presents a thickening of the omentum, which may be mistaken for an enlarged liver. But the resemblance between atrophic cirrhosis with ascites and tuberculous peritonitis with liquid effusion alone may be very close; and the diagnosis may be possible only after a tapping, when the abdominal organs can be better felt, and the fluid can be examined bacteriologically, or if there is evidence of tubercle in other parts of the body. Hæmatemesis is frequently the result of cirrhosis, and is valuable in diagnosis; but it also commonly occurs in acute gastric ulcer and splenic anæmia.

When the chief symptom is jaundice, and there is no ascites, the liver is mostly enlarged, and nearly smooth on the surface. An infiltrating carcinoma of the liver may closely resemble a large-livered cirrhosis, and the consistence of the organ may be very similar in the two cases. The persistence of bile pigment in the stools shows that the larger ducts are not obstructed, and rather favours cirrhosis.

The not infrequent coincidence of multiple neuritis and cirrhosis from alcoholism should be borne in mind.

**Prognosis.**—This is very bad. When ascites appears, the future course is often only a few months; on the other hand, repeated tappings with complete change of habits may prolong life for years.

**Treatment.**—Little, if anything, can be done with the cirrhotic liver itself; and treatment resolves itself into the prevention of further mischief, and the attempt to obviate the effects of the damage already done. In alcoholic cirrhosis the first essential is that the ingestion of alcohol should be absolutely stopped; and in early stages, where the liver is still uncontracted, and ascites has not yet appeared, the liver may regain its normal size, and the patient his health. It is, however, impossible in such a case to say how far fibrosis has progressed. The diet should be light and easily digestible; the bowels should be kept active, and sickness and any dyspeptic symptoms may be treated by effervescing salines, bismuth and bitter tonics. When ascites occurs an attempt to promote its absorption must be made by the use of diuretics and purgatives. Of the former acetate, nitrate, and bitartrate of potassium, spirits of nitrous ether, squills, and digitalis are most frequently given; and the resin of copaiba in doses of 15 grains three times a day sometimes has a good effect. But the kidneys act at a disadvantage from the pressure of the ascitic fluid. Of purgatives sulphate of magnesium, bitartrate of potassium, compound jalap powder, or elaterium may be employed. If these fail to remove the fluid, and the abdomen becomes very tense, paracentesis is required, and it may sometimes be repeated with success as the fluid reaccumulates. Attempts to develop a collateral circulation based on the view that ascites is mainly mechanical in origin (*see*, however, p. 477) have been made (1) by opening the abdomen, scraping the peritoneum on the opposed surfaces of the liver and diaphragm, and bringing them into contact by stitches (Drummond and Morison, Talma), and (2) by uniting the great omentum to the anterior abdominal wall (*epiplopexy*). It is even claimed that 30 per cent. of these operations have met with some success.

## BILIARY CIRRHOSIS

1. **Hanot's Cirrhosis** (*Hypertrophic Biliary Cirrhosis*).—The ætiology of this disease is obscure. It is not known whether the liver parenchyma or the bile ducts are primarily affected. It is a rare disease, commoner in males than females, and often occurring in children. There is considerable enlargement of the liver, and often in children still greater enlargement of the spleen, stunted growth of the patient, deep pigmentation of the skin, and marked clubbing of the finger ends (*splenomegalic cirrhosis*). Jaundice is a marked feature of the disease, and the urine contains bile pigment, whereas ascites is not present until just at the termination. The disease may last for several years. Towards the end the patient becomes delirious, even violently so, and relapses into coma. The temperature is high, hæmorrhages occur under the skin and from the mucous membranes, and he dies in three or four days.

The liver, besides being smooth and large, is deeply stained with bile on section. The fibrous tissue is much more delicately arranged than in portal cirrhosis, each lobule being surrounded by fibrous tissue (*unilobular cirrhosis*). Some fibrous tissue may also be present inside the lobule round the liver cells. There is marked proliferation of bile ducts. There is evidence that a large cirrhotic liver may become smaller during the patient's lifetime. Sir Frederick Taylor recorded a case in which a liver reaching below the umbilicus, in a patient with strongly marked jaundice and no ascites, was found fifteen months later to have contracted quite close under the edge of the ribs. The treatment is described under Portal Cirrhosis.

2. **Obstructive Form.**—Experimentally Rous and Larimore have pro-

duced a pure unilobular cirrhosis similar to Hanot's cirrhosis by ligaturing one of the bile ducts and the corresponding branch of the portal vein. The amount of bile formed was less than before, but being unable to pass along the usual channels, it passed out through the walls of the interlobular ducts, causing irritation and fibrous tissue formation.

Again, by ligaturing the other branches of the portal vein instead, all the portal blood was diverted to the area of the liver where there was biliary stasis. A larger amount of bile was formed, which escaped out of the intralobular bile canaliculi, giving rise to a pericellular cirrhosis.

These results have naturally a great bearing on the pathology of Hanot's cirrhosis, suggesting that it may really be due to obstruction of the smaller bile passages. There exists, however, a form of obviously obstructive biliary cirrhosis, which is sometimes, but not always, met with clinically, when there is long-continued obstruction of the bile ducts due to gall stones or carcinoma, etc. The bile ducts are seen to be greatly dilated, and the liver may show unilobular, or sometimes multilobular, cirrhosis. The treatment consists in removing the cause as far as possible by surgical methods.

### OTHER FORMS OF ATROPHY AND CONTRACTION OF THE LIVER

Some atrophy occurs as the result of old age or of inanition. The organ may be reduced to half its normal bulk, but there is no alteration in its structure and no induration, the lobules being diminished in proportion to the size of the whole organ. It produces no symptoms. Partial atrophy may result from the pressure of adjacent organs or of tight-lacing; the right lobe of the liver is elongated downwards, and where it is compressed between the lower ribs and the right kidney, the hepatic tissue is atrophied, and replaced by fibrous tissue in a transverse line below which a portion of liver extends down to the umbilicus level.

Perihepatitis also causes a certain amount of atrophy.

## FATTY LIVER

*Fatty Infiltration.*—The hepatic cells normally contain a small quantity of fat in the form of minute globules. Under certain conditions the fat is immensely increased, and each cell may contain such a large amount that the nucleus and outline of the cell are entirely obscured, and the cell itself might be supposed to be destroyed. This change takes place first at the periphery of the lobule; later the whole is invaded. The liver is much enlarged, it has a smooth surface, is somewhat rounded at the edge, on section has a whitish-yellow colour and uniform appearance, and it may actually float in water. This *fatty infiltration* occurs (1) physiologically, during the later months of pregnancy and during lactation; (2) in association with general obesity, which may be due to eating too much food, or possibly to a congenital deficiency in the power of oxidising foodstuffs; (3) from the consumption of alcohol; (4) in certain wasting diseases, particularly phthisis.

The fatty liver is painless; it can readily be felt as a large, smooth organ in phthisis; but it may be less easy to feel in obesity, on account of the thickness of the abdominal walls. Dyspeptic symptoms and deficient secretion of bile are referred to fatty liver. A partial form of fatty change occurs as a result of long-continued congestion in the nutmeg liver of heart disease; the fatty infiltration is most marked at the periphery of the lobules.

*Fatty Degeneration.*—This is regarded as a primary degeneration of the cell, the free fat being deposited in the cell as the result of damaged cell activities. The causes are (1) febrile conditions, where the toxins and high temperature

damage the cell: it may follow "cloudy swelling" of the cells; (2) severe anæmias and acute yellow atrophy; (3) poisoning by phosphorus, arsenic, carbon monoxide, chloroform, etc.

## LARDACEOUS DISEASE OF THE LIVER

Lardaceous degeneration has been already referred to in connection with empyema and phthisis; and as the liver is one of the organs which are most frequently implicated, a short account of the degeneration must here be given. It consists in the deposition in the tissues of a firm, colourless, translucent material (*lardacein*), which is stained by certain colouring agents. Thus iodine in aqueous solution turns it a rich brown-red or claret colour. The iodine may be applied to a section of the fresh organ after this has been washed free of blood, and the affected parts are then mapped out by the characteristic tint. The subsequent addition of dilute sulphuric acid changes this to a dark purple hue. Methyl-violet or gentian-violet turns lardaceous matter red, while the surrounding healthy tissue is stained blue.

Lardacein, which was at first thought from the iodine reactions to be of a starchy nature, is a combination of chondroitin-sulphuric acid and proteid. The old terms, *amyloid substance* and *amyloid liver*, are therefore incorrect. Lardacein is very resistant to chemical action and to putrefaction.

The tissues in which it is found are, first in point of time, the walls of the blood-vessels; secondly, various connective tissues; and lastly, if at all, the gland cells of an organ. Indeed, the material is mostly intercellular in its position: thus it is found in the small arteries deposited between, and separating from one another, the muscle fibre cells of the middle coat; in the spleen it exists as streaks and patches between the cells of the pulp; and in the liver it lies in similar particles between the capillaries and the gland cells. It is, indeed, not so much a degeneration as an addition to the structure; and solid organs affected by it are generally much enlarged. Its relation to the vessels suggests that it is deposited from the blood.

It occurs most often in the spleen, kidneys, liver, intestines, and stomach, and with decreasing frequency in the suprarenal capsules, lymphatic glands, thyroid, aorta, ovaries, and uterus.

The lardaceous deposit can, in the vast majority of cases, be attributed either to prolonged suppuration from phthisis, syphilis, tuberculous disease of bones and joints, and empyema, or to syphilis, without suppuration; and it is probable that bacterial toxins are the intermediate agent. Other cachectic conditions are sometimes present. Improvements in surgical procedure have very much diminished the frequency of lardaceous disease.

In the liver the lardaceous change is first observed in the middle zone of the lobules, where the capillaries are most intimately connected with the divisions of the hepatic artery. As the deposit increases the hepatic cells are compressed and atrophied, but they are only occasionally the seat of lardaceous deposit. The liver becomes enormously enlarged, has a smooth surface and somewhat rounded edge, and is entirely free from pain or tenderness. The disease causes no jaundice. It is accompanied by the signs of the causative disease, and often by an enlarged spleen, albuminuria and diarrhœa, the results of the deposit in other organs. A lardaceous liver, which is at the same time the seat of a syphilitic gumma or cicatrix, naturally loses its uniform smooth surface, but may be recognised by its other associations. The portal circulation is not obstructed by the lardaceous change, and although ascites is not infrequently present, it is mostly associated with general anasarca, and must be referred with it to co-existing disease of the kidneys, or it may be due to other complications, such as cirrhosis, gumma, or chronic peritonitis.

**Prognosis.**—This is very bad, but decrease of the enlargement after efficient surgical treatment has been recorded.

**Treatment.**—The cause must be, if possible, removed. This is impracticable in phthisis, but other sources of suppuration may perhaps be treated surgically; and potassium iodide, cod-liver oil, iron, quinine, and other tonics should be given. Mercury and potassium iodide should be used in syphilitic cases.

## SYPHILITIC DISEASE OF THE LIVER

This may be *congenital* or *acquired*.

**Congenital Syphilis** occurs first as *pericellular cirrhosis*, secondly as gumma. The former change begins as a cellular infiltration, which develops into a fibroid induration; it invades the lobules, and surrounds each cell with a layer of fibrous tissue, leading to considerable enlargement of the organ. Microscopic examination may also show: (1) increased fibrous tissue round the portal canals (periportal cirrhosis); (2) numerous embryonic red cells, due to the liver continuing its hæmatopoietic functions, which may help to compensate for the destruction of corpuscles by the syphilitic toxin; (3) miliary gummas (**47**). Spirochætes are present in the connective tissue. The spleen is often enlarged at the same time. Jaundice occasionally occurs, but ascites rarely. A multilobular cirrhosis has sometimes developed in those previously the subjects of the intercellular form.

**Treatment.**—Hydrargyrum cum cretâ should be given, or mercurial inunction should be employed for some months, and their use should be intermittently continued for two or three years. (*See also* p. 122.)

**Acquired Syphilis.**—The jaundice that is not infrequent in the early stages of syphilis, and was known before the introduction of arseno-benzol compounds, shows that this infection may produce an acute hepatitis. In the later stages syphilis produces gumma of the liver. This presents the general features of gumma in other situations, and spirochætes are found. They are more or less spherical yellow masses, tough and elastic, surrounded by a zone of grey fibrous tissue, from which branch off numerous bands into the adjacent hepatic substance. The contraction of the fibrous tissue produces a depression or fissure on the surface of the liver, at the bottom of which lies the gumma which has caused it; and so the organ may become coarsely lobulated and deformed. Gummas not infrequently break down in the centre into a puriform detritus; on the other hand, they may become completely fibrous, so that nothing remains but a depressed scar; or calcareous granules may be deposited in them. Gummatous livers often become lardaceous, and in consequence they may be of large size in spite of cicatricial contractions. Perihepatitis is another change resulting from syphilis. It is probable that syphilis is responsible for some cases of multilobular cirrhosis.

**Symptoms.**—Occasionally a large gumma may form a prominence on the anterior surface of the liver, smooth and elastic, and strongly suggestive of a hydatid or other cyst; it may cause elevation of the right costal margin (*see* p. 389). More often, but probably in later stages, syphilitic livers are large, hard, irregular on the surface, and deformed, from the contraction of the fibrous cicatrices. Neither ascites nor jaundice is necessarily present, but in particular cases they may occur from the pressure of a gumma upon the portal vein or the bile duct; and there is often albuminuria from co-existing lardaceous disease of the kidney. A gumma is sometimes accompanied by decided fever of hectic type.

**Treatment.**—In early cases, iodide of potassium will quickly reduce the gumma and check the fever accompanying it; salvarsan may also be tried cautiously, the patient being given plenty of carbohydrate (*see* p. 124). But when there are old cicatrices and extensive lardaceous disease, little good can be expected.

## TUBERCLE OF THE LIVER

This is almost invariably a part of a general tuberculosis. It occurs either as minute greyish-yellow granulations, less than a pin's head in size, or of somewhat larger (3 to 5 mm.), bright yellow masses, more easily detached from the surrounding hepatic tissue. These larger masses are often softened into a cavity in the centre, which is deeply stained with bile. As a rule, no local symptoms accompany hepatic tuberculosis; but occasionally a general enlargement of the liver results, and in rare cases there is jaundice (*see* Miliary Tuberculosis, p. 130).

## NEW GROWTHS IN THE LIVER

The only tumour of the liver that is at all common is carcinoma. Of others cavernous angeioma, simple cysts, and the lymphadenomatous deposits associated with Hodgkin's disease are the most frequent. They rarely cause definite symptoms. Cases of spindle-cell sarcoma, melano-sarcoma, cysto-sarcoma, myxoma, and adenoma have been recorded.

### CARCINOMA OF THE LIVER

**Pathology.**—Primary carcinoma occurs in two forms—as nodules appearing in any part of the liver and as a diffuse infiltration. Histologically carcinoma of the liver is of two kinds, consisting of (1) liver cells, which may secrete bile, or (2) bile duct cells. Primary carcinoma occasionally develops in a cirrhotic liver. The liver is not, as a rule, much enlarged; it presents, besides the fibrous overgrowth, multiple tumours which are at first firm and white, but later degenerate or undergo necrosis, and acquire a yellow or green colour. The clinical features are those of cirrhosis, and the condition has been called *cirrhosis carcinomatosa*.

By far the greater number of cases of carcinoma of the liver met with are secondary to carcinomatous deposits in other organs, especially the stomach, the intestine, the gall bladder, the glands in the portal fissure, the uterus, or the breast. The carcinoma cells are carried to the liver by branches of the portal vein, and lodged in the lobular capillaries. The form of the secondary carcinoma, whether soft or hard or melanotic, is determined by the nature of the primary growth.

If the carcinoma is diffused, the liver is merely enlarged; but when it exists in the form of nodules, or separate tumours, the liver takes at the same time the most varied shapes. Each nodule tends to grow evenly in every direction, and thus to keep a globular form, and when it reaches the surface it will project as a hard, convex, or hemispherical outgrowth. But as the nodules become larger—for instance, 1½ to 2 inches in diameter—they often break down in the centre into granular and fatty detritus, and as a consequence those that project on the surface, being unsupported on one side, sink in and form a central depression or *umbilication*, a condition which may sometimes be felt through the anterior abdominal wall. The lower edge of the liver is also irregular and nodulated. On section such a liver presents irregular areas of white carcinomal growth, with a more or less circular outline; the larger ones are softening in the centre, and many of them are blotched by hæmorrhages. The intervening hepatic tissue is often of a deep brown or yellow colour. Where the carcinoma has started from the gall bladder, or the bile duct, or has grown in from the portal fissure, the growth is most extensive in that region, or may be quite confined to it. Sometimes the empty gall bladder, or a gall bladder containing some calculi, is embedded in a mass of carcinoma. Carcinomatous nodules near the portal fissure may compress the bile duct or the portal vein, and the latter may be entirely filled by the new growth.

**Symptoms.**—Carcinoma of the liver usually gives rise to a good deal of pain, affecting the right hypochondrium, shoulder, and loins. At first not much more than a sense of weight and uneasiness, it afterwards becomes severe and lancinating, and is accompanied by tenderness. Occasionally, however, pain is absent. The liver, as already stated, is enlarged; it may reach far below the umbilicus, and over towards the left side; the nodules are prominent on the surface, and the irregular outline may even be seen in profile. For the most part the enlargement is in a downward direction, but large masses may grow from the convex surface, and force up the diaphragm so as to compress the base of the lung. The surface of the carcinomatous mass is, as a rule, of almost stony hardness, distinctly more hard than cirrhosis, or lardaceous disease, and the transition from hard carcinoma to the soft normal tissue can often be recognised. Jaundice occurs in about half the cases and can generally be shown to result from pressure on the main bile duct, especially in those cases where the carcinoma starts from the portal fissure. Van den Bergh's direct test is given. Similarly, ascites is often, but not always, present, and rarely is the fluid as abundant as it may be in cirrhosis. It mostly depends on direct pressure on the portal vein or its large branches, occasionally on a co-existing peritonitis. The emaciation, pallor, and prostration common to malignant diseases of the abdominal viscera are also present. Pyrexia occurs in many cases of carcinoma of the liver, and occasionally it has exacerbations and remissions like those seen in Hodgkin's disease (*see* p. 553).

**Diagnosis.**—A jaundice of some months' standing in an old person with an enlarged liver is, in the majority of cases, due to carcinoma of the liver or of the head of the pancreas, though occasionally the bile duct may be obstructed permanently by a gall stone. If hard, irregular nodules are felt on the surface of the liver, the diagnosis of hepatic carcinoma is highly probable; the prominence due to gumma is generally solitary, an inch or more in diameter, and soft or elastic. If the liver is of uniform and not very great hardness, carcinoma is only probable. In cases without jaundice, the large, irregular, and bossy liver and the emaciation of the patient are generally distinctive. Lardaceous and cirrhotic livers are less hard and more uniform. In both these cases the spleen is frequently enlarged also, in the first case by lardaceous deposit, in the second by venous stagnation, whereas carcinomatous enlargement of the spleen is relatively uncommon. Syphilitic livers may be irregular and painful, but often occur in younger people, and have their own special history. A long history of gall stones does not exclude, but rather favours, the possibility of carcinoma.

**Prognosis.**—This is hopelessly bad. The duration is rarely more than twelve months, but may be two or three years. The softer forms of growth may kill within a month or two.

**Treatment.**—This can be only palliative, and consists in relieving pain and in meeting other symptoms, mostly of the digestive organs, such as vomiting, flatulence, and constipation. The diet should be light but nutritious.

## CYSTIC DISEASE

In this uncommon condition there are numerous cysts, more or less aggregated together, varying in size up to an inch or more in diameter, containing a clear or yellowish-brown watery liquid. The condition is sometimes discovered soon after birth, and hence is clearly congenital; at others it is first recognised in the adult. In both cases it is very frequently associated with cystic disease of the kidneys and other organs.

The origin of these cysts is uncertain; but they are evidently malformations resulting from occlusion of bile ducts, or, as in the kidney, from a failure of union of ducts arising from two different sources.

The cystic change generally causes enlargement of the liver, but otherwise

produces no symptoms, and its diagnosis, prognosis and treatment are dependent upon the like change in the kidneys with which it is associated (*see* Cystic Disease of the Kidney).

## HYDATID OF THE LIVER

Hydatid tumours are cysts which contain a colourless, non-albuminous liquid, and which arise as a stage in the development of an intestinal worm, the *Tænia echinococcus* (also known as *Echinococcus granulosus*). They may occur in any part of the body, such as the brain, lung, spleen, peritoneal cavity, intermuscular spaces, or spinal canal, but are most frequent in the liver; and accordingly the description of their growth will be given in this place.

The *Tænia echinococcus* is a minute tapeworm, measuring only ¼ inch and consisting of four segments, of which the first has hooklets and suckers, and the last, longer than the other three put together, forms the mature *proglottis* (*see* p. 467). This worm inhabits the intestines of the dog, and its cystic form infests the sheep, just as the cystic forms of the human tæniæ are found in pigs and cattle. If the eggs of the dog's tænia by any accident reach the human intestine, an embryo in due time escapes, and finds its way to the liver, where it loses its hooks, and is transformed into a vesicle or cyst, containing a clear liquid. The wall of the cyst consists of an outer laminated very elastic layer and an inner *parenchymatous* layer, containing granular matter, cells, muscle fibres, and a vascular system. As it grows it sets up a certain amount of irritation in the tissue around, and a layer of fibrous tissue is developed in immediate contact with it. The liquid is clear, or just opalescent, of specific gravity 1,005 or 1,007, and free from albumin; but it is said to contain a small quantity of glucose and succinate of ammonium.

As the cyst grows it may be reproductive in three ways. First, when the cyst has reached the size of a walnut, it develops from its inner parenchymatous layer smaller cysts, which remain attached by a pedicle, and in which are formed from three to six or more *scolices*—small bodies with four suckers and a ring of hooklets at one end. The hooklets are 25$\mu$ in length, are slightly curved, and present a prominence on the concave side which makes them almost triangular. They may be recognised when the contents are withdrawn from a cyst at operation. The cysts containing scolices are called *brood capsules*, and are sometimes so numerous as to give a velvety appearance to the inner surface of the hydatid cyst. Secondly, the original cyst produces so-called *daughter-cysts*, either directly from the brood capsules which then become detached, or by independent growth between the two layers of the cyst and subsequent discharge into its interior. The daughter-cysts have the same structure as the original, or *mother-cyst*, and may produce within themselves *granddaughter-cysts*. Thus a mother-cyst may contain hundreds or thousands of cysts, of all sizes, from a pea upwards. This is called *endogenous* cyst formation. A hydatid cyst may be sterile, producing neither brood capsules nor daughter-cysts. "Secondary echinococcus" is the name given to the proliferation of hydatid cysts outside the original cyst if the latter has been ruptured so as to allow the escape of its contents. Secondary echinococcus may also follow exploratory puncture of a cyst; the peritoneal cavity, if infected in this way, may become full of cystic material. It has been recently discovered that the scolex is responsible for this multiplication, becoming itself converted into a complete living hydatid cyst.

A very rare kind of hydatid is the *multilocular hydatid*. It forms a hard globular mass, consisting of a number of cavities or alveoli, about the size of peas, with transparent, jelly-like contents, which Virchow showed to be the remains of hydatid cysts. Scolices are sometimes found, but the cysts are mostly sterile.

The ordinary hydatid cyst may grow to an enormous size, so as to contain several pints of fluid, and it thus constitutes a tumour which exerts considerable pressure on surrounding parts.

**Changes in the Cyst.**—A hydatid cyst may last for several years, without any essential change beyond its growth; but its existence may be shortened (1) by spontaneous rupture, (2) by death and conversion into a harmless mass, and (3) by suppuration. It is not quite clear what is the exact cause of death, whether the entrance of bile into the cyst or the impaired nutrition of the daughter-cysts, due to the rigidity of the capsule. In any case, the result is that the hydatid is converted into a mass of opaque membranes, more or less closely packed together, and mixed with a yellow pasty or putty-like material, in which calcareous salts and cholesterin crystals can be recognised. Very rarely the contents of the cyst are converted into a gelatinous substance, containing abundance of albumin and chlorides (Bruce and Sheild). If suppuration takes place, the hydatid is also killed, and the abscess thus formed contains shreds of hydatid membrane, and the hooklets from the heads of the scolices. Further, the spontaneous rupture of a cyst is followed by suppuration.

**Symptoms.**—Of these the most important is the swelling which the liver, enlarged by the presence of the hydatid, forms in the upper part of the abdomen. If the cyst is deeply seated, or on the upper surface of the liver, the swelling may be entirely due to the displaced or enlarged liver; if the hydatid is near the anterior surface, it forms a distinct globular or hemispherical prominence, which is tense, elastic, and, if of sufficient size, distinctly fluctuating. Such cysts sometimes present what is known as the *hydatid thrill*. If the finger or fingers of one hand are laid on the tumour and are struck with the tips of the fingers of the other hand, a vibration is set up which can be felt for some little time by those of the hand still applied. It is, however, by no means always present, and it is of doubtful significance, since it is probably due merely to the tension of the cyst wall, and may therefore presumably be obtained in other cysts, if tightly filled.

Cysts which do not merely project from the lower surface of the liver, but occupy its substance or upper part, frequently exert a local pressure upon the ribs, diaphragm, and right lung, with the same results as are seen in hepatic abscess, viz. bulging of the right side of the chest, elevation of the lower ribs, enlargement of the right costal angle, and dulness of the right base, with diminished breath sounds and diminished tactile vibration (*see* p. 389).

A comparatively small cyst may happen to press on the portal vein and cause ascites, or on the bile duct and cause jaundice.

Pain is not generally a prominent symptom in hydatid tumour of the liver, and it may be entirely absent. Or it may be severe, even when the tumour is small. It sometimes depends on the occurrence of peritonitis over the cyst, or on the size of the cyst being such as to cause much local tension. The health of the patient is generally good, and is at first entirely unaffected by the presence of the hydatid; but sometimes there are attacks of urticaria, and in most cases there is a moderate excess of eosinophil leucocytes in the blood, reaching 8 per cent. or more.

Suppuration of the cyst is commonly indicated by the onset of pain, or by its increase if formerly present; the patient loses health and strength, and has elevation of temperature, and perhaps rigors. He has, indeed, an abscess of the liver, with its accompanying conditions; and this abscess may similarly point and discharge its contents in different directions. Rupture through the abdominal parietes has occurred with varying results; perforation into the stomach or alimentary canal may be followed by recovery, daughter-cysts and portions of the mother-cyst escaping by the fæces or by vomiting. Or the cyst opens into the base of the lung, and pus, cysts, and bile pigment are expectorated, also with a favourable result in some cases. Rupture into the pleura or pericardium is nearly always fatal, but the patient may survive a rupture into the peritoneum for a long time. A large quantity of peritoneal fluid is effused, the eosinophilia may reach a high degree (30 to 50 per cent. of the leucocyte count), and urticaria is frequent. Sometimes the hepatic vein or the inferior vena cava has been

opened, and secondary cysts have been carried into the right ventricle and have blocked the branches of the pulmonary artery ; and the portal vein has also been invaded. If the cyst communicates with the biliary passages, bile stains the daughter-cysts, and causes the death of the parasite. Conversely, the cysts may lodge in the bile ducts and cause jaundice.

**Diagnosis.**—Hydatid is distinguished by its being a localised swelling of the liver of long duration, and not at first affecting the health of the patient. Where it is accessible to palpation, its round, elastic, and fluctuating properties distinguish it from most other enlargements of the liver. Sometimes a hydronephrosis may closely simulate a hydatid.

When suppuration has taken place, it is indicated by the local pain, tenderness, and prominence, combined with the constitutional disturbance ; and the previous history will generally help to distinguish the hydatid from the tropical or pyæmic abscess.

Information may also be obtained from the blood by a leucocyte count, when the eosinophils will be found to be in excess. There are three tests based on immunity reactions (**54**) : (1) Complement fixation. The test is carried out in the same way as the Wassermann reaction (*see* p. 114). The most reliable antigen is the fresh, clear, uncontaminated hydatid fluid from the sheep ; but a more stable antigen is obtained by digesting a saline solution extract of scolices with trypsin. The reaction is equally successful in whatever organ of the body the parasite is situate, whether the serum is taken during life or after death of the patient, and whether the parasite is living or dead ; thus it is not prevented by suppuration of the cyst, nor by the entry of bile into it. (2) Precipitin reaction (*see* p. 16). This is obtained by mixing antigen with the serum of the patient and allowing to stand. and is not so delicate as the former. (3) Casoni's intradermal test, depending on anaphylaxis (*see* p. 17). 0·4 c.c. fresh hydatid fluid from the sheep is injected intradermally. If positive, a wheal varying in size from a shilling to half-a-crown appears in twenty minutes, surrounded by erythema and swelling ; but the appearance of the erythema and swelling may be delayed for twelve or twenty-four hours. If both complement fixation and intradermal tests are carried out, a correct diagnosis should be arrived at in 90 per cent. of cases.

**Prevention.**—Since the hydatid of man is obtained from the *Tænia echinococcus* of the dog, and that is propagated by means of the sheep and pig, it is desirable, first, to prevent dogs from eating offal from sheep and pigs, and, secondly, to destroy the tapeworms (or their eggs) developed in the dog. For this last purpose it has been recommended to purge dogs periodically, and to burn or bury their excreta, further to scald frequently the floors of their kennels.

**Treatment.**—Drugs have no influence upon the growth of the parasite. It is unsafe either for diagnosis or treatment to remove its contents by exploratory puncture through the skin, owing to danger of dissemination. The best plan is to expose the cyst by a free incision and remove the cyst completely. The cyst may also be sterilised by "formolisation." The contents are removed by a needle and syringe, and 1 or 2 per cent. formalin injected and kept in for four or five minutes. If the cyst has already suppurated, it must be opened and treated in the same way as amœbic abscess.

## SUPPURATIVE CHOLANGITIS

This is always due to infection by micro-organisms, *e.g.* streptococci, staphylococci, pneumococci, the typhoid bacillus, and *Bacillus coli communis ;* and is either determined by local diseases, such as gall stones, the most common cause, by carcinoma, by hydatid cyst rupturing into the ducts, or by the more general infections of influenza, pneumonia, typhoid, and cholera. There

is swelling and thickening of the bile ducts throughout the liver; the organ becomes enlarged; the ducts are dilated, and numerous foci of suppuration, forming smaller or larger abscesses, occur. The inflammation may extend to the pancreatic duct, and cause suppurative pancreatitis; or abscesses near the surface may lead to localised or general peritonitis. Occasionally infection extends so as to cause a general pyæmia or infective endocarditis.

The symptoms are pain and tenderness over the liver, loss of appetite, nausea, vomiting, pyrexia and prostration, and often jaundice. The liver is enlarged, and increases in size as the illness progresses. The spleen may be enlarged. The duration is from a few weeks to some months, and the disease is often fatal.

**Diagnosis.**—*Suppurative pylephlebitis* and *tropical abscess* of the liver may resemble suppurative cholangitis. The distinction in the former case may be very difficult: in both the antecedents should be carefully considered, that is, local foci of suppuration in the one case and dysentery in the other; and the recognition of a localised swelling in the liver would, of course, be in favour of abscess.

**Treatment.**—This can only be surgical. The bile ducts must be drained, where possible, by opening the gall bladder, or any ducts which may be accessible.

## CHOLECYSTITIS

Inflammation of the gall bladder may be catarrhal, suppurative, or gangrenous. It is due to infection by micro-organisms, and thus it is occasionally a complication or the result of typhoid fever, pneumonia, malaria, cholera, pyæmia, septicæmia and puerperal infections. Streptococci, staphylococci, and *Bacillus coli communis* have been found. Cholecystitis may result from the presence of gall stones, though with the latter the gall bladder has frequently been found sterile (**16**). Injury, as by blows or falls on the hepatic region, is also an occasional cause. It may be associated with chronic pancreatitis.

The gall bladder, when inflamed, becomes distended, its walls are thickened, and the serous coat becomes dull, or adhesions take place to surrounding parts. The mucous membrane is congested or ulcerated, or presents lymph on its surface; and sometimes a complete cast may form (*membranous cholecystitis*). One of the commonest types is the *strawberry gall bladder*, in which the mucous membrane is covered with yellow specks, because the villi have been denuded of epithelium and are stained with bile. Within the gall bladder, which may extend several inches beyond the edge of the liver, is a serous, or sero-fibrinous, or purulent fluid. In very severe forms not only does the gall bladder contain pus, but the walls are intensely inflamed, œdematous, and infiltrated with pus (*phlegmonous cholecystitis*), and in a still more virulent form they are dark green, soft, friable, and sloughing in more or less extensive patches (*gangrenous cholecystitis*). The inflammation may of course extend in various directions, and give rise to local peritonitis with adhesions, in phlegmonous and gangrenous cases to intense general peritonitis, and in others, by extension upwards, to cholangitis.

If a gall stone is the cause of an ulcerative cholecystitis, adhesion to the colon or duodenum may be followed by a fistulous communication, so that the gall stone passes directly into the intestine. Not infrequently also the gall bladder adheres to the abdominal parietes, and an abscess is formed in them which communicates often by a sinuous track with the cavity of the gall bladder.

The organisms found in different cases are streptococci, staphylococci, pneumococcus, typhoid bacillus, and the colon bacillus.

The acute conditions may subside into a chronic form, with thickening and contraction of the gall bladder; or the gall bladder contains thick mucus; or it contains pus, forming *empyema* of the gall bladder.

**Symptoms.**—There is generally an acute onset with persistent or paroxysmal

pain in the region of the gall bladder, that is, near the tip of the right ninth costal cartilage, tenderness in the right hypochondrium at that point and over the costal margin, rigidity of the upper part of the rectus muscle, and resistance on deep pressure. After a time a definite tumour is formed by the distended gall bladder, and this may be followed by swelling of the abdomen. The pain may extend to the right iliac fossa, or it may be increased by taking a deep breath. Nausea and vomiting, anorexia, fever, slight or severe in different cases, with perhaps rigors, occur, and jaundice in about one-third of the cases. The liver is not enlarged, but the spleen is sometimes. There is leucocytosis in suppurative cases.

**Diagnosis.**—Cholecystitis without the presence of gall stones, not at all an uncommon condition, may be diagnosed when there is severe pain and tenderness beneath the right costal margin and some dyspepsia, and when careful examination has excluded duodenal ulcer and appendicitis. "Medical" drainage of the gall bladder, as described on p. 500, may be of value both in diagnosis and treatment. A distended gall bladder may be confounded with a renal tumour or a growth of the colon, or even with a large liver itself; and if it extends very low while giving rise to acute symptoms, it may be taken for appendicitis; but with care the fact that the swelling has connections with the liver, and not with the pelvis, may be made out.

**Prognosis.**—Milder cases recover with simple treatment, but in severer forms extension to the peritoneum may be fatal unless anticipated or treated promptly.

**Treatment.**—The milder cases require rest in bed, local anodynes to the right hypochondrium, and morphia injections if the pain is severe. The bile may be disinfected by giving 10 to 20 grains of sodium salicylate three times a day or hexamine (20 grains, increasing to 60 or 80 grains) at night with sufficient alkali, *e.g.* 60 grains of potassium citrate, to make the urine alkaline and to stop irritation of the urinary tract from the hexamine, which decomposes into formalin in acid solution (**40**). If there is good reason to believe that there is suppuration or gangrene, or that gall stones are the cause, surgical interference is desirable, the gall bladder being opened, and drained (*cholecystotomy*) or in suitable cases removed (*cholecystectomy*).

## GALL STONES

(*Cholelithiasis*)

Biliary calculi, or gall stones, are formed from the bile in the gall bladder, or very rarely in the bile ducts in the liver. They vary in size from a mere sand to ovoid masses of 2 inches in length by an inch in breadth; more often they measure from ¼ to ½ inch in diameter. They are often roughly cubical in shape, presenting facets when several have been in contact; otherwise they may be more rounded. The largest have the ovoid shape, which would result from their occupying the whole cavity of the gall bladder. They are olive-green, brown, or yellowish brown in colour, and on section often show a central dark nucleus with concentric markings and radiating lines. The chief constituents of gall stones are cholesterin, bile pigment, and calcium salts; and the bile pigment is mostly combined with calcium, as bilirubin-calcium. Those which consist mainly of bile pigment are small, dark and friable. Others have a nucleus or centre of bile pigment, and are surrounded by layers of cholesterin crystals which radiate from the nucleus; these stones are generally larger, harder, and have a paler colour. There may be only a single gall stone of larger or medium size, or there may be several hundred.

**Ætiology.**—Gall stones are commoner in advanced life, and occur in women more often than in men. Sedentary occupations and over-indulgence in

food seem to have some influence, and cholesterin-containing foods are thought to be more injurious. Pregnancy and typhoid fever may predispose to gall stones.

One view as to their formation is that they result from infection and catarrh of the gall bladder, due to *Bacillus coli communis*, or less commonly to typhoid and other bacteria. Experimental evidence is against this view (**48**), and there is also the fact that in the majority of cases the contents of the gall bladder have been found sterile at operation. This suggests that infection, when it occurs, is only secondary, and that some other factor is involved in their formation possibly hypercholesterinæmia (**49**). Pure bilirubin-calcium calculi are formed mostly in the intrahepatic ducts.

**Effects of Gall Stones.**—The most important of these are—gall stone dyspepsia; cholecystitis; passage directly from the gall bladder into the bowel; impaction in the cystic duct; impaction in the common bile duct with its results; carcinoma of the liver, gall bladder and other parts.

1. Gall stones may remain in the gall bladder for years without giving rise to marked symptoms, but they cause some dragging pain in the right hypochondrium; and a group of symptoms suggestive of indigestion is recognised as occurring in patients who, it may be, years subsequently are found to have gall stones. These symptoms (*gall stone dyspepsia*) consist of attacks of pain or discomfort in the right hypochondrium, coming on half an hour or an hour after food, in some cases particular kinds of food, and especially fat; the pain is accompanied by flatulence, a sense of distension, some chilliness, and frequently vomiting, which relieves the symptoms.

2. A gall stone or gall stones can sometimes be felt through the abdominal wall as a hard mass, which may, in somewhat rare instances, give a sensation of crackling on being handled.

3. In many instances, at some time or other, infection occurs, followed by catarrhal or suppurative *cholecystitis*, with all the consequences above described (*see* p. 496).

4. It is not a very uncommon event for a gall stone to ulcerate through the wall of the gall bladder directly into the duodenum or into the transverse colon: generally it is a large gall stone, an inch or more in diameter, which, if it enters the duodenum, will probably be impacted in the lower part of the ileum (*see* p. 460), or, if it enters the colon, may be passed *per anum* after more or less pain and difficulty from obstruction at the sigmoid or near the anus. Somewhat rarely a large stone has been vomited, and the case has been recorded of a fat Russian woman who had symptoms of intestinal obstruction with vomit of a fæcal odour. Within forty-eight hours of the first symptoms thirty or more small faceted gall stones were passed, and some twenty-four hours later a large gall stone measuring 3 cm. in diameter (A. R. Neligan).

5. In consequence of increased pressure or of some irritation in the gall bladder, one or more stones may be forced out of the cavity and may pass down or be impacted in the cystic duct. The immediate effect is violent peristalsis or spasm of the duct and intense pain, and symptoms of *biliary colic*, but without jaundice. In the case of impaction bile is unable to enter the gall bladder, which becomes distended with mucus, or muco-pus, or pus; cholecystitis ensues, with gangrene or ulceration of the gall bladder, adhesion of the gall bladder to surrounding parts, and other similar changes. Sometimes a chronic impaction in the cystic ducts leads to shrinking of the gall bladder and obliteration of its cavity.

The symptoms may be relieved by the stone falling back into the bladder, or passing on into the common bile duct and duodenum.

6. After passing the cystic duct the stone enters the common duct, and also gives rise here to biliary colic, accompanied, however, by transient jaundice, owing to the resistance offered by the presence of the stone to the outflow of bile from the liver. If the stone is impacted in the duct, the jaundice is deeper;

obvious bile pigment is seen in the urine; at the same time the fæces are pale, clay-coloured or colourless.

In an attack of biliary colic, the patient is seized, often suddenly, with agonising pain in the right hypochondrium and lower part of the chest, or in the epigastrium and lower sternum; and it radiates to the right shoulder or left hypochondrium, or downwards to the groin. The pain is often so severe that he is bent double, or writhes on the floor or bed. Rigors may occur, and the patient is pale, collapsed, with profuse sweating and a small, feeble, generally quick pulse. After a time the pain becomes dull and aching, until a fresh attack of the acuter kind occurs, with perhaps a rigor; or the pain persists, and within a few hours or a day or two of the beginning of the pain bile pigment appears in the urine, and the patient becomes jaundiced. This is an indication that the gall stone is impacted in the common bile duct, so that an obstructive jaundice has resulted. All the signs of this condition are present—pronounced jaundice, bile pigment in the urine, and pale fæces. This may end by the passage of the stone into the duodenum, when the bile again flows freely, the pain subsides, and more gradually the jaundice clears up. When this happens, the fæces should be searched for the gall stone, which may be found by washing them with water and passing the washings through a sieve.

But the time which elapses between the impaction and the discharge into the duodenum is very variable; it may be several weeks, during which the patient remains jaundiced, and is subject to more or less pain, the liver being also somewhat enlarged and the gall bladder distended. Even then the stone may pass, and the patient is free from further trouble until another stone travels down the duct. In this way several attacks may occur, some of which may be slight and transitory, consisting of pain alone, others more severe and prolonged, with more or less jaundice.

On the other hand, there may be a permanent impaction of the gall stone in the common bile duct. This is not infrequently in the ampulla of Vater, at the termination of the bile duct, where the very small orifice into the duodenum offers greater resistance than the calibre of the duct itself. This continued obstruction has effects ($\alpha$) on the liver and its ducts, ($\beta$) on the gall bladder, and ($\gamma$) on the pancreas.

($\alpha$) The liver is at first considerably enlarged from dilatation of its ducts, which are distended with bile. Sometimes the ducts are dilated uniformly, at others more irregularly into globular cysts; after a time, too, their contents become mucous in character. They exert a certain amount of pressure on the tissue of the liver, and cause it to atrophy, so that subsequently the liver becomes smaller and rather flaccid. The ducts are obviously inflamed, constituting cholangitis; and one of the effects of this with the associated infection by the colon bacillus, streptococcus and other bacteria, is the occurrence of febrile attacks, which have been described under the name of *intermittent hepatic fever*. The febrile events are periodic or irregular in their occurrence, and resemble a malarial attack. There is a rigor with a rise of temperature to 102° or 103°, sweating pain in the hypochondriac, epigastric or lower sternal region, itching, vomiting, more or less jaundice, and tenderness in the region of the gall bladder. Sometimes the spleen is enlarged, and the blood may present a leucocytosis.

($\beta$) The effect of the impaction on the gall bladder is to cause its distension, so that it projects below the edge of the liver, nearly in the mammary line; here it can be recognised as a large tense cyst, globular or ovoid in shape (since its fundus is the part that is felt), freely movable, unless fixed by adhesions, and descending on inspiration with the enlarged liver. The bile which it contains gradually gets mixed with mucus, secreted from its lining membrane, and ultimately mucus may be present alone. When the downward flow of the bile is impeded by an obstructing calculus, bacteria, especially the colon bacillus, usually present in the ampulla of Vater, are enabled to get up the biliary passages,

and thus infective and suppurative cholangitis and cholecystitis, with gangrene or suppuration, are enabled to occur, and may be followed by abscesses in the liver, by septicæmia and by pyæmia.

(γ) When a gall stone is fixed in the ampulla of Vater, the relations of the pancreatic duct, which opens here, are of importance, since it will also probably be blocked with a retention of pancreatic juice. Micro-organisms will readily pass through the walls of the bile duct into the pancreas, and with the retained secretions set up acute or chronic pancreatitis (*q.v.*).

7. Another result of the presence and persistence of gall stones in the gall bladder is *carcinoma* of the gall bladder or bile ducts, and this has been found in about 5 per cent. of cases of cholelithiasis. The carcinoma may subsequently spread to the substance of the liver, to the colon, or to the duodenum. Experimentally, it has been shown that the introduction of gall stones and other foreign bodies into the gall bladders of guinea pigs causes carcinoma from the chronic irritation (**51**).

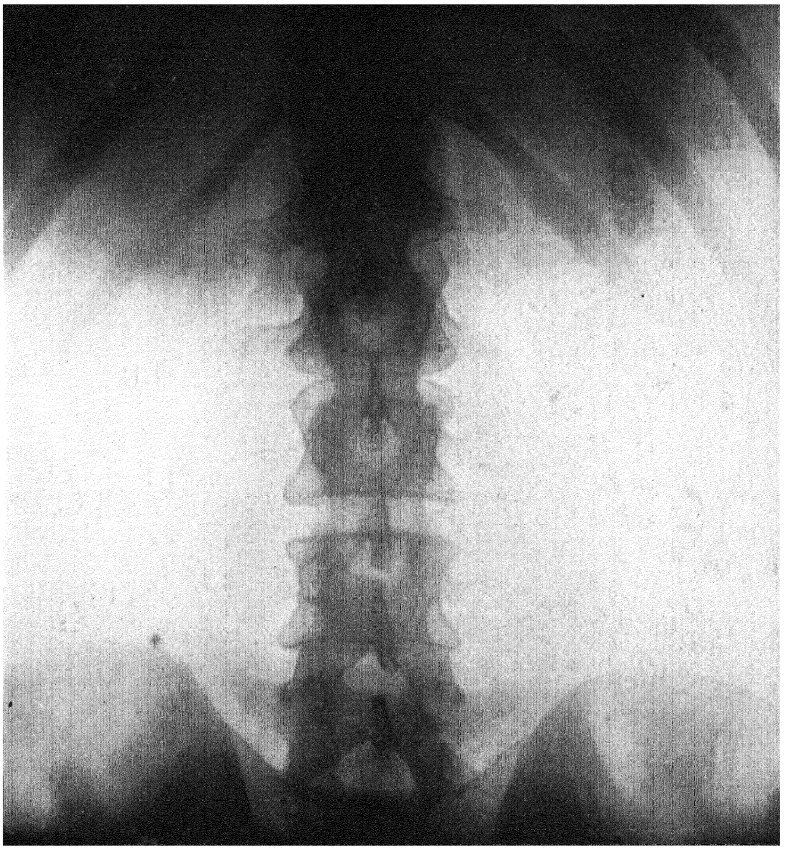
**Diagnosis.**—Biliary colic may be confounded with other sources of pain in that region: pleurisy, intercostal neuralgia, gastric pain, intestinal colic, renal colic, and appendicitis; these can generally be distinguished by the localisation of the pain or other features accompanying them. But the passage of gall stones is not always associated with pain, and cases of impaction with jaundice may be misunderstood from the absence of this symptom. In older cases, the history of repeated attacks is of great service. The recognition of the inflammatory complications described must depend on a careful analysis of the symptoms. In cases of jaundice due to obstruction of the common duct, if the gall bladder cannot be felt, the obstruction is due to gall stones; if there is a lump, the obstruction is probably due to some other cause, usually a growth (Courvoisier's law). This is correct in about 90 per cent. of cases in practice, and is due to the fact that inflammation of the gall bladder associated with gall stones causes fibrosis and shrinking up of the gall bladder. Gall stones differ considerably in the extent to which they intercept the X-rays, and thus reveal themselves by their shadows. Pure cholesterin gall stones are transparent to X-rays, and are therefore not shown by them. The bilirubin and calcium salts, when present, obstruct the rays, and the probability of detecting a gall stone by X-rays is in direct proportion to the amount of calcium salts it contains. A very characteristic appearance is a rounded opacity with an opaque centre and a clearer layer surrounding it (*see* Plate XXVIII.). The gall bladder can often be outlined by a radiogram after injecting oxygen into the peritoneal cavity.

Einhorn's duodenal tube provides a valuable method of diagnosing gall-bladder disease, though it will not differentiate between cholecystitis and cholelithiasis. After passing the tube into the fasting stomach, the patient lies on the left side until the passage into the duodenum has taken place, as is shown by the fact that on suction through the tube a finely frothing fluid, slightly bile-stained, and neutral or alkaline to litmus is obtained. After washing out the duodenum with sterile distilled water, 10 to 30 c.c. of a 25 to 50 per cent. solution of magnesium sulphate is injected into the duodenum. A copious flow of bile results, which is withdrawn and examined. The presence of cholesterin crystals and leucocytes is suggestive of gall-bladder disease. Cultivation may show the presence of *B. coli*, but this is not so characteristic, as it may be found in any case where the acid of the gastric juice is much diminished (**49**).

**Prognosis.**—This need not be unfavourable in a first attack of colic; many people recover even after several: but when jaundice is of long duration, the possibility of more serious causes, such as carcinoma, must not be forgotten.

**Treatment.**—When the presence of gall stones is recognised, or reasonably suspected, the diet should be carefully regulated and moderate in quantity, and cholesterin-containing foods, such as eggs, meat, and other cell-containing foods, should be limited, sufficient exercise should be taken, and alkaline waters

## PLATE XXVIII.



Radiogram showing two Gall stones in the Gall-bladder between the Eleventh and Twelfth Ribs. These are often not seen by X-rays; but in this case the appearances are highly characteristic. The stones are facetted so that they fit one against the other. In the upper stone there are two opaque foci and in the lower stone there is one. These are composed of calcium salts, chiefly compounds of calcium and bile pigment. The calcium is relatively opaque to X-rays. Round the foci is a clear zone of cholesterin, which is transparent to the rays, and round this again there is an outer shell of calcium salts.

[*To face p.* 500.

should be drunk freely. Those of Carlsbad, Vichy, Kissingen, Marienbad, and Ems are especially recommended. Supposed solvents of gall stones (sodium sulphate, carbonate and phosphate) are of doubtful efficacy; olive oil (2 to 10 ounces daily) seems to have done good in some instances. Sodium salicylate and urotropin promote the flow of bile, and may be given in doses of 10 grains three times a day. The daily "medical" drainage of the gall bladder by injecting magnesium sulphate into the duodenum through an Einhorn tube, as already described, has been recommended (**50**). The magnesium sulphate may also be given by mouth.

For an attack of biliary colic the patient should be placed in a hot bath, or hot fomentations or poultices should be applied to the right side. Most relief will be obtained from the subcutaneous injection of $\frac{1}{4}$ to $\frac{1}{2}$ grain of morphia, repeated, if necessary, in three or four hours; less speedy relief is given by opium, of which the dose may be 2 grains, followed by a grain every three hours till the pain is allayed. Sometimes chloroform may be inhaled with temporary relief. Papaverine hydrochloride ($\frac{1}{3}$ to $1\frac{1}{3}$ grain) by mouth may also be tried.

When the gall stones are a constant source of trouble, either by frequent attacks of biliary colic, or persistent gall stone dyspepsia, or by setting up cholecystitis with distension of the gall bladder, or by causing prolonged obstructive jaundice, the operation of cholecystotomy should be performed, and the stone or stones can be removed from the bladder or ducts. It may be advisable to remove the gall bladder (cholecystectomy).

## PERIHEPATITIS

**Pathology.**—Perihepatitis, or inflammation of the capsule of the liver, may be acute or chronic, localised or more generally diffused. It is set up by several of the lesions which occur in and about the liver, such as cirrhosis, lardaceous deposit, syphilitic disease, gall stones and cholecystitis, pylephlebitis, cholangitis, carcinoma, hydatid, and abscess, or by extension of inflammation from an ulcer of the stomach or duodenum or from the appendix, when this gives rise to a subphrenic abscess. In some of the most marked, and especially the chronic, forms, it is often only a part of a chronic peritonitis. In some such cases it is further associated with chronic mediastino-pericarditis, and with chronic pericarditis and pleurisy, forming, indeed, the complaint described as polyorrhomenitis (*see* p. 516). In other cases it is accompanied by chronic interstitial nephritis. It is often difficult to find a cause in these mixed cases, but spirit-drinking is not responsible for them, and the influence of micro-organisms is not yet clearly shown.

When the liver is affected with perihepatitis, the capsule is opaque and more or less thickened; often the thickening is distributed in patches irregularly over the surface; and such patches may be determined by the disease which causes the inflammation of the capsule. Sometimes the liver is completely enclosed in a membrane, or in a thick casing, from 2 to 10 mm. in thickness (German *Zuckergussleber*, sugar-icing liver). In such cases the organ has a rounded anterior edge, which is due to the actual margin of the liver being bent back upon the upper surface and held there by the thick capsule. This membrane may be stripped off without destroying the tissue beneath. The liver is rarely cirrhosed, but is generally soft and often fatty. In the severer forms ascites is generally present; and this has been supposed to arise either from constriction of the portal vein in the portal fissure, or from compression of the whole organ by its thick capsule, so as to hamper the portal circulation, or, lastly, to be independent of the liver, and to result from the accompanying peritonitis. The spleen is often at the same time similarly affected (*perisplenitis*).

**Symptoms.**—The perihepatitis which arises in consequence of localised diseases of the liver, such as hydatid, abscess, carcinoma, or gall stone, especially

if acute in its occurrence, may give rise to severe pain and tenderness on manipulation, and a friction sound may be heard, or the rub can be felt on laying the hand over the liver.

In the more extensive cases associated with general chronic peritonitis also, the early symptoms may be acute, with fever, pain and malaise; but these symptoms are not always present, and the early changes may be quite insidious.

In all cases ultimately ascites occurs; the liquid is abundant and appears again and again after paracentesis. There is sometimes a slight amount of anasarca, especially when the pericardium is involved. Jaundice is absent.

**Treatment.**—The pain of acute attacks may be relieved by opium and local anodynes. In the chronic cases paracentesis will require to be performed frequently.

## PYLEPHLEBITIS

This occurs in two forms, adhesive and suppurative, which have been already referred to, the one as a cause of ascites (*see* p. 477), the other in connection with multiple abscesses of the liver (*see* p. 479).

### ADHESIVE PYLEPHLEBITIS

This is more generally a thrombosis of the portal vein, in which the clot adheres to the wall of the vein, and becomes ultimately organised in the same way as a thrombus in any other situation. Its causes are those changes which bring about retardation of the blood current in the portal vein or its distribution, such as cirrhosis, syphilitic disease, the pressure of tumours on the trunk of the vein or its implication in perihepatitis, or chronic peritonitis near the fissure of the liver.

**Symptoms.**—The obstruction to the portal vein leads to symptoms closely resembling Banti's disease, viz. splenic anæmia combined with cirrhosis of the liver. There are engorgement of the portal vein radicles, showing itself in ascites, with enlarged spleen, diarrhœa, and hæmorrhage from gastric and intestinal vessels, and anæmia. The collateral circulation becomes developed in the same way as in cirrhosis, and the superficial abdominal veins are commonly enlarged, relieving thus for a time the portal vessels. The urine is scanty. Jaundice is rare, and only occurs as a result of the lesion which causes the pylephlebitis. The liver is generally smaller than normal.

The anæmia should be treated.

### SUPPURATIVE PYLEPHLEBITIS

This is nearly always due to infection from lesions in the abdomen—*i.e.* the area from which the blood is supplied to the portal vein—such as appendicitis (the most common cause), ulcers of the rectum, colon, or small intestines, gastric ulcer, and suppuration of the mesenteric glands, pancreas, or spleen. Nearer the liver gall stones may cause inflammation of the portal vein branches, and the same may happen from hepatic abscess or a suppurating hydatid cyst. In the new-born child the portal vein may be infected from a septic phlebitis of the umbilical vein, and rarely the lesion may be caused by direct injury.

The mischief commonly begins either in the branches or in the tributaries of the portal vein rather than in the trunk itself, to which, however, the suppurative process may ultimately extend. The wall of the vein inflames and suppurates, a thrombus forms in the neighbourhood, breaks down into pus, and its conveyance to other parts of the vessel sets up fresh centres of thrombosis, phlebitis, and suppuration. Finally, in many cases, multiple small abscesses of the liver are formed. The liver is enlarged, soft, flaccid, and anæmic. The branches of the portal vein are filled with disintegrating thrombi, or pus, or grumous fluid; and the walls of the corresponding veins are infiltrated or ulcerated. The spleen is enlarged, and there is occasionally peritonitis.

The **Symptoms** are nearly the same as those of multiple abscesses. There are epigastric and hypochondriac pain, fever of hectic type, rigors, sweating, vomiting, anæmia and prostration. The portal vein may be sufficiently obstructed to cause some ascites, and the spleen is enlarged, partly on this account, partly as a result of septic fever. Jaundice is often, but not always, present; and if abscesses are numerous, there may be enlargement of the liver. The fæces generally contain stercobilin. A typhoid condition supervenes with stupor and delirium, and the disease generally progresses to a fatal termination in from one to seven or eight weeks, exceptionally much longer.

**Diagnosis.**—The disease is easily overlooked. It may be confounded with pyæmia, septicæmia, malarial fevers, acute yellow atrophy, tropical abscess, subphrenic abscess, typhoid fever, or pneumonia. Fever with rigors, local evidences of the liver being involved, such as pain, swelling and jaundice, evidence or history of a local source of infection, and signs of portal obstruction, such as diarrhœa and enlargement of the spleen, point to suppurative pylephlebitis. When local signs are absent, the fact of an obvious pyæmia without external wound, and without endocarditis, might suggest some abdominal organ as the source of the sepsis.

**Treatment.**—The almost necessarily fatal course of the disease renders treatment useless, except as applied to the relief of pain, sleeplessness, and other symptoms.

# DISEASES OF THE PANCREAS

The pancreas contains two different types of secreting cells: (1) *acinar* cells, which secrete the pancreatic juice; (2) cells of the *islands of Langerhans*, which secrete a hormone necessary for the metabolism of carbohydrate. These cells may be affected by disease independently of one another. Thus in diabetes mellitus (*see* p. 562), which is a disease of the islands of Langerhans, there is usually no abnormality in the secretion of pancreatic juice. At the same time it is doubtful how far a pancreatitis may affect the acinar cells without at the same time causing some changes in the island cells, so as to produce an effect on carbohydrate metabolism, detectable by the most refined methods of investigation.

The pancreas itself is not easily palpable, being deeply seated, and only occasionally in thin people can it be felt lying transversely across the aorta. Even when enlarged by carcinoma, or chronic inflammation, it may be entirely concealed by the overlying liver. Cysts of the pancreas are, however, often large enough to form considerable tumours in the upper part of the abdomen.

## FUNCTIONAL TESTS OF THE PANCREAS

Most of the functional tests of the pancreas consist in determining whether there is a deficiency (*a*) in the external secretion from the acinar cells, (*b*) in the internal secretion from the islands. However, Loewi's *mydriatic test*, which is only positive in a proportion of cases, and is also positive in some cases of exophthalmic goitre, depends on some functional connection of the pancreas with the sympathetic system. The eyes are examined to see whether the pupils are equal and react normally. Into one of them a solution of adrenin (1 in 1,000) is dropped and the eye closed. This is repeated twice during five minutes. The eyes are not used for an hour. If during this time the pupil of the adrenalised eye is larger than that of the other eye, which is used as a control, the test is positive and indicates pancreatic disease.

**Deficiency of External Secretion.**—*Steatorrhœa* has up to the present been regarded as one of the most characteristic signs of pancreatic disease. It consists in the passage of liquid fat in the stools, which solidifies on cooling,

forming white or yellow lumps. The stools are bulky, soft and pale, and besides unaltered fat they may contain fatty acids and soaps. Where the stools are not obviously altered to the naked eye the microscope may reveal numerous fat globules and fatty acid crystals. In estimating the degree of steatorrhœa chemically, it is important to determine not only the fat in the stools, but also to know the fat taken in the diet. In severe cases from 50 to 60 per cent. of the fat intake is lost in the stools. The normal loss is only about 10 per cent. In sprue it may be 25 per cent.

There are two reasons for doubting that steatorrhœa is usually pancreatic in origin: (1) because it very commonly arises from defective absorption where the lacteals are obstructed from abdominal tuberculosis or growth (*see* p. 515). In these cases the fat in the stools is largely split. (2) Evidence is forthcoming that the presence of bile in the intestine is a much more important factor in fat absorption than the pancreatic juice. In fact, experimental ligature or extirpation of the pancreatic ducts do not seem to affect the absorption (**44**). Congenital steatorrhœa has been described, and in diarrhœa and cœliac disease the fat in the stools is increased.

*Creatorrhœa* is due to the absence of trypsin. Large numbers of undigested striped muscle fibres, derived from the meat eaten, are observed microscopically in the stools. Creatorrhœa also occurs in profuse diarrhœa from any cause.

*Diastase in the Urine.*—The test is carried out on a twenty-four hours' specimen. To get accurate results it is essential first of all to titrate the urine with $\frac{N}{10}$ NaOH or HCl until the acidity is optimum, *i.e.* the pH of the urine is 6·7. This is the case when after the addition of phenol red as an indicator the urine shows a faint pink tinge (**56**). 0·6, 0·4, 0·2, and 0·1 c.c. of the urine are put in four tubes and normal saline (0·9 per cent.) added, so as to make 1 c.c. in each case. The same procedure is adopted after the urine has been diluted ten times with normal saline, so that the eight tubes contain strengths of urine corresponding to 0·6, 0·4, 0·2, 0·1, 0·06, 0·04, 0·02, 0·01. To each tube are added 2 c.c. of 0·1 per cent. soluble starch. The tubes are incubated for half an hour at 39° C. After cooling, $\frac{N}{50}$ iodine is added drop by drop to the tubes in series, starting from the tube containing 0·01 strength of urine, until there ceases to be a change of colour. The first tube in which the blue colour ceases to be developed gives the amount of diastase in the urine, which is reckoned by dividing 2 by the strength of the tube. If, for example, the blue colour is not found in the 0·06 tube, the amount of diastase would be 33 units. The normal amount of diastase varies between 10 and 40. In pancreatic disease the amount of diastase in the urine (*i.e.* the diastase index) is increased. It is possible that this test is really concerned with the *internal* secretion of the pancreas. Cammidge believes that a deficiency of this leads to liberation of hepatic diastase into the blood and its increase in the urine.

**Deficiency of Internal Secretion.**—In a marked case this leads to glycosuria, and in less marked cases there may be a decrease in *sugar tolerance.* This is best tested by giving a patient a dose of dextrose and observing the rise in the blood sugar, as well as whether any sugar is excreted in the urine (*see* p. 564). Recent work by Cammidge, Forsyth and Howard is of very great theoretical interest, and may lead to valuable means of detecting a deficiency of the internal secretion of the pancreas in its earliest stages. They consider that the liver contains an amylolytic ferment which breaks down glycogen through a stage of dextrin-like bodies into dextrose. The activity of this ferment is normally kept in check by the internal secretion of the pancreas. If the latter is deficient, there is first of all an excess of these dextrins in the blood and in the urine, into which they are excreted, while the blood sugar remains at about its normal level. In

a later stage these dextrins are broken down completely into dextrose, so that there is now hyperglycæmia and glycosuria, while the dextrins disappear from the blood and urine. This latter is the condition found in diabetes mellitus. The amount of dextrins in the blood was measured by hydrolysing them with hydrochloric acid so that they were converted into dextrose. The total dextrose was then estimated. The "difference value" between this quantity and the amount of dextrose present in the original blood before hydrolysis gives the amount of dextrose which was due to the dextrins. The same method may be used for the urine, or a direct method involving the use of iodine can be employed. These determinations will probably take the place of the original Cammidge's reaction, which is not reliable.

## ACUTE PANCREATITIS

**Pathology.**—Experiments and clinical observations show that acute pancreatitis is in the majority of cases due primarily to bacterial infection, which activates the pancreatic juice by converting the trypsinogen into trypsin. This activation normally only takes place in the duodenum, owing to the presence of enterokinase. The characteristic appearances of the diseased gland are due to its digestion by the active pancreatic juice. Acute pancreatitis occurs most commonly in association with gall stones or cholecystitis; but it is not very common for a gall stone to be found impacted in the ampulla of Vater, and when this is the case the stone is probably large enough to block the exit of Wirsung's duct, and so prevent bile from passing up it into the pancreas. However, it is possible that small stones cause intermittent stasis in the pancreatic duct by impaction at the ampulla, and the associated infection spreading from the bile ducts starts the attack. It is quite common for a history of repeated mild attacks of biliary colic to be obtained, which would fit in with this hypothesis. Acute pancreatitis may start from other neighbouring foci of infection, such as duodenal ulcer and cholangitis.

Acute inflammation of the pancreas sometimes occurs in infective diseases, such as enteric fever, pyæmia, and septicæmia, and also in mumps, which is of interest from the structural resemblance of the pancreas to the salivary glands.

In *acute hæmorrhagic pancreatitis* the organ is swollen, friable, and mottled red or brown to black, with hæmorrhages on the surface and in the interstitial tissue; and the blood may extravasate in the surrounding structures, or there may be blood-stained fluid in the peritoneal cavity. Microscopically the parenchymatous tissue is found to be necrosed, and there is usually associated inflammation, as shown by infiltration with polymorphonuclear cells. This is probably secondary to the digestion by pancreatic juice. *Fat necrosis* is always a prominent feature. In the pancreas, and in the subperitoneal fat adjacent, sometimes in the perinephric, mediastinal, and pericardial fat and even in the subcutaneous fat, are small masses of dull yellow or opaque white colour, sharply differentiated from the adjacent healthy fat, and sometimes surrounded by a narrow hæmorrhagic zone. They are produced by the action of the fat-splitting ferment (*lipase*) of the escaped pancreatic secretion upon the fat, of which the fatty acids combine with calcium bases, while the glycerine is absorbed.

At a later stage, or if the process is more chronic, *suppurative pancreatitis* occurs. The organ is large, swollen and infiltrated with pus; or it contains separate abscesses. An abscess may burst into the peritoneal cavity, or into the stomach or bowel; thrombosis and infection of the portal and splenic veins may also occur with metastatic abscesses in the liver. The *Bacillus coli communis* and pyogenic organisms are found in different cases. In both forms death may follow from acute peritonitis.

**Symptoms.**—When pancreatitis occurs in the course of mumps, there

are vomiting and epigastric pain, with swelling and tenderness in the epigastric region. The more intense and hæmorrhagic forms are characterised by severe, even excruciating, pain in the upper part of the abdomen, tenderness, muscular rigidity, gradually increasing distension, nausea, vomiting, and collapse. In the hæmorrhagic cases, the symptoms often come on quite suddenly in the midst of apparent health, and they have been constantly mistaken for those of intestinal obstruction, or peritonitis from perforation of a gastric ulcer, or biliary colic. Often there is nothing distinctive; sometimes, after a few hours, a circumscribed, tender swelling appears in the upper part of the abdomen, but the difficulties of diagnosis are such that the abdomen has often been opened for the relief of a supposed intestinal obstruction. The cases are generally fatal within four or five days, but some have recovered after laparotomy. The symptoms of suppurative pancreatitis are similar, but less pronounced and acute, and the case may last several months. A tumour is only felt in one quarter of the cases, and this is due to accumulation of fluid in the lesser peritoneal sac. Symptoms referable to failing pancreatic functions have not very often been reported, but this is probably because they have not often been looked for. Glycosuria and an increase in the urinary diastase would be a valuable help in diagnosis. Jaundice is sometimes present.

**Treatment.**—Laparotomy should be immediately performed if the symptoms are urgent. The condition that is found may suggest local measures. Thus with a hæmorrhagic lesion the organ has been incised, the hæmorrhage checked by ligature, and drainage instituted with success. In a case of suppurative pancreatitis, incision of the abscess and drainage are absolutely necessary. In any operation for either lesion the common bile duct should be examined, and gall stones, if present, should be removed, and the gall bladder drained.

## CHRONIC PANCREATITIS

This affects the interstitial tissue, producing considerable fibrous growth, with consequent atrophy of the glandular structures, analogous to the changes in cirrhosis of the liver; and, as in that disease, the fibrous network may enclose large groups of acini (*interlobular*), or much more rarely single acini (*interacinar*). The head of the organ is usually most affected. The substance is rendered extremely dense and hard, and in the less frequent interacinar form considerable enlargement may take place. The ætiology of chronic pancreatitis is much the same as that of acute pancreatitis. It commonly arises from the spread of adjacent inflammations, such as those of the peritoneum, of the bile duct, of the stomach and of the intestines, such as appendicitis, by which infective organisms may be conveyed up the pancreatic duct. It also results from the presence of concretions in the pancreatic duct, or of retained pancreatic secretions; from compression of the duct by cancer; from the venous congestion of heart disease; and possibly from syphilis, and the abuse of alcohol. Gall stones are a common cause of chronic pancreatitis, especially when one lies in the ampulla of Vater, or in the common duct, or when there is suppurative cholangitis, the result of their presence.

In most of these instances of duct obstruction and infection it is the interlobular form which is caused. Arterio-sclerosis is also a cause of chronic pancreatitis, which is then commonly of the interacinar variety.

**Morbid Anatomy.**—In the interlobular form the pancreas is dense and hard, with broad bands of fibrous tissue running between the lobules and embedded areas, some of disintegrating glandular substance, some of well-preserved tissue. As a rule the islands of Langerhans are untouched. In the interacinar form the acini are separated from one another by coarse strands of fibrous tissue, the acini are atrophied, and the islands of Langerhans are frequently involved in lymphoid-cell infiltration and sclerosis.

Either form of chronic pancreatitis may be complicated by a large deposit of fat, *lipomatosis*, especially in persons the subjects of obesity.

**Symptoms.**—The symptoms attributed to chronic pancreatitis are uneasiness and distension in the epigastrium after food, anorexia, nausea, lassitude, and drowsiness, and later colicky pains and borborygmi, anæmia and emaciation. These symptoms are not, however, always prominent; and the fact of chronic pancreatitis has often been revealed at an operation undertaken to relieve some one of the various results of gall stone, of which jaundice and epigastric pain are the most common. But, apart from gall stones and often without pain, the swollen head of the pancreas may compress the common bile duct, which it surrounds, and thus cause jaundice and distension of the gall bladder; and these will be associated with the characteristic stools, and possibly glycosuria or other urinary indication. Mayo Robson records cases where chronic pancreatitis has resulted in glycosuria and fatal diabetes. If the body of the gland alone is involved, jaundice is absent, and the symptoms may be few or none unless the enlargement is so great as to render the organ palpable.

Where diabetes mellitus results from chronic pancreatitis, Opie states that it results almost entirely from the interacinar form, in which especially the islands of Langerhans are injured, and from the interlobular form only in extreme instances when the islands, situated in the centres of the lobules, are reached by the degenerative process.

**Treatment.**—As the chronic inflammation is attributable in so many cases to disordered conditions of the biliary and pancreatic ducts, and of the gastro-intestinal mucous membrane, the treatment of these primary disorders is the first consideration. The former will often require operation for the removal of calculi, and the latter will need suitable dietetic and medicinal treatment. Cases have been reported where prolonged drainage through the gall bladder has been successful in relieving symptoms.

## PANCREATIC CONCRETIONS

These may occur in middle-aged men; they are by no means common. They are attributable to catarrh of the ducts with delayed secretion, and consist of calcium carbonate and calcium phosphate, and sometimes calcium oxalate. They may be like grains of sand, or as large as hazel-nuts, and are usually round or oval, occasionally irregular or branched. In colour they are white or greyish white, sometimes brown or nearly black. They sometimes block the duct or its branches, and lead to dilatation of the ducts, retention cysts, acute inflammation with suppuration or chronic induration, and even to inflammation in the parts around. They rarely produce symptoms, except through their secondary effects—for instance, by the inflammation which they excite, or by the formation of cysts, or by the production of atrophy and cirrhosis of the gland.

Pancreatic calculi have occasionally been removed successfully by operation, in most cases from the duct of Wirsung.

## TUMOURS OF THE PANCREAS

Carcinoma, which is nearly always primary, is the most important tumour of the pancreas. It is often confined to the head of the gland. It is an irregular nodular hard tumour, which may be of sufficient size to be felt under favourable circumstances through the abdominal parietes. As the nodules increase in size the pancreatic duct is liable to be obstructed, with the formation of a cyst as a result; and the common bile duct is not infrequently blocked either by pressure or by the spread of a chronic inflammation, so that jaundice is produced. This is, indeed, a common cause of jaundice in persons of middle and advanced age.

In other instances the carcinoma may involve the stomach, duodenum, peritoneum, vertebræ, or other structures. The symptoms are variable; pain may be absent, but it is sometimes deep-seated, of aching, gnawing, lancinating, or burning character, often distinctly paroxysmal in its occurrence, and affected by food, coughing, deep breathing, movement, or posture. Nausea and vomiting may be present, and the stools are often fatty or contain undigested muscular fibres. Examination may reveal a tumour, of the characters described, in the situation of the head of the pancreas. In the later stages emaciation, anæmia, and prostration become prominent features of the case.

*Pancreatic Cysts.*—These are usually the result of obstruction of the duct of Wirsung by calculus, or by pressure from without; they may reach a considerable size and hold many pints of fluid, and are hence *retention cysts*. Others appear to be formed in the substance of the gland as the after result of a hæmorrhagic pancreatitis, and contain altered blood pigment. Possibly some are traumatic.

A cyst of sufficient size forms a globular tumour in the upper part of the abdomen, either in the median line, or in the left hypochondrium; occasionally it projects below the transverse colon. At first behind the stomach and colon, it may, if it is large, press the hollow viscera aside. A pancreatic tumour is often stationary during deep inspiration, but if in contact with the diaphragm, it may move downwards $\frac{1}{2}$ or $\frac{3}{4}$ inch. The surface is dull or resonant, according to the extent to which it is covered by either of these hollow viscera. The fluid within it is turbid, brown or greenish in colour, alkaline, of specific gravity 1,010 to 1,020; it contains albumin, sugar, mucin, and a trace of urea; and it may show one or more of the properties of the pancreatic secretion, namely, that of digesting proteids (*proteolytic*), of digesting starch (*amylolytic*) or of emulsifying fat (*lipolytic*). Hæmorrhage may take place into the cyst. There is emaciation and sometimes pain or jaundice. The urine sometimes contains sugar.

The swellings most likely to be confounded with it are a hydatid cyst of some other organ, hydronephrosis, circumscribed peritonitis, and ovarian disease; but if much to the left and moving on inspiration it may resemble a splenic or renal tumour. The nature of the aspirated fluid should help. Congenital cysts and hydatid cysts of the pancreas occur rarely.

**Treatment.**—Pancreatic cysts have often been successfully treated by incision and drainage. Other tumours are less easily dealt with, and treatment must be directed to the relief of symptoms. If the tumour cannot be removed, some relief may be obtained by cholecystotomy or cholecyst-enterostomy.

# DISEASES OF THE PERITONEUM

## PERITONITIS

The peritoneum lining the surface of the abdomen, and covering nearly all the viscera contained within it, is liable to inflammation from a number of causes originating in these organs as well as from more widespread infection. This inflammation may be acute or chronic, and general or circumscribed.

### ACUTE PERITONITIS

**Ætiology.**—The most frequent cause is some lesion of the abdominal viscera or adjacent parts, such as ulceration of the stomach, typhoid and tuberculous ulcers of the ileum, dysenteric ulcers of the colon, inflammation and sloughing of the appendix cæci, abscess of the liver, suppuration of the gall

bladder, infarction and abscess of the spleen, the numerous inflammatory lesions which are apt to involve the female pelvic organs—metritis, parametritis, ovaritis, salpingitis, and pelvic hæmatocele.

In many of these cases the peritonitis is set up by the discharge into the abdominal cavity of liquids, such as food, fæces, or pus, carrying with them infective micro-organisms; this happens in the case of the perforation of gastric and intestinal ulcers, in appendicitis, and in rupture of abscesses. In other cases there is an extension of inflammation to the serous layer; that is, the micro-organisms penetrate the tissues without coarse rupture. Peritonitis is the natural termination of most cases of intestinal obstruction, either from local inflammation, as in acute strangulation and hernia, or from rupture of an over-distended gut, as in the more chronic strictures. Perinephric and psoas abscesses may rupture into the peritoneum, and empyema occasionally sets up inflammation below the diaphragm, though it is much less common than pleurisy and empyema, as a result of a peritoneal abscess. Wounds of the peritoneum, whether from injury or surgical procedure, are liable to be followed by peritonitis.

Infection of the peritoneum from the blood apart from local lesions is less common. Pneumococcal peritonitis may be primary; but it may be associated with a thoracic lesion, such as pneumonia or pleurisy, and be due to a pneumococcal septicæmia. Gonococcal peritonitis is generally associated with vaginitis. Primary streptococcal and staphylococcal peritonitis are rare. Bright's disease, whether acute or chronic, is an occasional predisposing cause of peritonitis, which is then generally fatal.

**Morbid Anatomy.**—The changes which take place in the peritoneum are not unlike those which occur in the pleura when it is inflamed. There is at first redness from increased vascularity, and if the cavity of the abdomen is examined in this early stage, the redness of the intestines is commonly seen to form parallel streaks along the intestine in the position where three peritoneal surfaces meet, *i.e.* two coils of intestine and the anterior abdominal wall or three coils of intestine. The pressure is lower at this position than elsewhere, so that congestion and exudation occur first here, forming a space along the gut which is triangular in section. The exudation coagulates, producing a deposit of fibrin, and pus may also be formed. The process then becomes more general. There is at first a mere stickiness of the peritoneal surface, but the exudation soon becomes more abundant, forming yellow flakes, coating the surface of the bowel, or collecting in larger masses in the triangular spaces already mentioned. The yellow flakes, which are often loosely spoken of as *lymph*, consist of fibrin and leucocytes, and there may be present a varying amount of turbid fluid in addition. The exudation appears with great rapidity, as may be seen in some traumatic cases, where a quantity of yellow lymph may be formed in less than eighteen hours. In some less severe or less extensive cases, the exudation may be replaced by fibrous tissue (*organisation*); and the different viscera are united together, or the peritoneal cavity is obliterated, by the adhesions which are thus formed.

In other cases, the quantity of leucocytes increases, or is more numerous, from the first, and the inflammatory products are entirely purulent; this is often quickly fatal, but occasionally a large peritoneal abscess may slowly form and offer chances of recovery. Collections of pus, localised or occupying the whole peritoneal cavity, occur as a result of pneumococcal peritonitis, in which, however, the effusion may be serous or sero-fibrinous as well as purulent.

Acute peritonitis is sometimes, from the first, circumscribed, and results in a localised abscess which may point externally or open into one of the hollow viscera. Such abscesses occur in the pelvis or between the diaphragm and the liver, or between the diaphragm and the spleen. In these last two situations they may rupture into the chest, and set up pleurisy or pneumonia. A peritoneal abscess not infrequently contains air, either from direct communication with the

stomach or intestine by perforation of an ulcer, or from decomposition induced by contact with the coats of the bowel and transference of micro-organisms.

**Bacteriology.**—The micro-organisms causing peritonitis are usually the *Bacillus coli communis* when the peritoneum is infected from the intestine, as in appendicitis or perforation of the bowels, or from the biliary passages; streptococci and staphylococci are found in peritonitis derived from lesions of the pelvic organs, or the abdominal walls. These three organisms are often present together. The pneumococcus is the next most frequent organism, and others less frequently found are *B. typhosus*, *B. pyocyaneus*, *B. lactis aerogenes*, *Micrococcus tetragenus*, and the gonococcus. The amœba coli has been found in amœbic dysentery; the tubercle bacillus occasionally causes acute inflammation, but much more commonly a chronic form.

**Symptoms.**—*Acute general peritonitis* begins with pain, which is mostly very severe, and, if at first localised to one spot, soon becomes diffused over the whole abdomen. The pain is constant, but aggravated by every kind of movement, by coughing, straining, or vomiting. It is not relieved by pressure; on the contrary, there is marked tenderness over the whole of the abdomen. Vomiting, as a rule, soon sets in, and occurs repeatedly, either spontaneously or after attempts to take food. At first the gastric contents are brought up, subsequently bile, and later still, in some prolonged cases, the vomited matters may have an almost feculent character. The temperature commonly rises, reaching 102° or 103°, more rarely 104° or 105°; and the pulse is quick, 100 to 120. Sometimes also rigors occur at the commencement, but there is always a considerable degree of collapse.

In some cases of perforation of gastric or intestinal ulcer, death occurs from collapse in twenty-four hours. In others, the patient is soon obliged to take to his bed, and within a short time lies on his back, with shrunken face, dark sunken eyes, anxious expression, dry furred tongue, and quick small pulse. The legs are frequently drawn up to prevent stretching of the abdominal parietes, and every movement is avoided by the patient. The abdomen is at first tense, with rigid muscles and immobility during respiration. Later it becomes swollen from paralysis of the muscular coat of the intestines and the accumulation of gas within them. The surface is resonant, but if much fluid is poured out, it may cause dulness at the flanks, or occasionally all over. Gas may escape into the peritoneal cavity, and cause extensive resonance or even splashing from mixture with the liquid. The extravasation of gas into the peritoneum is sometimes recognised by its lying in front of the liver and replacing the natural hepatic dulness by resonance. But it must be remembered that the liver may be displaced from its contact with the anterior thoracic wall by much gaseous distension of the viscera, without any escape of gas from their interior. The tension of the abdomen causes pressure on the under-surface of the diaphragm, with consequent dyspnœa, and generally the respiratory movements are entirely thoracic. Hiccough is also a frequent symptom. The bowels are, as a rule, confined; sometimes, after two or three days, one or more motions may be passed, or even diarrhœa may set in; and occasionally there is diarrhœa from the first. The urine is scanty; it may be passed with pain, or be retained.

The patient gradually gets exhausted by vomiting and pain, the tongue becomes drier and brown, sordes form on the lips and teeth, the pulse is smaller and quicker, the bases of the lungs are compressed, and after an illness of from two to six days death takes place. It is not, however, every case that presents all the characteristic signs. Fever is absent in some cases; there is but little distension in others; occasionally a patient, instead of lying prostrate on his back, will throw himself about in the agony of pain.

In pneumococcal peritonitis, which usually arises as part of a pneumococcal septicæmia, rigidity and tenderness of the abdomen is a marked feature. The temperature is very high, and the respiration rate is raised, and there may be

evidence of pneumococcal infection elsewhere, in the lungs, joints, etc. After a time an abscess forms in one part of the abdomen, and this is indicated by the appearance of a swelling in the particular region.

In *acute circumscribed peritonitis* (of which many cases of appendicitis are examples), the general symptoms are much the same, but the local conditions are more or less limited to the region affected. If pus forms, a more or less defined tumour may be recognised, and fluctuation may be detected. The constitutional symptoms are often less severe than in general peritonitis, and the case may possibly run on into a chronic stage. The fever is variable. The later course of the illness is determined by the behaviour of the inflammatory products; a sero-fibrinous peritonitis may subside; a peritoneal abscess may burst into different cavities, or set up inflammation in the chest, in which case the symptoms of pneumonia, pleurisy, empyema, or pneumothorax may complicate the abdominal lesion, and a fatal result is rendered highly probable, but not absolutely certain. Disease of the appendix cæci and lesions of the uterus and its appendages are the more common causes of acute circumscribed peritonitis.

**Diagnosis.**—As a rule, this is not difficult: the severe pain, tenderness, vomiting, rigidity, and immobility of the abdomen during respiration, followed by distension, constipation, small quick pulse, and collapse, form the important features. But peritonitis may be simulated by the severe pain of colic, by ruptured aneurysm, and by acute hæmorrhagic pancreatitis; it may itself be mistaken for intestinal obstruction; and it may be set up, and cause death without its presence being suspected, in enteric fever, and after operations on the abdominal walls, such as herniotomy. *Colic* and *hysterical pain* are mostly to be distinguished from peritonitis by the contracted abdomen and the absence of tenderness, indeed the relief on pressure, in the former case, and the extreme sensitiveness to the merest touch, without pressure, in the latter. Indications of lead-poisoning in the former or a history of hysterical attacks in the latter would assist the diagnosis. A high temperature or extreme collapse is in favour of peritonitis. A *ruptured aneurysm* causes pain and collapse, and may be mistaken for the perforation of a gastric ulcer; and the antecedents in the two cases may be difficult to discriminate. Perforative peritonitis is sometimes simulated by the fatal coma of *diabetes*. This often begins suddenly, with severe abdominal pain and small, thready pulse. The air hunger of diabetes and the presence of much aceto-acetic acid in the urine and absence of abdominal rigidity should clear up the diagnosis. Peritonitis resembles *intestinal obstruction* in its pain, distension, vomiting, and constipation, which may hamper the diagnosis for two or more days; the generally diffused tenderness, early distension, and simple gastric or bilious vomiting, are in favour of peritonitis; and this is often at length confirmed by the passage of fæces. The onset of peritonitis in typhoid fever may be very insidious; the patient is perhaps semi-comatose or delirious, and his senses are dulled; the abdomen is already much distended and tense. On the other hand, the tympanites and pain of this fever may be thought to be due to peritonitis when none exists.

As to the differential diagnosis of peritonitis, its cause must be looked for in the preceding history. Where severe acute peritonitis ensues in a person previously considered well, ulceration of the appendix cæci, perforating gastric ulcer, and lesions of the pelvic organs are the most likely causes. The first is more probable in both sexes before and about the age of puberty; the last occurs almost exclusively in females, and in girls the possibility of a neglected vulvo-vaginitis causing gonococcal peritonitis should be thought of. Perforations of the intestines, whether typhoid, tuberculous, or dysenteric, are generally preceded by recognisable illnesses; but the deceptive mildness of some cases of typhoid fever must not be forgotten.

Pneumococcal peritonitis may be suspected if there is a sudden onset of abdominal pain, tenderness, sickness and rigidity or distension in the course of pneu-

monia, pleurisy or empyema, or if herpes labialis or local inflammations such as arthritis, meningitis, nephritis, or endocarditis accompany an acute peritonitis. It must not be forgotten, however, that basal pneumonia or pleurisy will cause pain over the abdomen.

**Prognosis.**—General peritonitis is a very fatal disease. The probable result must be estimated by the character of the pulse, the persistence of vomiting, the amount of collapse, and the probable extent of the inflammation. Severer cases can only be judged of from day to day. There is more hope when some days have elapsed, but in cases that are apparently improving accumulations of pus may reveal themselves, and become dangerous in the way indicated. Pneumococcal and gonococcal forms are relatively favourable.

**Treatment.**—The majority of cases of peritonitis, and especially those due to perforation of a gastric, duodenal, or typhoid ulcer, or sloughing of the appendix cæci, or other similar accident, are only likely to recover if promptly treated by surgical methods: the abdomen must be opened, and the causative lesion dealt with, and collections of pus must be drained. One of the first considerations then is whether the case is one of this kind. Moreover, if the origin of the peritonitis is doubtful and the patient is acutely ill, it is safer to do an exploratory laparotomy, and deal with whatever lesion is found, than to delay until a fatal septic condition is established. In peritonitis having its origin in a septicæmia, *e.g.* pneumococcal peritonitis, an operation should not be carried out unless an abscess has formed, since it will not be possible to remove the primary focus.

Exceptionally a peritonitis of slight extent and less acuteness may be justifiably treated by other means. The first principle of treatment is to keep the intestines completely at rest. For this purpose the patient must, of course, be in bed; food may be given by means of rectal enemata containing 6 per cent. dextrose; and purgatives should be strictly avoided. The patient's thirst may be quenched by small pieces of ice sucked from time to time, but no food should be allowed by the mouth. Opium or morphia should not be used, as their administration may mark an increase in the symptoms which might render operation necessary.

Relief is afforded locally by the application of hot linseed-meal poultices, or flannels wrung out of hot water and sprinkled with turpentine or liniment of belladonna. Ice compresses or pieces of ice between layers of flannel are sometimes used, but they do not generally give so much relief as the hot applications. Stimulants are often required, and are best given in the form of brandy, in small quantities frequently.

**Subphrenic Abscess** may occur in the peritoneum or in the cellular tissue beneath the diaphragm. The most frequent positions are classified as follows: (1) the right anterior intra-peritoneal abscess is above the right lobe of the liver and to the right of the falciform ligament; the pus often spreads beneath the liver posteriorly. The commonest causes are appendicitis and hepatic abscesses. (2) The left anterior intra-peritoneal abscess, due principally to perforated gastric ulcer, is situated above the left lobe of the liver and around the spleen. (3) The right extra-peritoneal abscess is situated in the cellular tissue above and behind the liver, and originates from inflammation in the liver, right retroperitoneal tissues and the thorax. There is little subphrenic cellular tissue on the left side, so that inflammation here tends to produce a lumbar abscess. Suppuration in the lesser peritoneal sac, which arises from perforated gastric ulcer, is not so common as in the other situations mentioned (**55**). Pain is the earliest symptom of subphrenic abscess. Besides the general symptoms of a febrile disturbance due to suppuration, there are important localising signs, viz. an abdominal swelling which does not descend on inspiration, bulging of the thoracic walls on the side of the abscess, with deep tenderness. At the base of the corresponding lung there are dulness and diminution of breath sounds, vocal resonance

and tactile vocal fremitus. X-ray examination shows that the corresponding diaphragm is raised and immobile. When the abscess contains air, as may happen if it results from perforation of a viscus, a pneumothorax is simulated by the occurrence of tympanitic note, amphoric breathing, metallic tinkling, and bell sound. Exploratory puncture is best carried out under an anæsthetic, and if positive the cavity should be drained by open operation.

## CHRONIC PERITONITIS

This may arise as a sequel of acute peritonitis, especially in its local forms; it is often the result of local irritation about particular organs—for instance, the liver or the spleen may be surrounded with a thickened capsule (perihepatitis, perisplenitis); or it may occur after repeated tappings for ascites. The growth of tubercle and carcinoma in the abdominal cavity gives rise to forms of chronic peritonitis, which will be spoken of shortly. Often it is impossible to explain its occurrence, but it is assumed that it is due to some form of infection (*see* Perihepatitis; Polyorrhomenitis). Bright's disease appears sometimes to supply the toxic agent.

**Morbid Anatomy.**—Chronic peritonitis, in its lesser degrees, causes no more than thickening, with opacity of the peritoneum; in more advanced cases it results in the formation of false membranes, or layers of fibrous tissue, which cover the different viscera, and cause them to become adherent to one another and to the abdominal wall (*chronic proliferative peritonitis*). The intestines may thus be matted firmly together, and bound down towards the spine; the omentum is shortened and contracted into a transverse band at the upper part of the abdomen; and the liver and spleen may be covered with a thick adventitious membrane. (*See* Perihepatitis.) This membrane can sometimes be peeled off, leaving the natural serous layer beneath. Serous fluid may be present at the same time in sufficient quantity to cause enlargement of the abdomen, or there may be chronic effusion, with but little false membrane and only some opacity or pigmentation of the peritoneum. As already mentioned, chronic peritonitis may result in intestinal obstruction.

**Symptoms.**—These vary with the extent of the abdominal lesion. There is usually some colicky pain, which may be very severe, or a sensation of tightness and oppression. The appetite is likely to be indifferent, and occasionally vomiting may occur. The bowels are generally constipated. Constitutional disturbance may be but slight, but fever, if present, is variable, and often the patient is about, though unequal to much exertion. The condition of the abdomen depends on the nature of the effused products; if serum exists in any quantity, the abdomen is enlarged, and gives the sense of fluctuation on percussion. Sometimes there is dulness in the flanks, and resonance in front, with a change of relative position of these signs when the patient lies on one side; in other cases the abdomen is completely dull from the intestines being bound down to the spine. When there is little or no fluid the abdomen may be flat, and it presents irregular resistance where the intestines are matted together.

**Diagnosis.**—Cases with much effusion resemble ascites, such as that produced by hepatic cirrhosis; in women, if the surface is completely dull from the intestines being bound down, the peritoneal fluid may be mistaken for that of an ovarian cyst; in other cases, the resistance felt in the abdomen may be confounded with different forms of tumour.

**Prognosis.**—This is, on the whole, unfavourable in cases with pronounced symptoms.

**Treatment.**—There is not the same necessity for absolute quiet on the part of the intestines as in the case of acute peritonitis; food should be light and nutritious; pain may be relieved by poultices and hot fomentations, and by

opium in small doses. The absorption of the effused products may be attempted by the application to the abdomen of mercury in the form of compound ointment, liniment, or oleate; while iodide of potassium and iodide of iron may be given internally, with general tonics, such as quinine, cod-liver oil, or arsenic. Constipation may be relieved by enema, or by the mildest laxatives. Paracentesis is often necessary and may have to be repeated.

## TUBERCULOUS PERITONITIS

**Ætiology.**—Tuberculous peritonitis occurs at all ages, but most commonly in children and young adults. It is very commonly associated with tubercle in other parts of the body. It is hence often secondary to pulmonary phthisis, to tuberculous ulceration of the intestine, to caseous mesenteric glands, and to diseases of the pelvic organs—*e.g.* the Fallopian tubes, or the testes and vesiculæ seminales. It may form part of an acute general tuberculosis. Sometimes, perhaps, the peritoneum is infected directly from the bowel. Not infrequently the pleura and the pericardium are simultaneously affected (*see* Polyorrhomenitis).

**Pathology.**—There are four types of tuberculous peritonitis: (1) The *ascitic* type. The surface of the peritoneum is covered with small, flat whitish grains, from 2 to 5 mm. in diameter, slightly raised above the surface, and closely aggregated together. These tubercles are most abundant on the under-surface of the diaphragm and in the flanks. There is an exudation of serous fluid, which may amount to several pints, and the abdomen may be as much enlarged as it is in ascites from cirrhosis or heart disease. More rarely the liquid is sero-purulent or purulent. (2) The *adhesive, fibrinous* or *plastic* type. The exudation of fluid is much smaller in quantity, and fibrin is deposited extensively between the coils of intestine. Organisation takes place so that the coils adhere together and the peritoneal cavity becomes obliterated. In later stages firm fibrous tissue is formed. The tubercles may not be at all obvious. (3) The *caseous* type. The tubercles become larger, and in places they coalesce to form yellow caseous lumps. This often occurs in the great omentum, which becomes contracted up, forming a sausage-shaped mass across the abdomen. Extensive ulceration of the bowel occurs in this form. The adherent intestines may open into one another through the bases of the ulcers, and render it impossible to trace the natural course of the alimentary canal. Perforation may take place, causing acute general peritonitis, or there may be collections of pus shut off from one another in different parts of the peritoneal cavity. The mesenteric glands are often caseous, and when these form large palpable lumps, the type is often called (4) *tabes mesenterica*, which term is used to denote a primary tuberculosis of the mesenteric glands, involving the peritoneum secondarily. The glands may suppurate and burst externally or into the peritoneal cavity. It must be remembered that cases often do not correspond to these types in any hard-and-fast way. Mixtures of all of them may be present, and the case may change from one type to another during the course of the illness.

**Symptoms.**—The symptoms are sometimes *acute*, and the case is similar in every respect to acute peritonitis from other infections. More often they resemble those described under Chronic Peritonitis. They have come on slowly and insidiously, consisting of pain and discomfort, and frequently distension of the abdomen. The patient loses strength and flesh; there is irregular pyrexia; the appetite is diminished, and the bowels are irregular, but often loose. The motions are sometimes yellow, and in acuter cases may suggest enteric fever. In the ascitic form with much fluid the abdomen will be enlarged, tense, resonant in its upper part, or dull all over, according as the intestines are free to float or bound down to the spine. In the adhesive form the abdomen is tender, slightly swollen, very tense, presenting to palpation increased resistance in certain parts,

or a doughy sensation. In the caseous form and in tabes mesenterica there are firm rounded masses with a more or less definite outline. Such tumour-like masses often occupy the lower half of the abdomen, reaching perhaps higher on one side than on the other; they are irregular or nodular on the surface. Sometimes the indurated masses of tuberculous infiltration are felt as bands running across the abdomen. Thus the thickened omentum often forms a transverse band at its upper part, and the tissue about the obliterated urachus forms a vertical band below the umbilicus. In many cases the skin around the umbilicus, for a distance of from 1 to 3 or 4 inches, is reddened and infiltrated, pitting slightly on pressure; this is probably due to obstruction of the veins by the tuberculous growth. The percussion resonance is variable, and often muffled. Occasionally the abdomen is retracted from the excess of fibrous tissue and consequent contraction. Tuberculosis of the abdominal lymphatic glands with obstruction of the lymphatics causes steatorrhœa (*see* p. 515).

**Diagnosis.**—A distended abdomen associated with a chest showing undue prominence of the ribs and recession of the intercostal spaces from wasting is very characteristic of advanced tuberculous peritonitis. Obvious veins may be seen on the surface of the abdomen, and there is tenderness. The recognition of tuberculous masses and inflammatory redness about the umbilicus are important aids to diagnosis.

There are three conditions in children, characterised by wasted limbs and a large abdomen, which are liable to be spoken of as "consumption of the bowels." They are tuberculous peritonitis and tuberculosis of the abdominal lymphatic glands, cœliac disease, and simple indigestion with diarrhœa. Of these the last is the most common. The X-rays may help in doubtful cases. In tuberculous peritonitis, owing to the adhesions, the opaque meal forms small aggregations at irregular points in the small intestine, whereas normally it passes uninterruptedly through to the end of the ileum. The infiltrated omentum may be mistaken for the lower part of an enlarged liver, but the resonance of the stomach above it should prevent this error. Sometimes, but not always, the diagnosis may be confirmed by the presence of tubercle in other parts of the body. In children or young people, a simple ascites, otherwise unexplained, is likely to be tuberculous, but it is often difficult to distinguish from the ascites of hepatic cirrhosis, which indeed sometimes co-exists; and it has often been mistaken for an ovarian cyst until operation has proved the contrary. The liquid withdrawn by paracentesis may be tested by inoculation into animals, or tuberculin may be used (*see* p. 258).

**Prognosis.**—This is more hopeful than the prognosis of many other tuberculous lesions, and many patients treated early have apparently recovered completely. Not only does liquid become absorbed, but large masses of induration, infiltration, or matting have disappeared entirely in some cases. Death results from exhaustion, or from acute tuberculous inflammation in the pleura, pericardium, or meninges.

**Treatment.**—Rest in bed is important, and this should be combined with fresh air, as described in the sanatorium treatment of phthisis. Exposure of the body to sunlight (*heliotherapy*) is a valuable method of treating not only tuberculous peritonitis, but tuberculosis of the lymphatic glands, bones and joints. The extent of body surface exposed is gradually increased from day to day, beginning with the feet and passing upwards, until finally the whole body is exposed for two or three hours a day. The head is kept covered. Albinos and people whose power of responding by producing pigment is limited are not very suitable subjects (**52**). Cod-liver oil and general tonics may be used internally, and the diet should be easily digestible. Mercurial ointment applied to the abdomen is an old method of treatment. Operation should not be carried out unless there are complications. Steatorrhœa should be treated by reducing the fat in the diet.

### POLYORRHOMENITIS

The names *polyorrhomenitis* (ὀρρός, serum) and *polyserositis* are given to the condition of simultaneous inflammations of two or more of the four great serous membranes, pericardium, pleuræ, and peritoneum, an association to which attention has been especially called by Italian writers. It is the result of a common infection, whether it commences in the membranes simultaneously or affects them successively, and this last is the more usual event.

It may be acute, subacute, or chronic. Acute polyorrhomenitis is seen occasionally in acute rheumatism, in pyæmia, and septicæmia, and as a result of pneumococcal invasion. Subacute and chronic polyorrhomenitis are often the result of tubercle, and the recognition of this fact is an important element in the diagnosis of some tuberculous lesions. The successive invasion of the membranes may take place in almost any order, but the most frequent event is that the peritoneum is first attacked, and then the pleuræ, beginning generally with the right pleura. Some cases of indurative mediastino-pericarditis (*see* p. 384), though certainly coming under the definition *polyorrhomenitis*, appear to be exceptions to the tuberculous origin of chronic cases. The prognosis of the combined lesions is obviously worse than when one membrane is alone involved. (*See also* Perihepatitis *and* Chronic Peritonitis.)

### PERITONEAL ADHESIONS

Some importance attaches at different times to the adhesions which form between adjacent viscera or parts of viscera covered with peritoneum as a result of acute or chronic inflammation.

Perigastric adhesions have already been mentioned as the result of gastric and duodenal ulcers. They occur also in connection with carcinoma of the stomach, with operations on this organ, and with gall stones or inflammation of the gall bladder. They are no doubt in most cases latent, but they give rise sometimes to troublesome pains, or interfere with free movement and function of the parts concerned. The pains are situated in the epigastric or hypochondriac region; they are variable, in some cases determined by distension of the stomach, in others occurring when it is empty; if on the posterior surface they may be relieved by the recumbent position, if in front by the converse. In other cases lateral positions may determine or relieve them, or stretching the arm above the head may drag upon them and so cause pain. It may be difficult to discriminate between the pain of adhesions and the pain of the associated lesions. In some cases relief may be obtained by operation, either freeing the organs or dealing with the original disease; but operation does not usually bring much relief.

Adhesions in connection with the bowel have been regarded both as the result and as the cause of constipation, and much importance has been attached to them in regard to intestinal stasis and alimentary toxæmia (*see* p. 438). But it has been shown that adhesions can be found in the majority of persons of all but the youngest ages, being most frequent about the colon, and especially the ascending colon and hepatic flexure, and that even in fœtal life an adhesive process sets in causing adhesions at the splenic and hepatic flexures, and in some cases fixing the ileum in the pelvic fossa. Their action in impeding the flow of the bowel contents is open to investigation by X-rays after a bismuth or barium meal. Adhesions resulting from pelvic peritonitis, and fixing the pelvic colon, certainly sometimes contribute to produce this constipation. In suitable cases the adhesions may be divided.

### PERITONEAL EFFUSIONS AND CONTENTS

Liquid effusions into the peritoneal cavity are: (1) The serous, serofibrinous and purulent liquids which result from inflammation or peritonitis. (2) Ascites,

or the liquids effused in the different forms of hepatic, cardiac or renal dropsy, and the chylous and chyliform liquids sometimes present. Ascites has been described above (*see* p. 477). (3) Liquids provided by the rupture of vessels, or other adjacent structures. Thus (*a*) blood may be found in the peritoneal cavity as the result of traumatism, rupture of an aneurysm, hæmorrhagic pancreatitis, embolism or thrombosis of mesenteric vessels, rupture of vessels in carcinomatous growths, extra-uterine pregnancy and other conditions. (*b*) Bile may reach the peritoneum during rupture of a hydatid cyst in the liver, and possibly from rupture of the gall bladder. (*c*) A hydatid cyst, whether simple or suppurating, may rupture and discharge its contents into the peritoneum. (*d*) Pus from abscesses and (*e*) the contents of any of the hollow abdominal viscera may from traumatism or disease be passed into the cavity, for instance, those of the stomach, intestines or bladder, etc.

## NEW GROWTHS IN THE PERITONEUM

One of the most common growths in the peritoneum is *carcinoma*, secondary to disease in the viscera, especially the stomach and the ovary. It is more common in females than males, and occurs mostly at an advanced age. It occurs in the form of flat circular deposits, covering the abdominal surface, and, like tubercle, it is most abundant on the diaphragm and in the flanks; similarly the omentum may be thickened and infiltrated, and eventually large carcinomatous nodules may occur all over the abdomen. *Colloid carcinoma* is present in a certain number of cases. Considerable liquid effusion is commonly present (*carcinomatous peritonitis*), and blood is not infrequently mixed with it, so that it acquires a brown, brownish-red, or even red colour. Occasionally nodules of carcinoma are felt in the skin around the umbilicus, and the glands in the groin may be infiltrated with the same growth. Colloid carcinoma must be distinguished from so-called *pseudo-myxoma peritonei*. If an organ, *e.g.* the appendix, affected with catarrhal inflammation, ruptures, mucus may be extruded through the opening into the peritoneum, forming large masses, to which this name is given.

*Sarcoma* is another common form of malignant disease; it occurs in the retro-peritoneal tissues, in the omentum, mesentery, or broad ligament. Retro-peritoneal *myxo-sarcoma* may form very large tumours.

**Symptoms.**—The more rapid and malignant forms of growth are characterised by pain, emaciation, cachexia, and the presence of the tumour; if inflammatory conditions predominate, there will be a resemblance to other forms of chronic peritonitis. In other cases the intestine may be involved (*see* Intestinal Obstruction).

**Prognosis** is absolutely unfavourable, and **Treatment** must be directed to the relief of symptoms, the temporary removal of fluid when it is considerable, or an operation for obstructed bowel. Sarcoma may do well with deep X-ray therapy.

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# DISEASES OF THE BLOOD, SPLEEN, AND LYMPHATIC SYSTEM

## DISEASES OF THE BLOOD

### EXAMINATION OF THE BLOOD

In relation to disease, the blood may have to be examined in many ways. The inquiries of most importance are those into the total numbers of the corpuscles, red and white, into details as to the kind of corpuscles and their relative proportions, and into the amount of hæmoglobin.

**Coagulation Time.**—Of the various methods proposed that of Dale and Laidlaw gives consistent results, and has the advantage that the blood is kept throughout at approximately the temperature of the body. A capillary glass tube is used of approximately 2 cm. length and 1·3 to 1·4 mm. internal diameter. A spherical lead shot of nearly 9 milligrammes weight is inserted in the tube and kept in place by slightly sealing the ends of the tube. For the determination the finger is pricked with a clean, sharp needle and the resulting blood allowed to flow rapidly into the tube. The ends of the tube are then closed by means of a metal clip carrying plasticine in two minute cups, and the whole placed in water and rotated till the shot sticks in the tube. The time is taken from the appearance of the blood till the shot ceases to move. Normal time is about one minute forty seconds.

**Fragility of Corpuscles.**—In some diseases it has been found that the fragility of the red corpuscles is greater than normal, or, in other words, that the corpuscular or globular resistance to hæmolysis by dilute fluids is less. For this determination a few cubic centimetres of blood are mixed with an isotonic solution of oxalate of potassium (pot. oxal. 0·28 gramme, sod. chlor. 0·8 gramme, aq. dest. 100 grammes), centrifuged, the plasma decanted, and the corpuscles washed in a solution of 0·9 per cent. of sodium chloride, and then tested with sodium chloride solutions of different strengths. Washed normal red corpuscles show slight hæmolysis in 0·45 per cent. sodium chloride, and complete hæmolysis in 0·35 per cent. Abnormally high fragility is shown if hæmolysis occurs with stronger solutions, as is the case in acholuric jaundice.

**Enumeration of Corpuscles, or Blood-count.**—This is effected by the *hæmocytometer* of Thoma-Zeiss or Bürker-Zeiss.

The former consists of a glass slide on which a "cell" is constructed $\frac{1}{10}$ mm. in depth and ruled at the bottom into squares measuring $\frac{1}{20}$ mm. in the side, which are again ruled into groups of sixteen small squares. In a specially constructed pipette the blood is diluted to the extent of 1 to 100 by a saline solution (sodium phosphate or chloride) which does not injure the corpuscles; and a drop of this is placed in the cell and covered with thin glass. The corpuscles settle down upon the squares, each of which corresponds to $\frac{1}{4000}$ cubic millimetre. The red corpuscles in several groups of sixteen squares are counted, and the total multiplied by 100 (the dilution) and 4,000 (representing the volume of fluid over each small square), and divided by the number of small squares counted, gives the corpuscles in a cubic millimetre. In Bürker's instrument, which is

preferable to that of Thoma-Zeiss, the cover glass is first of all arranged in position by clamps, and the diluted blood is then run in by capillary attraction. Bürker's method of effecting dilution is also much superior, but this is not yet much used in this country.

The average number of red corpuscles per cubic millimetre is taken to be 5,000,000 for males and 4,500,000 for females. Any greater or less number can be stated as such or represented as a percentage of the normal. Thus for males 3,100,000 corpuscles = 62 per cent.

The number of leucocytes is from 7,000 to 10,000 per cubic millimetre. They require to be separately counted, and are usually stated in absolute numbers A second pipette provides for a dilution of 1 in 10 with 0·9 per cent. acetic acid, by which the red corpuscles are rendered invisible.

**Platelet-counting.**—A drop of sterile 2·5 per cent. solution of sodium citrate is placed on the finger, and the latter is pricked through the drop, so that blood diffuses out into it. The mixed blood and citrate are examined fresh with the microscope, drying being prevented by ringing the cover-slip with vaseline. If the number of red cells per cubic millimetre is known, the number of platelets can be determined from the ratio of the two in the film. The normal figure varies between 200,000 and 500,000.

**Estimation of Hæmoglobin.**—For clinical purposes Haldane's modification of Gowers' hæmoglobinometer is generally used. Two tubes are provided. One of them, which is the standard, contains a 1 per cent. solution of normal blood saturated with carbonic oxide, and is hermetically sealed. The other tube is graduated in divisions up to 100, and in this a measured quantity of blood is diluted till it matches the colour of the standard after saturating with coal-gas to convert all the hæmoglobin into CO-hæmoglobin. The figure on the scale which the solution then reaches represents the percentage amount of hæmoglobin.

*Colour Index.*—The concentration of hæmoglobin in the corpuscles remains much the same in various diseases of the blood; but the size of the corpuscles varies considerably. The percentage of hæmoglobin is less than that of the corpuscles if individual corpuscles are smaller than normal; the hæmoglobin percentage is greater if the corpuscles are larger than normal. This relation of the hæmoglobin percentage to the corpuscle percentage is called the *colour index*, and it may be less or greater than 1, according as the cells are on the average smaller or larger than normal (**1**). Thus with hæmoglobin 40 per cent., corpuscles 50 per cent., the colour index is $\frac{4}{5}$ or 0·8; with hæmoglobin 30 per cent., corpuscles 20 per cent., the colour index is $\frac{3}{2}$ or 1·5.

**Microscopic Examination of the Corpuscles.**—This can only be done satisfactorily after the corpuscles have been stained. The blood is spread on a glass slide, or cover-glass, in a thin layer, or film, and stained with a mixture of eosin and methylene blue. Jenner's or Leishmann's stains are usually employed.

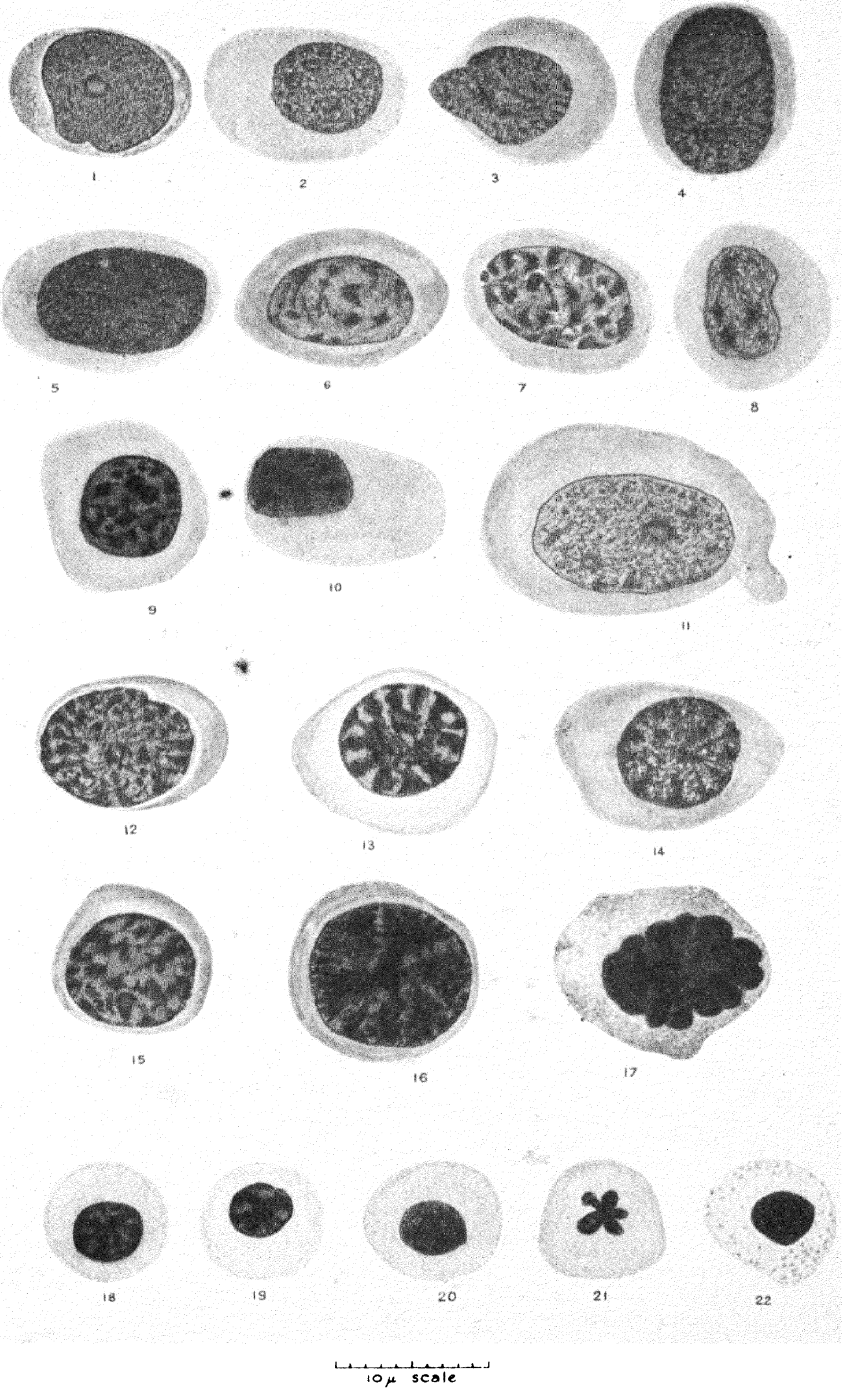
The different corpuscles which may be seen in health and disease may be now enumerated (*see* Plates XXIX., XXX. and XXXI.).

*Red Corpuscles or Erythrocytes.*—The normal red corpuscle measuring in diameter 7·5$\mu$; small corpuscles, or *microcytes*, from 2 to 6$\mu$; large corpuscles, or *megalocytes*, from 8 to 15$\mu$. Inequality in the size of the red cells is called *anisocytosis*. In addition there are misshapen, distorted, often pear-shaped corpuscles, or *poikilocytes;* fragments of corpuscles are called *schizocytes;* nucleated red corpuscles (*erythroblasts*), divided according to their size and shape into *normoblasts*, *microblasts*, *megaloblasts*, and *poikiloblasts*, also occur.

Nucleated red cells occur normally in the bone marrow. Their presence in the blood indicates increased production in the marrow.

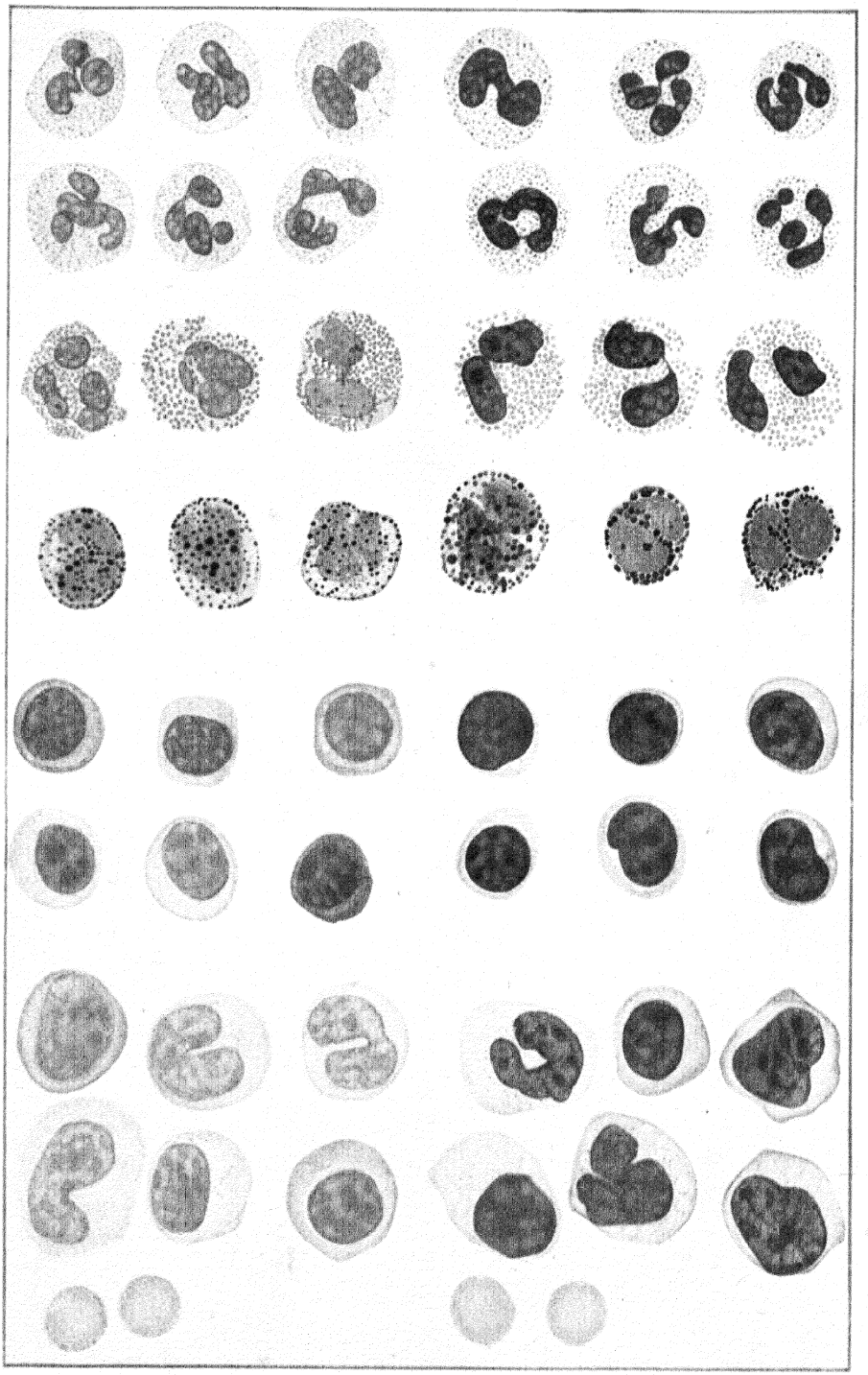
Free nuclei from rupture of erythroblasts may be seen. *Polychromasia* is a condition where the cytoplasm stains bluish instead of pink. It is apt to occur when the cells are nucleated. *Punctate basophilia* is an allied condition; blue

# PLATE XXIX.




Coloured illustration of different types of large nucleated red cells. Five normoblasts are seen in the bottom row. Note the polychromasia in many of the cells and the punctate basophilia in No. 22. (Reproduced by kind permission of Dr. A. Piney, *Journ. Path. and Bact.*, vol. xxvii., July, 1924.)

[*To face p.* 520



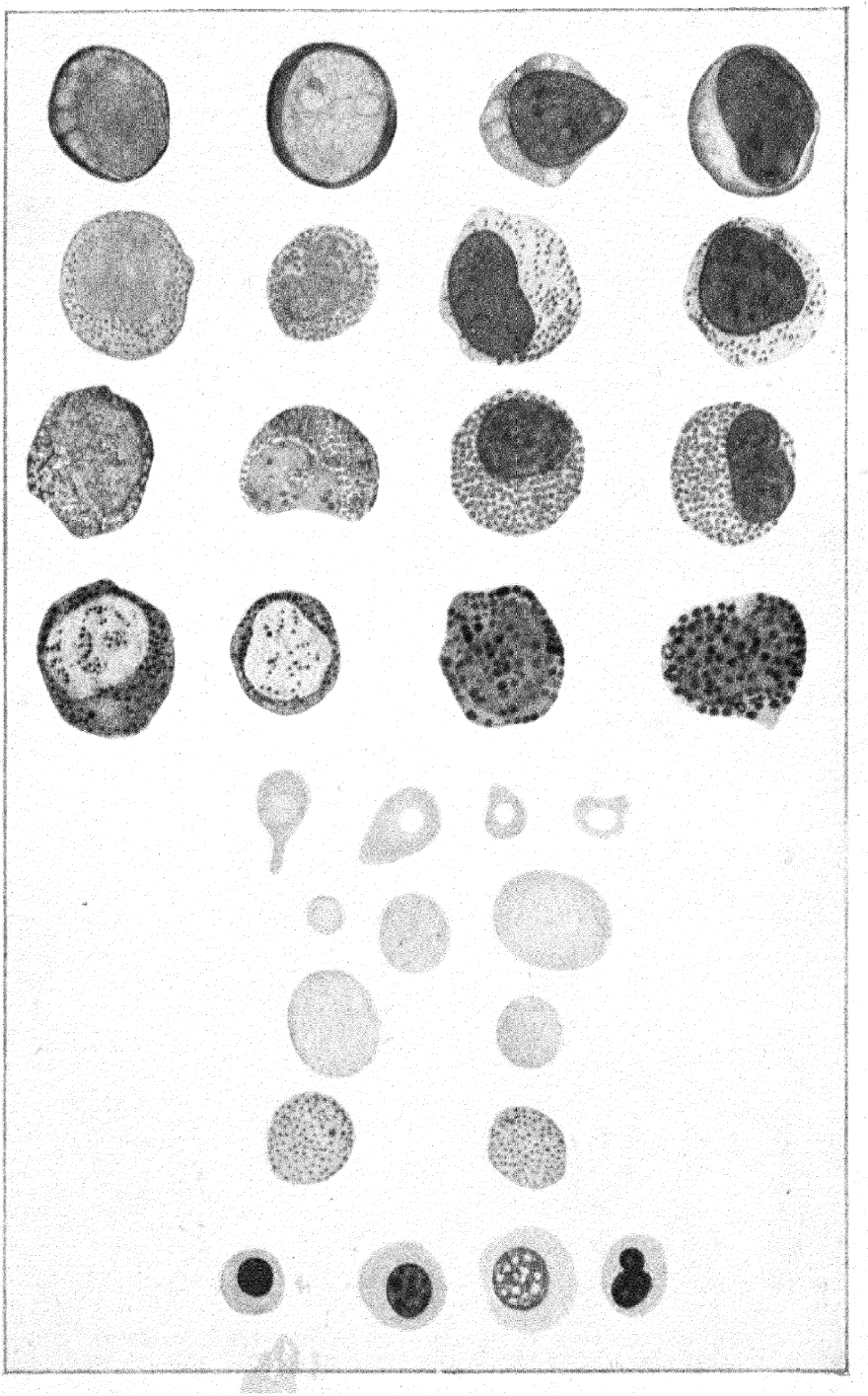
Normal Blood Cells.

[*To face p.* 521.

NORMAL BLOOD

| JENNER'S STAIN | LEISHMAN'S STAIN |
| --- | --- |
| POLYMORPHONUCLEAR LEUCOCYTES | |
| EOSINOPHILS | |
| MAST CELLS | |
| LYMPHOCYTES | |
| LARGE MONONUCLEARS | |
| RED CELLS | |

# PLATE XXXI.



Abnormal Blood Cells.

[*To face p.* 521.

# ABNORMAL BLOOD

| JENNER'S STAIN | LEISHMAN'S STAIN |
| --- | --- |
| MYELOBLASTS | |
| GRANULAR | MYELOCYTES |
| EOSINOPHIL | MYELOCYTES |
| BASOPHIL | MYELOCYTES |

JENNER'S STAIN

| |
| --- |
| POIKILOCYTOSIS |
| ANISOCYTOSIS |
| POLYCHROMASIA |
| PUNCTATE BASOPHILIA |
| NUCLEATED RED CELLS |

spots are seen in the cytoplasm. A *metrocyte* is a megaloblast in which the nucleus shows signs of mitosis.

*Leucocytes.*—The white cells, of which there are several varieties, fall into two groups: (1) granular cells, sometimes called *leucoid*, viz. *polymorphonuclear* cells with small acid-staining granules in the cytoplasm and variable nucleus, *eosinophil* cells with coarse granules and horseshoe-shaped nucleus, and *mast* cells with granules staining purple and faintly basophil nucleus; (2) non-granular, or *lymphoid*, cells, viz. *lymphocytes*, large and small, with large, round, strongly basophil nucleus and scanty cytoplasm, which is only faintly stained, and large *mononuclear* cells, with faintly staining nucleus and cytoplasm. Sometimes these cells contain a horseshoe-shaped nucleus, and since they have been supposed to be intermediate between the polymorphonuclear cell and the lymphocyte, they have been called "transitional cells," but this is not the case, and so the term should be avoided.

Large mononuclear cells are also known as *macrophages*, and polymorphonuclear cells as *microphages*. Eosinophil cells are also phagocytic. All the cells, except perhaps lymphocytes, show amœboid movement.

*Differential Count.*

| | Measuring about | Per cent. |
|---|---|---|
| Polymorphonuclear cells | 10—12$\mu$ | 55—65 |
| Coarsely granular eosinophil cells | 11—13$\mu$ | 3— 6 |
| Mast cells | 10—12$\mu$ | 0·3— 1 |
| Lymphocytes | 7—12$\mu$ | 20—30 |
| Large mononuclear cells | 12—20$\mu$ | 6— 8 |

Abnormal leucocytes are found in the blood in certain diseases. In the first place, there are degenerated cells, which show vacuoles in the cytoplasm and have badly staining nuclei; in the second place, there are various immature cells which come from the bone marrow and are known generically as *myelocytes*. The *myeloblast* is a large primitive cell (10-20$\mu$), arising from the giant cell of the marrow, with large, faintly staining nucleus, surrounded with a band of deeply staining cytoplasm. The *promyelocyte*, which is the next stage in development, resembles the preceding, but there are acid-staining areas in the cytoplasm. Then come (1) the *granular myelocyte*, in which the cytoplasm resembles that of the polymorphonuclear cell, and (2) the *eosinophil myelocyte*, with coarse acid-staining granules. All these cells are about the same size. The granular myelocyte passes through the *proleucocyte* stage (with round, darkly staining nucleus) into the polymorphonuclear cell, and the eosinophil myelocyte becomes the coarsely granular eosinophil cell. In addition, there are *basophil myelocytes*, which give rise to mast cells. The myeloblast also gives rise to the lymphocyte, the intermediate stages being the *lymphomyelocyte*, which is smaller, but retains the same kind of nucleus as the myeloblast, and the *prolymphocyte*, in which the nucleus stains deeply basophil. This cell by division forms the lymphocyte.

The granular cells of the blood (*i.e.* the polymorphonuclear, eosinophil and mast cells) with their precursors are grouped together under the term *leucoid cells*. The non-granular cells (*i.e.* lymphocyte and large mononuclear) and their precursors are described as *lymphoid* cells, in contradistinction.

In chronic suppuration the leucocytes sometimes contain fat granules; and in the same condition they may give the *iodine reaction*, indicative of glycogen. To show this, dried blood films are placed for a few minutes in a stoppered bottle containing crystals of iodine, and are then mounted in a saturated solution of lævulose; the glycogen gives a deep mahogany-brown colour.

**Price-Jones Blood Cell Distribution Curve.**—It has already been pointed out that the *colour index* gives a measure of the average size of the corpuscles; but it gives no information as to the variation in size of individual corpuscles.

The latter may be of use in diagnosis and is determined by measuring, under a magnification of 3,000, 500 or more red cells in a stained film in two diameters, and taking the average (**2**). The results are expressed in the form of a Price-Jones blood curve (*see* Fig. 53), which shows by its peak what is the most frequent size of cell met with and by the distance apart of its ascending and descending limbs how closely the remaining cells approximate to this value. The mean diameter of normal cells is 7·2$\mu$ and all the cells are much of a size. In anæmia from hæmorrhage the mean diameter is smaller and the variation is greater. In Addison's anæmia the mean diameter is greater; the variation is very large.

**Nomenclature.**—The advances in hæmatology have called for an increasing number of complex names. Originally *anœmia* or *spanœmia* signified pallor in

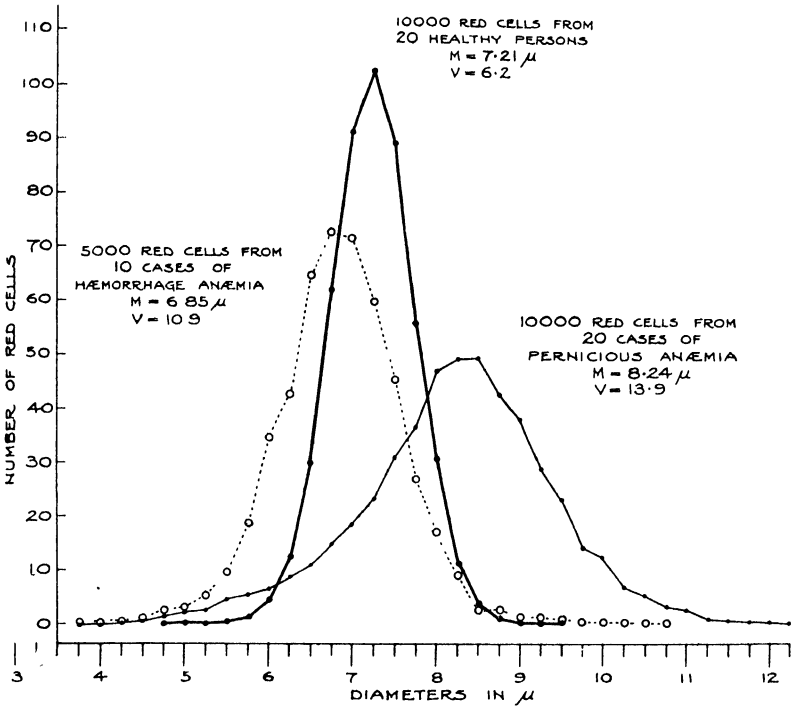



FIG. 53.—Price-Jones Cell Distribution Curves (for description see text).

the patient, and a thin pale blood as it issued from a wound; and the excess of white corpuscles recognised by Virchow in 1845, and called by him *leukœmia*, has been since called by that name, or by the name *leucocythœmia*, proposed by Hughes Bennett in 1851. When enumeration of corpuscles became possible, less degrees of white-cell increase, both physiological and reactive in disease, were recognised as *leucocytosis*, while their deficiency was called *leucopenia* (πενία, poverty). It is now recognised that the red corpuscles, or erythrocytes, may be in enormous excess. This condition is called *polycythœmia rubra*, and is of two types, *erythrœmia* and *erythrocytosis*, of which the former denotes a primary excess of red cells, the latter an increase in response to special demands. We thus have:

Deficiency of hæmoglobin with or without deficiency of red cells—anæmia.
Deficiency of white corpuscles—leucopenia.
Excess of hæmoglobin and red corpuscles—polycythæmia rubra, erythræmia, erythrocytosis.

Excess of white corpuscles—leukæmia, leucocythæmia, leucocytosis.
Excess of different forms of leucocytes—lymphatic leukæmia, myelocytic leukæmia, eosinophilia.

It will be observed that all pronounced forms of leukæmia are accompanied by anæmia, and, further, that both absolute and relative increase and decrease of particular kinds of corpuscles may have to be estimated.

## ANÆMIA

The normal pink colour of the skin and the deeper red colour of the mucous membranes are due to the blood circulating in their vessels · if a considerable loss of blood occurs—as, for instance, after injury, or from gastric ulcer, or during parturition—the natural colour is materially altered, and the skin becomes of a waxy whiteness, blanched or bloodless, and even the lips and mucous membranes have only a very pale pink colour. This condition is spoken of as *anæmia*, and occurs in a great variety of circumstances, besides the direct loss of blood in quantity; moreover, it is accompanied in all its forms by numerous other disturbances, which directly result from the deficiencies which exist in the blood itself. It must be remembered that other conditions besides anæmia give rise to pallor, and, in particular, alteration in the distribution of the blood from vaso-motor action, etc.

**Causation.**—The distinction commonly made between primary anæmia and secondary or symptomatic anæmia is obviously unsatisfactory. In so-called secondary anæmia there is an apparent cause. The bloodlessness results either from direct losses of blood from the system, or from more or less definite diseases of particular organs, whereby the income or expenditure of the body is materially affected. In so-called primary anæmia the change seems to depend on the blood itself; but the high probability is that such cases are due either to toxins operating upon the blood-forming organs, including the bone marrow, or to *hæmolytic* agencies destroying the blood corpuscles.

The two diseases which have been regarded as typical instances of a *primary* anæmia are *chlorosis* or *green sickness*, which during the last century occurred so frequently in females about the time of puberty, and *idiopathic* or *pernicious anæmia*, or *Addison's anæmia*, which occurs in adults of both sexes.

The anæmia called *secondary* or *symptomatic* includes that due to (1) hæmorrhages of all kinds; this may be from incised or lacerated wounds, from epistaxis, gastric, intestinal (typhoid), or rectal ulceration, piles, excessive menstruation, uterine fibroid, ulcerating cancerous tumours (notably carcinoma of the stomach), and other lesions. Many of these hæmorrhages are profuse, but do not recur, or only at long intervals; the anæmia is in direct proportion to the loss, and recovers perhaps quickly within a short time. Repeated small hæmorrhages may take place from piles, rectal ulcer, and uterine diseases, in which case the anæmia is persistent. Hæmorrhages into the skin and mucous membranes may also occur, as in the various forms of purpura and in scurvy. (2) Anæmia is also caused by destruction of blood corpuscles in the circulating blood, as by the malarial parasite and in hæmoglobinuria. (3) Anæmia occurs in many other conditions in which defective formation of hæmoglobin may be the cause, though little is known about it. These conditions are chronic suppuration, prolonged fevers, particularly rheumatic fever, tuberculosis, Bright's disease, syphilis and lead poisoning. The anæmia of aortic regurgitation is due to a coincident infective endocarditis. (4) The intestinal worms, *Bothriocephalus latus* and *Ankylostoma duodenale*, cause anæmias of different types, probably due to some poison produced by the worms, though in the latter case loss of blood may be an additional factor. (5) Diminution of the red corpuscles occurs in Hodgkin's disease and in the different varieties of leukæmia. These are primary diseases of the spleen, lymph glands, and bone marrow, or other blood-forming organs.

The above enumeration shows that some forms of anæmia are caused by a great loss of corpuscles (hæmorrhage) or by excessive destruction of them (malaria), and others by their defective formation (mal-nutrition, chronic diseases, diseases of blood-forming organs). Anæmia may result in any case in which the balance of these two processes fails to be maintained, either from excess of hæmolysis or defect of hæmogenesis.

There is much yet to be learned as to the exact cause in particular cases either of undue hæmolysis or of defective hæmogenesis, and of the influence of toxins or other poisons upon the blood-forming organs, especially the bone marrow.

**Symptoms of Anæmia.**—Some features are common to all cases of pronounced anæmia, however caused. The skin is pale and waxy-looking. In cases of recent hæmorrhage the colour is almost white, but in pernicious anæmia there is a yellow tinge, due to bile-pigment in the serum (*see* p. 527); in chlorosis a greenish tint is sometimes detectable, and a dirty yellow, earthy or sallow tint in the anæmia of lead-poisoning, malaria, and syphilis. The lips are pale pink, and the cheeks may show a faint pink flush. The visible mucous membranes are pale pink, as seen in the mouth, the tongue, and the inner side of the eyelid. The altered colour of the blood is also manifest in the tint of the veins on the back of the hand, which show pink through the white skin, instead of dark purple through the pink skin. The patient is languid and weak, unfit for physical or mental exertion and liable to headache, vertigo, the appearance of spots before the eyes, ringing in the ears, and attacks of syncope. In sudden and large losses of blood, such as occur in parturition, the anæmic condition of the brain may produce convulsions, but these do not occur in chronic cases, unless it may be near the fatal termination. There is breathlessness on exertion, palpitation of the heart, and throbbing of the vessels; in some cases œdema of the feet may be present. The appetite is generally diminished, and the ingestion of food is often followed by weight or oppression at the epigastrium, or by severe heartburn.

Any marked degree of anæmia probably always causes an increase in the output of blood from the heart per minute, which is to be regarded as a compensatory mechanism, but of course means that the work of the heart is increased. Hence rest is the first desideratum in treatment. Auscultation discloses murmurs over one or more of the cardiac orifices. The most common is a systolic murmur, often harsh in quality, heard loudest in the second left intercostal space, and traceable outwards along that space and towards the left clavicle—that is to say, in the area of the pulmonary artery. This murmur is often loudest in the recumbent position, and diminishes or even disappears when the patient stands up. In some cases a murmur is heard at the apex, and in more severe anæmia systolic murmurs may be heard at all the orifices, or even over the whole cardiac area, as well as behind. A similar murmur is often heard in the carotid vessels in the neck. A murmur is also commonly heard at the root of the neck over the jugular vein, the *bruit de diable*, which has already been described (*see* p. 303). Neither this nor the systolic pulmonary hæmic murmur is peculiar to anæmia, nor should either be regarded as evidence of it; but they are almost constantly present in this condition, and are generally more marked in proportion to its intensity.

The dilatation of the heart already mentioned carries the impulse into the line of the nipple, or even external to it. The pulse is commonly rather quicker than normal, and generally soft; the heart's action is readily quickened by exertion or excitement.

In severe degrees of anæmia hæmorrhages are liable to occur, and the temperature may be raised. Retinal hæmorrhages and pyrexia are, indeed, common in pernicious anæmia, but they are also seen in chlorosis and other forms occasionally. Another occasional result of chlorotic anæmia is venous thrombosis.

## CHLOROSIS

This name—or its English equivalent, green sickness—is applied to a form of anæmia which especially occurs in girls and young women between the ages of fourteen and twenty-four, though a similar condition is exceptionally seen in boys. There has been a gradual decline in the incidence of chlorosis since the beginning of the century, and this may well be due to the better conditions under which women's work is carried on and to the greater amount of fresh air and exercise taken (**3**). It was commonest among those in domestic service. It presents the characteristics already described. Languor, feebleness, breathlessness and palpitations on exertion, vertigo, fainting attacks, headaches, noises in the ears, spots before the eyes, nausea, eructations, pain after food, and constipation, occur just as in cases of secondary anæmia. The patient may present all degrees of pallor, often with some temporary flushing of the cheek or lips; sometimes also, no doubt, there is a tint which justifies the name *chlorosis*, but it can scarcely be recognised in all cases—Zimmermann says it occurs only in those of a dark complexion. Both blood corpuscles and hæmoglobin as tested in the usual way are diminished; the former are rarely below 60 per cent. of the normal, but the hæmoglobin may be only 25 or 30 per cent. Thus each corpuscle, because it is smaller, has on an average less than its normal amount of hæmoglobin—that is, the colour index is less than unity. The lower the percentage of hæmoglobin, the smaller is the colour index. The same rule applies to hæmorrhagic anæmias (**3**). This relation of corpuscles to hæmoglobin is often called the *chlorotic type;* but it occurs also in forms of secondary anæmia. Microcytes and schizocytes may be found. Where the bone marrow is excited, regeneration forms of red cells may be seen, *i.e.* normoblasts, and there may be polychromasia and punctate basophilia. The leucocytes are normal, or there may be a slight excess of lymphocytes. Coagulation of the blood is not delayed. There are murmurs at the base of the heart and over the jugular veins. The pulse is not necessarily feeble; its tension may be, but it is not always, increased. A symptom present in 80 per cent. of cases, which more than all attracts the attention of the patient or her friends, is the suppression or diminution of the menstrual flow. In the younger patients the menses may never have appeared, and may continue absent as long as the chlorosis lasts; in those who have already menstruated the flow becomes scanty, pale or irregular, or ceases altogether. It is possibly in connection with these menstrual disorders that the mental condition of the patient is often markedly affected, as shown in irritability and a tendency to hysterical manifestations. Constipation is frequent, and the urine is pale. Optic neuritis occasionally occurs, and may be followed by atrophy, exceptionally also ocular paralysis, retinal embolism, and retrobulbar neuritis. These are conceivably attributable to the occurrence of intracranial thrombosis in a limited area (Hawthorne). A more extensive thrombosis is well known to cause profound cerebral symptoms, with a fatal result (*see* p. 848).

**Pathology.**—Numerous hypotheses have been put forward to explain the occurrence of chlorosis. However, there is no certain knowledge on the subject. Bunge thought that the organic iron compounds of the food (*hæmatogen*) were broken up by decomposition in retained fæces and assumed forms less capable of being absorbed. Undoubtedly there must be some connection between the sex, puberty and the onset of chlorosis; but amenorrhœa is a consequence, and not a cause. Whether defective hæmogenesis or hydræmic plethora is the chief fault in the blood, there is no adequate explanation of its occurrence as yet forthcoming.

**Diagnosis.**—The recognition of chlorosis is generally easy; but it may be difficult, in some severe cases where the heart is dilated and murmurs of mitral regurgitation are heard, to decide whether the cardiac lesion is primary or a result of the anæmia. Amenorrhœa, the absence of rheumatism, scarlatina, or

chorea in the history, and extensive præcordial murmurs, rather than one localised to the heart's impulse and the back, would be in favour of chlorosis. The presence of any primary lesion (*see* p. 523) must always be carefully excluded.

**Prognosis.**—Chlorosis is rarely fatal; it often lasts months or years, if no treatment is adopted, and frequently recurs after cure.

**Treatment.**—Any of the above conditions which may seem to be in operation should, if possible, be dealt with. But for the restoration of the blood state in chlorosis the use of iron is of the first importance. This may be given in several forms; if there is irritability of the stomach, the less astringent forms are advisable, such as reduced iron, 2 or 3 grains three times a day, the ammonio-citrate, or the tartrate, 5 to 10 grains. The more astringent forms, however, when they can be borne, are more quickly efficient—*e.g.* the perchloride, 10 to 20 minims of the tincture, or the sulphate, from 3 to 5 grains. A well-known remedy is Blaud's pill, containing 2½ grains of sulphate of iron and the same of potassium carbonate. One, two or three pills are given thrice daily. The Pilula Ferri, B.P., is a similar combination. Iron should always be given immediately after meals. It is desirable to combine some laxatives with iron, both to counteract its astringency as well as to overcome the tendency to constipation which is so frequent. This may be done by the use of sulphate of magnesium, in combination with the perchloride or the sulphate of iron; or by the use of aloes, in the form of aloes and iron pills; or an aloes or myrrh pill may be given at night with the iron mixture. In some cases arsenic is of value in connection with iron. Inorganic or organic preparations of arsenic are used. Among the latter sodium cacodylate has a reputation when given subcutaneously. The maximum dose is 3 grains a day. A diet containing large amounts of organic iron should be chosen, and so it would include plenty of meat, eggs and green vegetables, and may be modified somewhat according to the fat or lean condition of the patient. In severe or advanced cases, treatment by drugs may fail entirely until the patient has had a prolonged rest or three or four weeks on the couch, or even in bed. Exercise increases both the tendency to dilatation of the heart and the overstrain upon the blood-forming organs; but fresh air is desirable during the treatment.

## PERNICIOUS ANÆMIA

(*Addison's Anæmia, Glossitic Anæmia*)

Cases of this disease were first described by Addison under the name of *idiopathic anæmia*, because they presented distinctive features, and he was unable to find a cause for them. Later Biermer and other Continental writers described similar cases, under the name of *progressive pernicious anæmia;* and no doubt included cases arising secondarily from definite causes, such as syphilis, cancer, and repeated hæmorrhages.

**Ætiology.**—It affects both sexes equally, and 85 per cent. of cases are over the age of forty. The suggestion has been made that it is due to a toxæmia from a hæmolytic streptococcus.

**Anatomical Changes.**—Besides the universal pallor of the organs, one of the most constant conditions *post mortem* is fatty degeneration of the heart, which shows itself by alternate dark and pale striations of the muscle as seen through the endocardium (tabby-cat striation). It is due to deficient oxygen supply, owing to the diminished amount of hæmoglobin in the blood. It occurs in the left ventricle and the papillary muscles. There is also fatty degeneration of the liver and kidneys, and of the intima of the arteries. Hæmorrhages are found not only in the retina, where they have been seen during life, but in the serous membranes, the endocardium, the mucous membrane of the stomach, the lungs, the surface of the brain, and other parts. Fenwick and others have found fatty degeneration or atrophy of the tubular glands of the stomach, and infiltration of

leucocytes between the tubules, but this is not a constant phenomenon. The spleen is sometimes enlarged, and of dark red or purple colour. The marrow of the bones has been found to be excessive in amount, of a reddish-purple colour, with large numbers of nucleated red corpuscles, especially megaloblasts, while the fat cells are all or nearly all destroyed—a return to the condition of the embryo. There is, moreover, an abundant deposit of iron in the cells of the liver, in the spleen and in the kidneys, as can be shown by the organ turning black with ammonium sulphide or blue with potassium ferrocyanide and dilute hydrochloric acid. In doubtful cases it is essential to examine microscopic sections. Whereas the normal iron content of the liver is 0·1 per cent., in this disease it may rise to 1·0 per cent. The iron is most abundant in the peripheral zones of the lobules. The percentage of iron in the kidneys may rise from 0·01 to 0·09, and in the spleen from 0·18 to 0·3. In cases with spinal symptoms a degeneration of the lateral and posterior columns is found after death.

**Pathology.**—The striking feature is the evidence of blood destruction (hæmolysis) in the excess of urobilin in the urine, the deposit of iron in the liver, and the presence of bile pigment in the blood, which gives Van den Bergh's indirect test (*see* p. 475). Similar features have been obtained by injecting poisons such as saponin, pyridine, etc., into animals. In pernicious anæmia the hæmolysis probably takes place within the portal circulation.

W. Hunter's view is that the hæmolysis is the primary feature of the disease, and that this is due to an infection which shows itself in a special inflammation of the tongue (infective glossitis), of the stomach and alimentary canal, as indicated by vomiting and diarrhœa in some cases. He regards oral sepsis, so frequently present in these cases, as contributing to the severity of the anæmia, but not as the cause of the disease (**4**). Peculiar features of the disease are the irregularity in size and shape of the red cells and the presence of megalocytes and megaloblasts. Such features do not appear in man in severe anæmias from hæmorrhage ; but they are found in rabbits which have been bled artificially (**5**). In man these blood changes are ascribed to an abnormal and excessive response on the part of the marrow to the blood changes.

Another view, however (Gulland and Goodall), is that the primary lesion is in the bone marrow, and that the disease is a *megaloblastic anæmia*, due to exhaustion of the bone marrow, or to the influence of toxins upon it, the destruction of the corpuscles being the result of their undue vulnerability in these circumstances. However, megaloblasts may only be present during the exacerbations of the disease, whereas megalocytes are constantly present, so that the disease would be better described as a *megalocytic anæmia*.

It is interesting that the anæmia due to *Bothriocephalus latus* and sprue resembles pernicious anæmia in the diminished red cells and hæmoglobin, the high colour index, and the presence of megalocytes (**6**). It is possible that a hæmolytic streptococcus is responsible for the infection that causes pernicious anæmia, because it has been obtained from the duodenum. There is every probability that the streptococcus grows there because the hydrochloric acid of the stomach, which normally forms a germicidal barrier, is absent, and so the streptococcus gets through from the mouth. On this view the achlorhydria is primarily responsible for the disease. Three factors probably play a part in causing the achlorhydria : (1) the destruction of the gastric glands, (2) the anæmia, and (3) excessive neutralisation from regurgitation from the duodenum, a feature of certain dyspeptic states (*see* p. 418).

The association of subacute degeneration of the spinal cord with pernicious anæmia may be due to the toxæmia simultaneously affecting both the spinal cord and the blood ; but either of these effects may occur independently, or either of them may follow the other (**7**).

**Symptoms.**—The patient with pernicious anæmia gradually loses strength and becomes paler ; his skin acquires a yellow tint, different from the waxy white of ordinary anæmia, but it is a lemon-yellow tint rather than the greenish yellow

of bile pigment. Sometimes there is brown pigmentation in small or large patches. At the same time, even with extreme anæmia, the patient does not lose flesh, and the subcutaneous fat may be abundant. There are the same languor, indisposition for mental and physical exertion, giddiness, noises in the ears, etc., as have been described in the other forms of anæmia, as well as dyspnœa on exertion, cardiac palpitation and vascular murmurs. The patient complains of soreness of the tongue, which may present vesicles and red patches. In long-standing cases the tongue may show atrophy of the filiform papillæ and even fissures. Loss of appetite and nausea are frequent, and in a large proportion of cases there are vomiting and diarrhœa. Free hydrochloric acid is usually completely absent from the gastric juice. The urine is high-coloured from excess of urobilin, and is free from albumin. The spleen is enlarged. The bones are often tender on percussion or pressure. The pupils are mostly dilated; and the retina may show numerous small hæmorrhages, which are abundant round the optic disc. These are striated, or flame-shaped, and may be accompanied with white spots. Another feature of idiopathic anæmia is the presence of fever, which may give a temperature of 101° or 102° but is generally irregular. It may be absent for days together, and the temperature is often subnormal before death. Sometimes the liver is enlarged and tender. The blood is excessively pale, and the red corpuscles are reduced to 2,500,000 per cubic millimetre, or even to 500,000 or lower; but the diminution of the hæmoglobin is less in proportion. Thus the colour index is usually greater than 1. This contrasts with the condition in chlorosis and secondary anæmia, and is due to the fact that individual corpuscles are larger than normal. However, values as low as 0·7 have been sometimes found. Poikilocytes are more numerous than in any other condition. Usually there are some nucleated red cells, especially megaloblasts, but the leucocytes are commonly less numerous than normal, with a relative lymphocytosis (*see* Plates XXIX., XXX., XXXI.). The Price-Jones distribution curve of the blood is characteristic (*see* p. 522). There may be a diminution of coagulability, but there is little or no increase of fragility in the corpuscles.

The course of the disease is not always progressive: indeed, remissions are frequent, especially under treatment; and the patients regain colour, and the condition of the blood is much improved, though megalocytes are still found. Ultimately, however, in most cases they gradually get weaker, become drowsy and apathetic, and death takes place.

Patients with pernicious anæmia often have numbness and weakness of the legs, ataxy, altered knee jerks, and other symptoms of degeneration of the spinal cord (*see* p. 795); this occurs, however, in other severe forms of anæmia such as leukæmia, arsenical poisoning, and chronic alcoholism, and the spinal symptoms may even appear long before the anæmia. Mental disorders have sometimes been observed.

**Diagnosis.**—In any case supposed to be one of *Addisonian* or *pernicious anæmia* it is important to search most carefully for organic disease, such as cancer, which might be the cause of the bloodlessness, and for septic foci, and in certain circumstances to exclude intestinal worms (Bothriocephalus), or sprue, by proper examination of the fæces. The distinctive features of pernicious anæmia are the bile pigment in the serum which gives it its yellow colour, the blood picture with high colour index, and the typical cell distribution curve, all of which serve to distinguish it from chlorosis and other anæmias.

**Prognosis.**—This is unfavourable, but recovery has been known, while temporary recovery is often seen under the use of arsenic or blood transfusion, with relapse after some months. Many cases get progressively worse and die in about six months, whereas others last from twelve months to two years.

**Treatment.**—Sources of sepsis, whether due to teeth, tonsils, nasal sinuses, etc., must be dealt with. To combat achlorhydria, ac. hydrochlor. dil. ℥i or more, may be given, well diluted with water, at meals. Arsenic seems to be the most

efficient remedy ; it should be pushed to full doses, and it has been given in the form of salvarsan or neo-salvarsan. It must not be forgotten that the pharmacopœial forms of arsenic may cause neuritis if used to excess, and that salvarsan has its own dangers. Very successful results have been obtained by means of blood transfusion.

## ALLIED FORMS OF SEVERE ANÆMIA

**Septic Anæmia.**—Hunter distinguishes from pernicious anæmia a septic anæmia, also arising from oral, gastric, or intestinal sepsis ; but in this the colour index of the corpuscles is not very different from unity ; the evidences of hæmolysis (lemon-yellow skin and urobilinuria) are absent, and the prognosis is relatively favourable. Oral sepsis may be present with the glossitis in Addison's anæmia, and materially delays the success of treatment by arsenic.

**Aplastic Anæmia** (*Aleukia hæmorrhagica*).—This disease may result from infection of the bone marrow (**8**), from poisoning with salvarsan and other benzol compounds (**9**), and from excessive exposure to X-rays. The red corpuscles and hæmoglobin are reduced to about 20 per cent. of the normal, and the colour index is below unity ; nucleated red cells are not frequent ; there is leucopenia, due to a diminution in polymorphonuclear cells. The bone marrow differs from that of pernicious anæmia in being remarkably pale and fatty, and in wanting all signs of blood regeneration. Thus there is *aplasia* of the bone marrow, which is regarded as the primary cause of the disease. The most striking abnormality in the blood is a great diminution or complete absence of blood platelets, and associated with this there is a severe hæmorrhagic tendency with delay in the bleeding time, but not in the clotting time. In fact, the disease has been regarded as identical with a severe type of purpura (*see* p. 542).

It will be remembered that the forms of jaundice called acholuric jaundice and family cholæmia are accompanied by a decided amount of anæmia, or oligocythæmia ; and that in the acquired form the resemblance to pernicious anæmia may be very close (*see* p. 532).

## SPLENIC ANÆMIA

This name is given to a condition in which great enlargement of the spleen is associated with profound anæmia. The first event is either a general anæmia or an attack of hæmatemesis, or some complaint of pain in the left side, probably due to attacks of perisplenitis. When first observed the spleen has often reached a great size, and in the course of the illness it may be large enough to extend forwards to the umbilicus and downwards to the iliac crest. The anæmia is considerable, of chlorotic type, the red corpuscles ranging from 2,000,000 to 3,000,000, and the hæmoglobin from 35 to 50 per cent. The colour index is less than 1, but not usually lower than 0·6. The leucocytes are generally in less number than normal (leucopenia), often only 1,000 or 5,000 per cubic millimetre. Normoblasts and a few megaloblasts may be present.

The disease runs a long course, often three or four years, sometimes ten or twelve years, and the anæmia slowly increases. The hæmatemesis may be repeated, and other hæmorrhages may occur, such as epistaxis or retinal hæmorrhage. The liver is enlarged, and there are digestive troubles ; but there is no enlargement of the lymphatic glands, nor, as a rule, pyrexia. In some cases there is marked pigmentation of the skin. It occurs in both sexes, and at all ages from childhood up to late middle age. In some cases after a long time the liver becomes still more enlarged, and definitely cirrhotic. Ascites then follows, though it also sometimes occurs without cirrhosis. The supervention of definite cirrhosis of the liver with ascites constitutes *Banti's disease*.

Some differences in the clinical features as well as in the results of histological examination of the spleen have been recognised in some cases, known as forming

the *Gaucher type*. The onset is usually before the age of twelve; the disease may occur in more than one member of a family, though not hereditary; and patients may survive from twenty to thirty years. The skin of the face, neck, and hands takes on a brownish-yellow tint; the blood shows leucopenia, but a diminution of the red cell count may be long delayed.

**Pathology.**—The spleen is found to be greatly enlarged, weighing from two to four pounds, firm and generally smooth, presenting hæmorrhages and infarcts on section. In thirteen cases of the Gaucher type the average weight was more than seven pounds. The histological features in the more usual cases are a great increase of the fibrous trabeculæ and septa, atrophy and fibrous transformation of the Malpighian corpuscles, and the presence of great numbers of large endothelial cells containing numerous nuclei and blood corpuscles. In the other form, or Gaucher type, there are groups of small rounded spaces filled with large cells of from 20$\mu$ to 40$\mu$ in diameter, each cell with deeply stained nuclei and homogeneous protoplasm. Around the spaces are bands of coarse connective tissue. These characteristic Gaucher cells have also been found in the lower lymph glands and even the bone marrow.

The sequence of events is by no means clear as yet. That the spleen has some influence in the production of the anæmia is shown by the fact that splenectomy has in several cases been followed by cure, the blood corpuscles more or less rapidly reaching the normal amount. It is probable that toxic processes start in the spleen, which account for its enlargement, the fibrous changes, and the anæmia. The poisons are also supposed to be conveyed through the portal system to the liver, producing the cirrhosis characteristic of Banti's disease. Gibson has described a *streptothrix* in the spleen in certain cases where it was excised. In other cases the condition is undoubtedly syphilitic, and Hurst has found that in a case of this kind, where the Wassermann reaction was repeatedly positive before excision, it became negative afterwards. In other cases splenic anæmia has been associated with thrombosis of the spleen veins. Chronic perihepatitis and perisplenitis are invariably present in long standing cases.

**Diagnosis.**—The disease may be confounded with myelocytic leukæmia, with pernicious anæmia, and with infective endocarditis. Its characteristic features are the anæmia of chlorotic type, the leucopenia, the large size of the spleen, the absence of leukæmia and of enlargements of the lymphatic glands, the long duration, and the occurrence of hæmorrhages. An examination of the blood at once distinguishes it from leukæmia: a confusion with malignant endocarditis is possible, because in this last disease the spleen may be very large, the anæmia may be pronounced, and purpura and hæmorrhages may occur; while hæmic murmurs may be present in splenic anæmia.

A diagnosis of Banti's disease may be too readily made in a case of cirrhosis with unusually large spleen; and there is a somewhat close resemblance between Banti's disease and cases occurring in Egypt where splenomegaly is associated with unilobular cirrhosis, anæmia, febrile attacks and ultimately ascites.

**Treatment.**—Iron and arsenic are of little value; and if after a period of observation the diagnosis is established the removal of the spleen is justified. Although it is not free from the danger of hæmorrhage, cases have been completely successful. In one case a preliminary examination of the bone marrow, obtained by trephining, showed that it was diseased, and splenectomy was abandoned. Röntgen rays may be tried, as in leukæmia (*see* p. 535). Blood transfusion is a valuable palliative measure.

## ANÆMIA INFANTUM PSEUDO-LEUKÆMICA

*(Splenic Anæmia of Infancy)*

This condition was described by von Jaksch, and is found in infants and young children under four years, but especially between seven and twelve months.

There is anæmia with enlarged spleen and liver, sometimes enlarged glands, and leucocytosis. The red blood cells are usually less than 60 per cent. of the normal, and poikilocytes and nucleated red cells (normoblasts and megaloblasts) are present. The leucocytes number from 30,000 to 40,000; there is a slight relative increase of lymphocytes; and a few myelocytes (1 to 6 per cent.) are present. The hæmoglobin is much diminished.

The spleen is the subject of hyperplasia with moderate fibrosis. It is still a debated question whether these cases are different from, or only an extreme form of, the cases of moderate splenic enlargement with slight anæmia which are so commonly seen in infants, and which are often associated with syphilis and rickets. The condition of the spleen is not distinctive, and a small proportion of myelocytes in the blood is normal in young infants. In any case the condition is probably due to a toxin. It has been suggested that the disease is really the same as splenic anæmia, the difference in the blood picture being due to alteration in the response of the infantile blood-forming organs. The prognosis is good, and the disease usually disappears as the child grows older.

The **Treatment** consists in the correction of any dietetic irregularities and the use of mercury in cases suspected of a syphilitic origin.

### SECONDARY ANÆMIA

The condition of the blood in cases called *secondary anæmia* is somewhat like that of chlorosis. The corpuscles may not be very much diminished in number, but the loss of hæmoglobin is relatively greater, because the cells are smaller. Microcytes as well as poikilocytes occur in the severe cases, and in the worst cases nucleated red cells, especially normoblasts. The leucocytes are variable; they are often increased in number, especially in cases dependent on inflammatory and pyogenic conditions. The coagulation time is shortened.

## ACHOLURIC JAUNDICE

*(Chronic Splenomegalic Hæmolytic Jaundice)*

In this comparatively rare form of jaundice there is no obstruction of the ducts, for the fæces retain their normal colour, and the urine is, except in aggravated attacks, free from bile pigment; but it contains urobilin. The blood serum, on the other hand, contains bile pigment, but is free from urobilin or urobilinogen. It gives Van den Bergh's indirect test. The blood, moreover, shows a marked anæmia, and the spleen is enlarged. The origin of these cases is doubtful, but they are possibly hæmatogenous, that is the bile pigment may be formed from hæmoglobin in the blood and tissues, and the hæmoglobin comes from the red cells owing to their increased fragility. That the spleen has something to do with the hæmolysis is shown by the fact that splenectomy cures the disease. It is also noteworthy that if the spleen is removed in healthy animals the normal fragility of the corpuscles is diminished. The disease occurs in two forms, congenital and acquired.

*Congenital Acholuric Jaundice.*—The jaundice is often noticed immediately after birth, or develops slowly at a later time; it may persist for years, or it clears up and recurs from time to time. The patient is anæmic; the red corpuscles are reduced to 3,000,000 or 3,500,000 and present moderate degrees of poikilocytosis, anisocytosis, polychromasia, and punctate basophilia; while nucleated red cells are present. The hæmoglobin is reduced to 50 or 45 per cent., and the colour index is slightly below unity. The leucocytes are generally fewer than normal, but occasionally there is leucocytosis; the large lymphocytes are sometimes in excess, and a few myelocytes may be present. The spleen is enlarged, and appears to grow harder with the progress of the case. The liver is only slightly enlarged,

and has not been known to be cirrhosed; it often enlarges during exacerbations and diminishes afterwards.

A striking feature in the blood is the fragility of the red corpuscles, which are broken up (*hæmolysis*) by sodium chloride solution at much lower degrees of dilution than will affect normal corpuscles (*see* p. 519).

The patients, as children, are not generally stunted as in splenomegalic cirrhosis (*see* p. 487), nor are the fingers clubbed. Moreover, they may be little troubled by their complaint, and may live for many years.

The only feature in the ætiology is its occurrence in several members of one family, and congenital syphilis does not appear to be a cause.

*Acquired Acholuric Jaundice.*—In this form of the disease the symptoms come on insidiously in adult life; the anæmia is often pronounced, and the red corpuscles may fall to 2,000,000 or 1,500,000 or less; the colour index is sometimes above unity, as in pernicious anæmia; the jaundice is often very slight; the spleen is enlarged. The fragility of the red corpuscles is less pronounced than in the congenital cases.

**Treatment.**—Arsenic appears to have no influence upon either form of the disease, and salvarsan has also failed. But the removal of the spleen has brought about recovery in both acquired and congenital cases, and this should be carried out if the anæmia is severe.

## LEUKÆMIA

(*Leucocythæmia*)

These names are given to those cases of disease in which there is a considerable and persistent increase in the number of white corpuscles in the blood, associated with changes in the marrow of the bones, and in the spleen or the lymphatic glands.

In all varieties of leukæmia, the bone marrow is diseased, in many the spleen, in some the lymphatic glands, and in a few all three of these blood forming organs; in all varieties the natural proportions of the various leucocytes to one another are profoundly altered, and in some the blood contains myelocytes or marrow cells, which are not present in it in health.

It is customary to base divisions of leukæmia upon the condition of the blood, and the predominance of this or that form of leucocyte.

The two chief varieties which are known to occur are (1) one in which myelocytes are found in the blood to the extent of 30 or 40 per cent. of the leucocytes; this is *myelocytic leukæmia*, or *myeloid leukæmia*, or *leucoid leukæmia;* (2) another in which lymphocytes are in the blood to the extent of 90 or 95 per cent. of the leucocytes; this is *lymphocytic leukæmia*, or *lymphocythæmia*, or *lymphoid leukæmia*. In some of these cases the cells present are large, and are the precursors of the lymphocytes; in other cases the cells are almost entirely normal lymphocytes, large or small. In some cases patients who have suffered from one variety of leukæmia have subsequently developed the other variety of the disease.

The red corpuscles are always diminished in number, and the hæmoglobin also, with a colour index less than unity.

**Ætiology.**—The cause of leukæmia in any form is really unknown, and there are no constant antecedents to explain its occurrence. Occasionally, however, leukæmia follows closely upon some septic, toxic, or other general condition, *e.g.* sepsis, broncho-pneumonia, syphilis, variola or cancer, so as to have a recognised causation and deserve the name *secondary* (G. Ward). The cases are wanting in some of the features of primary cases, and the patients often recover. The myelocytic variety occurs in men more often than in women, and mostly in middle life, though sometimes in quite young children,

but rarely in infants. Lymphocytic leukæmia is more common in young people.

**Pathology.**—Apparently in all cases the bone marrow undergoes some form of irritation, by toxins or otherwise, and produces an excess of leucocytes with a less formation of red corpuscles or erythrocytes. The various types of leucocytes have already been described (p. 520). There is over-production of leucoid cells or of lymphoid cells, giving rise to the two characteristic types of the disease and leading to the flooding of the circulating blood with immature forms of leucocytes. As the disease continues still greater pressure is brought to bear on the depôts where the white cells are manufactured, and the cells are turned out into the blood in a still more primitive form. In certain cases this increased activity of the normal depôts is accompanied by the formation of fresh leucocyte-forming areas in abnormal situations, which will partake either of myeloid or of lymphoid character. These infiltrations may form nodules beneath the skin, or in different parts of the body. They may have a green appearance - so-called chloroma. What is the share of the spleen and lymph glands in the production of the leucocyte excess is very uncertain Their enlargement, when it occurs, may be due to their diseased activity, and many regard an excess of small lymphocytes as mainly dependent upon the lymph glands, or the swelling may be, as some think, due to the organ being densely packed with the leucocytes circulating in excess in the blood.

In leukæmia the basal metabolism (*see* p. 558) is increased.

## MYELOCYTIC LEUKÆMIA

(*Spleno-medullary Leukæmia, Leucoid Leukæmia, Myeloid Leukæmia*)

**Condition of the Blood.**—In well-marked examples of leukæmia the blood is pale and thin as it issues from a wound, and as seen after death it is grumous-looking, or forms pale pus-like clots. Under the microscope the white corpuscles are seen to occupy nearly the whole of the field, instead of being few and scattered, as in health; on the other hand, the red corpuscles are less numerous than in health. The white corpuscles are found in number from 200,000 to 900,000 in the cubic millimetre, instead of 8,000 or 9,000; and the red corpuscles may be from 3,000,000 to 2,000,000, or even as low as 1,000,000. The hæmoglobin is less in proportion than the red corpuscles; *i.e.*, the colour index is less than unity. Poikilocytes are present; nucleated red cells (normoblasts and megaloblasts) are numerous.

In the earlier stage of the disease polymorphonuclear cells are most abundant; most of them are much larger than normal, and some are obviously degenerated. Proleucocytes with horseshoe-shaped nuclei are also present, and some typical granular and eosinophil myelocytes. The eosinophil and mast cells are increased, while the lymphocytes are relatively greatly reduced. At a later stage there are few normal leucocytes present, but there are proleucotytes, myelocytes and promyelocytes and mast cells in abundance. Later still myeloblasts predominate. If the disease runs a rapid course, so-called acute myeloid leukæmia, the more primitive cells are seen in large numbers from the beginning.

Charcot-Leyden crystals (*see* p. 228) have been found in the blood, and in the spleen and other organs after death. Chemical examination shows a great increase of the xanthin bases and uric acid, which are believed to result from the destruction of the leucocytes. Lactic, formic, and mucinic acids have also been found.

**Leukanæmia** is a condition where the red cells show the characters of pernicious anæmia, but where, as far as the white cells are concerned, the picture is that of leucoid leukæmia. However, the number of white cells is not so large as in true leukæmia.

**Anatomical Changes.**—The spleen often weighs 5 or 6 lbs., but a weight

of 18 lbs. has been recorded. It is uniformly enlarged, and retains its normal shape; on the surface are often patches of thickening of the capsule, and the organ is more or less adherent to the abdominal wall, diaphragm, or adjacent viscera. On section it often has a brownish rather than a red colour, which is uniform, or marked with paler lines due to thickened trabeculæ. It is smooth, hard and dry. Not infrequently there are large wedge-shaped infarcts, either yellow and caseous or red and hæmorrhagic. The change in the spleen itself is one of great increase of the splenic pulp, which is full of the same cells as are found in the blood, and the outlines of the Malpighian bodies are badly defined; in long-standing cases the stroma becomes more fibrous, and the trabeculæ are increased in thickness.

The *liver* is enlarged, and may reach twice or three times its normal size. It is pale and smooth, and may present under the microscope a dense infiltration with leucocytes (myelocytic infiltration), which for the most part surround the portal vessels in their distribution, but are partly in nodular masses. The vessels also are full of leucocytes. The *kidneys* are pale, and enlarged from granular degeneration of the cells, and distension from leucocytes; or they present greyish, white deposits, running like striæ between the cortical tubules. There may be stomatitis or pharyngitis, swelling of the *tonsils*, and of the *follicles* at the root of the tongue, and swelling and superficial ulceration of the follicles, of the intestine. The *thymus*, *thyroid*, and *suprarenal glands* may also be diseased, and tumours in the skin have been recorded. Sometimes the *lungs* present hæmorrhagic infarcts. The *marrow* of the bones is either yellow and pus-like, or pink and firm, the fat of the marrow being replaced by a tissue like that of active marrow, in which myelocytes and nucleated red cells are abundant, with eosinophils sometimes, and myeloblasts or large lymphocytes. Besides the occasional hæmorrhage into the brain, diffuse sclerotic changes and scattered areas of acute inflammation have been found in the *brain* and *spinal cord*.

**Symptoms.**—One of the first indications of leukæmia, in a great number of cases, is the swelling of the abdomen from the *enlargement of the spleen*, which may have been developing for some time without giving any sign. It may then be found occupying the whole of the left side of the abdomen, forming a firm, hard tumour, which extends backwards into the flank, while its anterior margin begins about the ninth costal cartilage, reaches the middle line at the umbilicus level, and not infrequently below this extends 2 or 3 inches to the right. This position is determined by its attachment to its vessels, which compel it to enlarge along the circumference of a circle of which the cœliac axis is a centre. The anterior margin is more or less sharp, and presents one or two notches. In earlier stages the spleen only occupies the left hypochondriac region, like the enlargements in malaria, and in some cases of typhoid fever. The *liver* is moderately enlarged, and can be felt for 1 or 2 inches below the right costal margin. The implication of the *bone marrow* is sometimes shown by tenderness on pressure or percussion of the corresponding bone.

The general effects of the illness do not always show themselves early; especially it must be observed that when the spleen is already very large and the leukæmia unmistakable, there may be no pallor of the skin, lips, and mucous membranes. Later on, however, the patient loses colour, and becomes sallow, or markedly anæmic.

The *temperature* is generally affected in this disease; there is either continuous moderate pyrexia, or there are periods of pyrexia alternating with periods of apyrexia; and the febrile reaction sometimes gives a flush to the skin, which may help to mask the approaching anæmia.

The altered condition of the blood shows itself in the occurrence of *dyspnœa* and of *hæmorrhages*, which last chiefly take the form of epistaxis, bleeding from the gums and mouth, and purpuric spots under the skin, but also occasionally bleeding from the lungs, stomach and intestines, kidneys, or uterus, or hæmor-

rhage into the brain. Hæmorrhages also occur in the retina, where they may be seen with the ophthalmoscope associated with white streaks and spots, said to consist of masses of leucocytes; and the retinal veins are often remarkably tortuous (*leukæmic retinitis*). Other organs may be affected, but the changes are chiefly observed after death. The urine is acid, of high specific gravity, uric acid and xanthin bases are in excess, and indican is often present. Albumin is rare unless the kidneys are diseased.

The course of the disease is generally progressive until its termination in death, and it lasts from six months to five years. Cases of much shorter duration (*acute myeloid leukæmia*) are rare; and in them, as well as in the terminal stages of more chronic cases, *myeloblasts* may to a large extent replace the myelocytes (*see* p. 520).

Towards the end the pallor increases, the feet and other parts of the body become œdematous, ascites and hydrothorax may be added, the pulse is quickened, and palpitation is frequent. Diarrhœa is occasionally a prominent symptom. Finally, death takes place from loss of blood, asthenia, diarrhœa, pleurisy, pneumonia, bronchitis or cardiac dilatation, and occasionally from cerebral hæmorrhage.

**Diagnosis.**—The diagnosis generally depends upon the recognition of an enlarged spleen (*see* p. 549) and the examination of the blood; the last is absolutely essential. An equally large spleen may be seen in *lymphocytic leukæmia*, in *splenic anæmia*, in *polycythæmia*, and in malignant endocarditis. Even when the patient has a good colour there may be pronounced leukæmia. The presence of myelocytes, as well as the excess of polymorphonuclear cells and eosinophils, are the distinguishing features of this disease.

The **Prognosis** is unfavourable, and recovery is uncommon.

**Treatment.**—Arsenic is the drug which seems to have given most promise; it must be used perseveringly and in increasing doses as long as it can be borne; and under its use the spleen has diminished in size considerably, and the leucocytes in number. Mosler has injected arsenic into the substance of the spleen. Under treatment by benzol remarkable diminution in the number of the leucocytes and in the size of the spleen has been observed. The daily dose is 30, 60 or 90 minims taken in capsules, with an equal amount of olive oil. This treatment was introduced by Selling, who found that benzol workers often suffered from grave leucopenia. The local application to the spleen of the icebag, cold douche, or electricity (galvanic current) may reduce its size. The Röntgen rays, especially the penetrating rays used in deep X-ray therapy, applied to the splenic region and to the epiphyses of the long bones (femur) have a decided influence both in reducing the number of the leucocytes and the size of the spleen, which may both become normal. The rays are applied every day or every other day for fifteen or twenty minutes, and the treatment must be continued for months. But a relapse is certain to occur, and if the leucocytes are reduced beyond the normal proportion, disagreeable symptoms ensue.

Splenectomy has been uniformly fatal from collapse or hæmorrhage.

### LYMPHOCYTIC LEUKÆMIA

(*Lymphatic Leukæmia, Lymphoid Leukæmia, Lymphocythæmia*)

This is of rarer occurrence than the myelocytic form, and the cases present much wider differences.

**Condition of the Blood.**—The feature of the blood is the immense predominance of the *lymphocyte*. A few prolymphocytes and large mononuclear cells are also present in the earlier stages. These lymphocytic cells often form from 90 to 95 per cent. of the white cells. The *polymorphonuclear* cells form only from 2 to 5 per cent., and there are a very few *eosinophils* and mast cells In later stages there are fewer normal lymphocytes, but there are prolymphocytes,

lymphomyelocytes and myeloblasts. In the latest stage of the disease myeloblasts and lymphomyelocytes are practically the only cells seen. In acute lymphatic leukæmia running a rapid course the larger immature type of cells predominates. The total number of leucocytes per cubic millimetre may be several thousands, or may be not much above the normal; but if the proportion of lymphocytes on a differential count amounts to 85 per cent. or more, the condition of lymphatic leukæmia must be recognised. The red corpuscles may be about 3,000,000; a few nucleated red cells are seen.

**Lymphanæmia** (cf. Leukanæmia).—The picture of pernicious anæmia is combined with that of lymphoid leukæmia, except that the number of lymphoid cells is never very large.

**Symptoms.**—As contrasted with myelocytic leukæmia, cases of this form are usually more rapid in their course, the spleen is rarely quite so large, and the various glandular organs all over the body, racemose as well as ductless, are often extensively involved. It is also to be especially noted that, although the blood contains an extraordinary number of lymphocytes, the lymphatic glands, whether superficial or deep, are often not appreciably or much enlarged. The following groups of cases may be recognised:—

First, those described as *acute leukæmia*. This occurs in both sexes, and at all ages between seven and fifty-eight. Fraenkel mentions as antecedent conditions anæmia, pregnancy, injury, and some infectious diseases, especially influenza. The illness is fatal in from two to eight or nine weeks. It begins insidiously with general weakness and malaise or pains in the spleen or joints. The external glands may enlarge, but are not always very prominent; there is slight enlargement of the spleen and liver, and the bones may be tender. A striking feature in many cases has been severe stomatitis with sloughing and gangrene of the gums; and with this there are fever, a high degree of anæmia, and hæmorrhages from the gums and bowels and under the skin. The characteristic feature is the presence of the various immature types of lymphocyte.

Secondly, there are cases, almost equally deserving the title of acute leukæmia, which are fatal in three or four months, and in which sloughing gingivitis or stomatitis is not a feature, and the lymphocytes may be of normal type. In these cases also the external glands may not be very prominent, but many or all of the solid glands in the body are densely packed with lymphocytes, and hence considerably enlarged, for instance the liver, spleen, kidneys, suprarenals, pancreas, salivary glands, and lachrymal glands, while the thymus persists and is greatly enlarged, and the cardiac muscle may be also infiltrated with lymphocytes. Exophthalmos has been also observed from leukæmic infiltration of the orbital fat. More or less fever, hæmorrhages, and dropsy occur, and death soon follows.

There are, again, other cases in which the glandular enlargement occurs first of all, and the duration is from six months to three or four years. The lymphocytes vary in type with the duration of the disease. The lymphatic glands all over the body are affected, and they may be felt in the neck, groins or axillæ. They are moderately large, not very hard, and move freely upon one another. The mesenteric glands are even more often enlarged than the above; the retroperitoneal, thoracic, portal, and iliac glands less so. On section the glands are whitish pink in colour, and microscopically are found to be distended with the lymphocytes. The bone marrow is full of the same leucocytes, and the thymus and other organs may also be involved as above. In these cases also the spleen and liver may be very greatly enlarged. Sometimes the excess of lymphocytes in the blood is not apparent until a late stage, so that the glandular enlargement, with no excess of leucocytes, may simulate Hodgkin's disease (*see* p. 551).

Some cases in which nodules of lymphatic tissue appear in the skin, bones, wall of the bowel, and elsewhere, have been called *nodular leukæmia*.

**Diagnosis.**—In any obscure illness with pallor, or enlarged glands, tonsils

or spleen, or hæmorrhages, or purpura, or sloughing gums, the blood should be examined and the lymphocytes carefully estimated. A leucocytosis of any degree with 85 or 90 per cent. of lymphocytes must be regarded as lymphatic leukæmia. The **Prognosis** is bad in the acute cases, in which there is little time for **Treatment** either by arsenic or by deep X-ray therapy. In less rapid cases these remedies should be tried.

### CHLOROMA

Presenting close relations with lymphatic leukæmia is the condition which has been called chloroma. In this there are numerous tumours or lymphoid deposits, especially in the orbits (so that exophthalmos may take place), in the temporal fossæ and in the periosteum of the bones of the skull. Tumours may also grow on the conjunctiva and under the skin, and sometimes even during life these tumours have a green colour (*green cancer*).

The patients suffer from a cachexia similar to that of leukæmia; there are prostration, anæmia, hæmorrhages into the retina and elsewhere, and optic neuritis. The blood presents a condition of lymphocythæmia; the lymphocytes reach from 70 to 90 per cent.; they are often, but not necessarily, large cells of primitive type. Cases of myeloid chloroma also occur, but they are rarer.

After death the various tumours are seen to have a green colour, which fades away on exposure; and the lymphatic glands, spleen, bone marrow, and other organs are in a condition similar to that accompanying lymphocytic leukæmia. The exact nature of the green colour is not known: it is not bile pigment.

## LEUCOCYTOSIS

Leucocytosis is the name given to a temporary increase of the white corpuscles either as a physiological event or in response to irritation by various infective toxins. The leucocytes rise to 15,000, 20,000 or 30,000, but rarely higher than this. It occurs in pregnancy and as a temporary event during the process of digestion (*digestion leucocytosis*). The following are some of the diseases in which it is constantly found: acute inflammatory diseases, suppuration from any cause, pyæmia, erysipelas, cerebro-spinal fever, pneumonia, pleurisy, empyema, phthisis, scarlet fever and rheumatic fever. The polymorphonuclear leucocytes are usually increased in these conditions.

There are, however, diseases in which other cells than the polymorphonuclear are predominant; for instance, in typhoid fever the small lymphocytes increase beyond 30 per cent. (*lymphocytosis*), and in malaria the large mononuclears reach from 11 to 20 per cent. An increase of the eosinophil cells (*eosinophilia*) occurs in some skin diseases, in asthma, and in some parasitic diseases (filaria, bilharzia, trichina, ankylostoma, ascaris, hydatid). As a rough guide, it may be said that an excess of polymorphonuclears accompanies a *coccal* infection, of lymphocytes a *bacillary* infection, and of large mononuclears a *protozoal* infection.

## LEUCOPENIA

Diminution of the leucocytes cannot be considered to be a disease in itself, but it has important relations with other changes in the blood. It occurs in long-continued fevers and in different forms of anæmia; and is a constant feature of splenic anæmia, when the leucocytes may number only 4,000 or 3,000.

## POLYCYTHÆMIA RUBRA

Polycythæmia, or polycythæmia rubra, in which the red cells of the blood are increased, occurs (1) as a primary disease of the red cell-forming organs

(*erythræmia*); (2) it also results from some disturbance in the circulatory respiratory systems, causing oxygen want and requiring for compensation a larger number of carriers of hæmoglobin, such as in congenital heart disease. This secondary polycythæmia is called *erythrocytosis*.

Cases of polycythæmia rubra have an increased basal metabolism (*see* p. 558).

### ERYTHRÆMIA

This occurs mostly in patients of between thirty and sixty years of age, though occasionally they are above or below these limits. In its pronounced form the red corpuscles vary in number from 9 and 10 to 13 and even 14 millions per cubic millimetre, and on standing the corpuscles may be found to occupy nine-tenths of the volume of the fluid The hæmoglobin is raised to 130, 160 or 180 per cent. of the normal. The colour index is low, which shows that the red cells are small.

The leucocytes are not always increased in number, but they may reach 24,000 per cubic millimetre. The viscosity of the blood is increased to three or four times the normal. The specific gravity and the coagulation time are not constantly either below or in excess of the normal. The blood pressure is sometimes, but not always, high.

The conditions usually associated with this polycythæmia are *cyanosis* and a moderate or considerable enlargement of the spleen.

The cyanosis is shown chiefly on the face, ears and mucous membranes. The face has a characteristic florid appearance. The veins of the retina are engorged and very dark.

The patients suffer from headache or a sense of fulness in the head, lassitude, vertigo, dyspepsia, constipation, thirst, and various forms of hæmorrhage, which include epistaxis, bleeding from the gums, menorrhagia and cerebral hæmorrhage.

Sometimes the arteries are sclerotic, and the urine may contain a little albumin. In some cases extensive venous thrombosis has occurred. In fatal cases the enlarged spleen has been found to be engorged, with some hyperplasia of the splenic pulp, but generally without evidence of erythroblastic or myeloid activity. The lymph glands are generally unaffected: the liver may be engorged. The bone marrow is generally deep red in colour, and no fatty marrow is seen, so that there appears to be a great increase in the function of red corpuscle formation.

In accordance with this, the generally accepted view of the pathology of the condition is that, from some cause or other, the bone marrow is stimulated to an excessive formation of erythrocytes, and that the other changes are secondary. There is no evidence that the polycythæmia is secondary to oxygen want in the tissues. One patient, who was kept for five days in a chamber containing 40 per cent. oxygen, showed no alteration in the blood at the end of that time. For some unexplained reason there is a lowered alkali reserve of the blood in this disease.

Differing somewhat from the above cases, which were first described as *polycythæmia with splenomegaly*, are some cases, less frequently occurring, in which the spleen is not enlarged, but the blood pressure is greatly increased, and may even reach 300 mm. Hg. The patients are often turgid in the face and may have hypertrophy of the heart, albumin in the urine, and signs of arterio-sclerosis. They were first described by Geisböck, who called them *polycythæmia hypertonica*. The patients are liable to apoplectic seizures, and may have chronic nephritis.

The progress of cases of erythræmia is variable; death has occurred from increasing cyanosis, or from cerebral vascular complications, or from tuberculosis.

**Treatment.**—Periodic venesection is the best treatment for this disease. From ½ to 1 pint of blood should be removed every six months through a wide

hollow needle. Citrate may be used to prevent clotting, as in the withdrawal of blood for transfusion purposes.

### ERYTHROCYTOSIS

Under this term it is proposed to include the cases in which the polycythæmia is again due to increased activity of the bone marrow (Parkes Weber), but this activity is stimulated by recognisable antecedent conditions. Thus one class is constituted by chronic cardiac and pulmonary lesions, of which congenital malformation of the heart is the most striking; and others are the various forms of acquired valvular disease, emphysema and chronic pulmonary diseases, which may be accompanied by cyanosis (Ayerza's disease). In these the deficient oxygenation of the blood is the stimulus to the bone marrow. Patients who have been "gassed" form another group. Here the erythrocytosis has been lessened by treatment in an oxygen chamber containing 40 per cent. oxygen (Hunt and Dufton). Another group is formed by the polycythæmia of persons resident at high altitudes, where the increase of erythrocytes compensates for deficient oxygen tension in the air available for respiration.

Erythrocytosis has also been observed to be secondary to tuberculosis of the spleen, and also to thrombosis of the splenic and portal veins.

In both classes of cases the red corpuscles may reach seven, eight, or nine millions; but the numbers may be different in blood taken from arterial, venous, and capillary vessels. As in erythræmia, the hæmoglobin and the viscosity of the blood are raised in amount.

Polycythæmia also occurs in poisoning by phosphorus and carbon monoxide; it may be induced by injection of serum from an animal in which an erythroblastic reaction is in progress; and it has been found in some infective diseases (syphilis, tubercle).

## HÆMOGLOBINÆMIA

Hæmoglobinæmia arises when blood corpuscles are broken up in the blood vessels, so that hæmoglobin escapes into the plasma, giving it a pink tinge. The hæmoglobin is then excreted by the kidneys, so that the urine is coloured deep red; this condition is called *hæmoglobinuria*, and is distinguished from *hæmaturia*, in which blood itself with its corpuscles is mixed with the urine. Very slight hæmolysis may sometimes be seen in healthy individuals.

A partial destruction (*hæmolysis*) of the corpuscles takes place under several circumstances: (1) The action of certain poisons, such as chlorate of potassium in large doses, pyrogallic acid, arseniuretted hydrogen, and naphthol. (2) The transfusion into one mammal of the blood of another; each kind of corpuscle becomes destroyed, and the serum is stained with hæmoglobin. In other words, the blood of one animal is hæmolysed by the serum of an animal of another species. (3) Exposure of the skin to extremes of temperature, such as burns or frost bite. (4) The action of some fevers, so that a moderate degree of hæmoglobinæmia may result from scarlet fever or typhoid fever. (5) In blackwater fever (*see* p. 157). (6) In an *epidemic hæmoglobinuria* of infants (Winckel's disease). (7) The paroxysmal form of hæmoglobinuria, in which the altered condition of the serum has been also demonstrated.

Under all these circumstances the plasma has a reddish colour, and the blood corpuscles have little tendency to form rouleaux. Very pale corpuscles (*shadow corpuscles*) are seen, and the hæmoglobin is deficient. Possibly the excretion by the kidney does not occur if the destruction is confined to the blood in the portal circulation (*see* p. 527).

In hæmoglobinuria the urine is red, dark red, or reddish-brown; it is acid and deposits a dirty brown sediment of epithelium, pigmented *débris* or corpuscles, perhaps casts containing blood pigment, darkly stained urates, and opaque red

granules of hæmoglobin. No blood corpuscles can be seen. When examined by the spectroscope, the urine gives the two bands in the green and yellow characteristic of oxyhæmoglobin, and frequently another band nearer the red end of the spectrum, which is due to methæmoglobin. The latter is produced by the action of the urine on the oxyhæmoglobin. The urine always contains a protein, either serum albumin or globulin.

## PAROXYSMAL HÆMOGLOBINURIA

In this comparatively rare complaint hæmoglobinuria occurs in isolated attacks.

**Ætiology.**—It is seen in young adults and middle-aged people up to fifty years of age, and is much more common in males than in females. In a few cases there is a history of malarial poisoning, in very many a history of syphilis; and rheumatism is said to be an occasional antecedent. Hereditary tendency does not play a prominent part. The most common immediate cause of an attack or paroxysm is exposure to cold, as from going out insufficiently clothed on a winter's day, or driving or walking far in the cold, or bathing in cold water. Exertion is another exciting cause.

**Pathology.**—Paroxysmal hæmoglobinuria is consequent upon hæmoglobinæmia; hæmolysis takes place within the blood vessels, and the serum is stained with hæmoglobin. This escapes into the urine probably as oxyhæmoglobin, and by contact with urine is converted into methæmoglobin. In slighter attacks probably only a small number of corpuscles are disintegrated, the hæmatin is disposed of in the liver, while the globulin alone is excreted in the urine; the protein found in the urine in such conditions is indeed globulin and not serum albumin.

The occurrence of the hæmolysis as a result of cold and other excitations has to be explained; and the view is put forward that there is a potential toxic *hæmolysin* in the blood of sufferers from this complaint, and that this consists of two parts, amboceptor and cytase, or complement. It is suggested that the action of cold is to cause the union of amboceptor with the corpuscle, and that subsequent co-operation of the complement with the return of warmth brings about solution of the corpuscles. The complement exists in normal blood, and it has been shown that the patient's serum will hæmolyse normal corpuscles, hence proving that there need be no specific liability of the patient's corpuscles to break down.

The toxic origin of the hæmolysin is suggested by the fact that in the majority of patients there is a history or evidence of syphilis, and a positive Wassermann reaction.

**Symptoms.**—The beginning of the attack is marked in different cases by languor and weariness, a disposition to yawn, chill or rigor, pains in the limbs, nausea, vomiting, diarrhœa, and abdominal pain. The patients often suffer from Raynaud's disease (*see* p. 381). The fingers become white and cold, or the fingers, tip of the nose, and edges of the ears may become cold, livid, and even slough.

The temperature may rise at the commencement, but soon subsides; and the whole duration of these symptoms is only from two to twelve hours. A slight enlargement of the liver and of the spleen is sometimes also observed. Either immediately after the first symptom, or only after three or four hours, the blood-coloured urine is passed, having the characters already described. But even this condition is only of short duration: in a few hours more the urine may be perfectly clear, and free from albumin and hæmoglobin; and in the intervals between the attacks it is always perfectly normal. Fagge pointed out that in some subjects of hæmoglobinuria slighter chills are followed by transitory albuminuria. The effect of a paroxysm upon the blood corpuscles is striking: whereas before an attack the red corpuscles are perfectly normal, after an attack

they are found to present all shapes and sizes, with microcytes and a few megalocytes, and great variability of hæmoglobin content, from excess to entire absence (*shadow corpuscles* or *ghosts*). Globules of hæmoglobin may be seen in the serum. Towards the end of an attack an icteric tinge of the skin is observed; and after many attacks in quick succession the patient becomes anæmic, the red corpuscles fall in number to 3,000,000 or 2,000,000, and the hæmoglobin to a somewhat less extent proportionately. Recovery of the red corpuscles takes place with remarkable rapidity.

Paroxysmal hæmoglobinuria is not in itself dangerous; but the presence of renal cells and casts in the urine indicates that a nephritis is set up by the passage of the hæmoglobin; and in experiments with toxic agents producing hæmoglobinuria the kidneys are found to be of a dark chocolate colour, from masses of the pigment collecting in the straight and convoluted tubes and in the glomeruli. The disease may last several years, and if the attacks are frequent the patient becomes persistently anæmic, and acquires a sallow or faintly icteric tinge.

**Treatment.**—Exposure to cold must be carefully and systematically avoided by the use of warm clothing, residence in warm rooms, and protection from night air as far as possible. It is much more difficult to diminish the susceptibility to its influence which practically constitutes the disease. If there is any reason to suppose that syphilis is a predisposing condition, the corresponding remedies should be employed; if malaria is known to be an antecedent, quinine and arsenic should be tried. Their use must, of course, be continued for some time, and they may be given in daily doses of 5 or 6 grains of quinine and 10 or 12 minims of the liquor arsenicalis. During the attack the patient should be made thoroughly warm by going to bed, and probably any renal irritation will be lessened by taking large quantities of fluid.

## METHÆMOGLOBINÆMIA AND SULPHÆMOGLOBINÆMIA

(*Enterogenous Cyanosis, Microbic Cyanosis*)

A general lividity or cyanosis of the skin and mucous membranes is caused in rare cases by the conversion of the oxyhæmoglobin of the corpuscles into methæmoglobin and sulphæmoglobin.

*Methæmoglobinæmia.*—This has happened from the use of certain drugs, especially acetanilide, phenacetin, antipyrin, veronal, and also from the absorption of nitrites in some intestinal lesions when diarrhœa is a prominent symptom, and when the production of nitrites may be due to organisms, as, *e.g.*, the *Bacillus coli*. In some cases polycythæmia has been present, and sometimes enlargement of the spleen and clubbing of the fingers and toes. Methæmoglobinæmia may arise acutely from the inhalation of poisonous fumes in workers dealing with coal-tar products, nitrobenzol, etc. The patients have a ghastly chocolate colour. The blood shows no hæmolysis, but the corpuscles are very dark. In one case of poisoning about half the hæmoglobin had been converted into methæmoglobin. The urine is generally normal. The **Treatment** is to remove the cause. Emetics and lavage may be required in poisoning by drugs. Since death is caused by deficient oxygen-carrying power of the blood, continuous oxygen administration by mask and valves or nasal catheter must be employed. After some hours the blood becomes normal spontaneously.

*Sulphæmoglobinæmia.*—About fourteen cases of this condition have been described. The patients, who are constipated, suffer from attacks of cyanosis, sometimes leading on to unconsciousness. Other symptoms in the attacks are headache, nausea and vomiting, and abdominal pain. The hæmoglobin in the corpuscles is largely converted into sulphæmoglobin. This substance resembles methæmoglobin in its absorption spectrum, a band being seen in the red. How-

ever, in the case of methæmoglobin this band disappears on adding ammonium sulphide, whereas in the case of sulphæmoglobin it remains unaltered. Another test is to pass acid-free CO through the mixture. All the bands of sulphæmoglobin are shifted towards the blue end of the spectrum. The bands of methæmoglobin are unaltered.

The condition is due to the formation of poisons in the alimentary canal. Sulphuretted hydrogen, nitrites, or other reducing substances may be concerned.

**Prognosis.**—The disease is not fatal.

**Treatment.**—Purges should be given for the constipation. Carious teeth should be removed. Oxygen should be administered continuously during the attack, especially if the patient is unconscious.

## PURPURA

This term is applied to a diseased condition in which a number of hæmorrhages occur under the skin, so as to produce blotches of a more or less purple colour. It has been already seen that there are similar hæmorrhages in a number of diseases, for instance in scarlatina, measles, variola, typhus, cerebro-spinal fever, and plague; in cirrhosis, acute yellow atrophy of the liver, and leukæmia, aplastic anæmia, and malignant sarcomatous growths (*sarcomatosis*); in malignant endocarditis, and other diseases of the heart; and in some nervous diseases, *e.g.* tabes dorsalis. Hæmorrhages will be again mentioned in connection with hæmophilia, Hodgkin's disease, Bright's disease, and scurvy. As a direct result of poisoning from without, purpura arises from overdosing with potassium iodide, and from the commercial use (handling and inhalation) of benzol or its chief constituent, benzene. *Purpura rheumatica* (*Peliosis rheumatica* or *Schönlein's disease*) is regarded by some as a hæmorrhagic erythema occurring during acute rheumatism, or in a rheumatic patient; while by others the arthritis is held to be the result of the purpura or its antecedent. Osler includes under the term any case in which purpura, erythema exudativum, purpura urticans, or urticaria is associated with multiple arthritis. The illness may set in with sore throat, and pyrexia occasionally causes endocarditis or pericarditis, and is very liable to relapse. Others limit the term to actual hæmorrhages, which may occur during rheumatism or in rheumatic subjects, first seen on the legs, worse in the evening or reappearing then after improvement, persisting for long periods, but rarely fatal. In all these cases it is clearly recognised that a cause for the hæmorrhage exists, and this cause is often an infective toxin or other poison.

Purpura also occurs under conditions where there is no obvious primary cause, although recently in these cases a hæmolytic streptococcus has been implicated. The disease has been divided clinically into four different varieties: *P. simplex*, *hæmorrhagica*, *fulminans* and *Henoch's purpura*. These are probably all varieties of the same disease.

**Pathology.**—In severe cases of purpura a hæmolytic streptococcus can usually be isolated from the blood during the febrile period, and in fatal cases it can be cultivated from the heart's blood. The endothelium of the aorta is often stained with hæmoglobin pigment. These facts point to this organism as the cause of the disease, the variety of purpura in any case depending on the severity of the infection. The blood shows the characters of a secondary anæmia.

The blood platelets probably play an important part in the clotting process inside the body, since by their lysis they liberate a precursor of the fibrin ferment. In purpura the platelets are absent or greatly diminished (*i.e.* under 100,000 per cubic millimetre (normal 200,000 to 500,000))—a condition known as *thrombocyto penia*—and purpuric conditions have been produced experimentally in animals by injecting them with anti-platelet sera, so as to destroy the platelets. The coagulation time of the blood as tested outside the body is normal; but the "bleeding time," *i.e.* the length of time that blood continues to flow after

puncturing the ear, may be increased from the normal two to four minutes up to ten or even twenty minutes. Further, after the blood has clotted outside the body the clot does not retract and allow serum to ooze out (**12, 15**).

**Symptoms.**—In its mildest forms (*P. simplex*) purpura consists in the appearance of spots of a dull red, deep red, or bluish-purple colour in different parts of the body. They are circular, vary in diameter from a millimetre to ⅛ inch, do not disappear on pressure, and are generally, when of this small size, not raised above the surface. They have no special relation to the position of the hairs. In some cases they occur only on the feet and legs, but in others are scattered uniformly or indiscriminately. Each spot fades after a time, becoming brown or yellow in tint, and the larger patches go obviously through the changes characteristic of a bruise. Very little constitutional disturbance accompanies the eruption; the patient may be pale, and loses appetite. Recovery generally takes place in from ten to twenty days.

In severe cases the hæmorrhages are more extensive, the skin may be raised by large masses of blood beneath it, and bleeding takes place from the various mucous membranes (*P. hæmorrhagica*). The nose, mouth, stomach, and intestines, the kidneys, the female genital organs, and occasionally the bronchial mucous membrane may thus be the source of the blood. The gums are never swollen as in scurvy, but sometimes a spot of hæmorrhage is seen in their substance If the loss of blood is considerable the patient becomes anæmic, and in the severer cases there may be some rise of temperature, and a stage of prostration ensues which terminates in death. Indeed, the hæmorrhagic forms are very often fatal, and post-mortem examination may reveal other ecchymoses in nearly all the mucous membranes, in the pelvis of the kidney, in the pleura, pericardium, peritoneum, in the meninges, and even in the lungs and the medulla of the bones. A cerebral hæmorrhage may be the cause of death. Sloughing and ulceration of the intestinal mucous membrane have also been found, leading to perforation and peritonitis.

*Purpura fulminans* is the name given to some cases which are fatal in from five hours to three days. Extensive hæmorrhages take place under the skin, but in the majority of cases there is no bleeding from the mucous membranes. Many of these cases have occurred after scarlatina (*see* p. 30).

In *Henoch's purpura* the lesion of the skin, which may be erythematous or urticarious as well as hæmorrhagic, is accompanied by joint pains or swellings, attacks of abdominal pain, vomiting and hæmorrhage from the bowel, hæmaturia, and nephritis. It occurs in children, and recurs frequently during weeks or months. The sequence of symptoms in these cases varies a good deal, and the purpuric eruption is often late in its appearance, and not always very extensive. On the other hand, the early occurrence of the joint pains may give rise to a diagnosis of rheumatism, and in many instances the abdominal symptoms are the most prominent. Thus abdominal pain, vomiting, and distension sometimes suggest intestinal obstruction or appendicitis; or the same symptoms with hæmorrhage from the bowel and a palpable tumour occurring in a child lead to a diagnosis of intussusception. Laparotomy has been performed in such circumstances: and sometimes an intussusception has been found, and has been easily reduced; or the supposed intussusception has proved to be a portion of bowel infiltrated with effused blood. The urine may contain much albumin, without blood or casts, or pure or altered blood. Many cases are fatal; others recover, but in them the albuminuria may persist for months.

**Diagnosis.**—In making the diagnosis, all the possible causes of a petechial eruption mentioned in the first paragraph must be excluded. *Scurvy* is distinguished by the spongy condition of the gums, the subcutaneous or fascial indurations, the greater degree of ill-health, and generally by its causation. *Malignant sarcomatous* growths may present some resemblance to *P. hæmorrhagica*. It is well also to remember that the children of the poor sometimes present

extensive petechial eruption as the result of *flea bites*. The spots are uniformly about the size of a pin's head, and all disappear entirely after a few days in better circumstances.

**Treatment.**—In milder cases, rest in bed, tonic medicines, and good simple food will often rapidly effect a cure. Iron, arsenic, and quinine may be given in the usual doses. When the purpura affects the lower extremities chiefly, it often disappears directly the patient takes to bed, and returns if walking about is too hastily resumed. In severe cases also, arsenic may be given; but if hæmorrhage take place from the mucous membranes, astringents must be employed, such as turpentine, acetate of lead, ergotin, or dilute sulphuric acid. Turpentine (10 minims three times a day) is especially recommended for *P. rheumatica*. Intramuscular injections of calcium chloride (1 grain in 100 minims of water) once a day may be of value; also an injection of 10 c.c. serum, preferably human, may be given, and this may be repeated.

In severe cases blood transfusion may be of value, and the spleen has been removed with beneficial results on the supposition that it is destroying the blood platelets (**13**).

## HÆMOPHILIA

Hæmophilia, or the *hæmorrhagic diathesis*, is a disease almost entirely restricted to the male sex, and characterised by a tendency to excessive or uncontrollable bleeding, either spontaneous or traumatic. It is congenital, and very often hereditary, so that the subjects of the disease, often known as "bleeders," are the children of bleeders, and their brothers suffer perhaps from the same malady. The disease is transmitted through the female, who is herself entirely free from it; in this it resembles pseudo-hypertrophic muscular paralysis. Female "bleeders" have occasionally been met with (**10**).

**Pathology.**—The main fact about the disease is the greatly delayed coagulation time of the blood, which may be increased to forty minutes or more. Consequently there is nothing to stop any small hæmorrhage that may occur accidentally. There is a deficiency or complete absence of the precursors of thrombin or fibrin ferment, viz., thrombokinase or pro-thrombin. In contrast to purpura, the platelets in the blood are normal. If a state of anaphylaxis is induced the coagulation time is much shortened, and this has been made use of in treatment. The fatty degeneration of the heart and of the arteries found in some cases is probably the result of secondary anæmia.

**Symptoms.**—These generally appear within the first year of life, though they are sometimes delayed till the seventh or eighth year. In the most severe degree, spontaneous hæmorrhages occur from the nose, the gums, and the mouth, and less commonly from the stomach, the lungs, or the genitalia; they are sometimes preceded by a feeling of fulness. Bleeding from the nose is the most common and also the most fatal. Alarming and even fatal hæmorrhages may also occur after the most trivial operation, such as lancing the gums, vaccination, the extraction of a tooth, incision of an abscess, and the application of leeches, or after accidental wounds or a cut finger. In all these cases there is the greatest possible difficulty in stopping the flow of blood. Besides these losses, hæmorrhage takes place readily under the skin from slight blows, or even spontaneously, producing bruises or blood tumours.

In the intervals between the bleedings the subjects of hæmophilia may appear to be in perfectly good health, but the enormous quantity of blood which is sometimes lost may cause a high degree of anæmia, which lasts for many months. Hæmorrhage also takes place into the synovial cavity of the joints, especially the knee joint; this occurs most commonly between the ages of seven and fourteen, and results from blows, or from exposure to cold or to damp. The swelling and pain closely resemble those of rheumatism or synovitis, for which, indeed, the

symptom has been mistaken. This condition of the joint is accompanied with fever; it may recover, but returns again and again. Eventually the joint may become ankylosed or fixed by periarticular adhesions. A rheumatic affection of the muscles and the occurrence of trigeminal neuralgia are described as occasional complications of hæmophilia.

Three degrees of the disease may be recognised: one in which there is a tendency to every kind of hæmorrhage, spontaneous or traumatic, interstitial or superficial; a second, in which spontaneous hæmorrhages from the mucous membranes only are present; and a third, which shows itself only by spontaneous ecchymoses, and which occurs amongst the women of bleeder families.

Subjects of hæmophilia often die from loss of blood before they are eight years of age; and though the chances of survival are greater after this period, even in middle age death may occur in the same way.

**Diagnosis.**—It must be remembered that women sometimes suffer after puberty from a hæmorrhagic tendency, shown by ready bruising, menorrhagia, etc., who have never been bleeders in early life, are not descended from bleeder families, and do not transmit the tendency to their offspring. In the absence of exact information as to the nature of true congenital hæmophilia, the relation to it of these cases must remain doubtful.

**Treatment.**—Patients who are the subjects of hæmophilia should live on a simple, unstimulating diet, and should pay particular attention to the bowels, any tendency to constipation being met by occasional laxatives. It is most important always to bear in mind the liability to bleed from any breach of surface; and the extraction of teeth and all operations, large or small, should be avoided, unless absolutely necessary. For a surface hæmorrhage the best treatment is to apply some fresh human serum or blood clot to the bleeding spot, but fresh animal tissue may also be used. This presumably supplies the missing thrombin. In other cases, where the bleeding is inaccessible, the serum of the horse, rabbit or, preferably, man has been injected subcutaneously or intramuscularly in doses of 10 or 20 c.c. or more. This treatment is uncertain, but has usually been most successful when urticaria supervenes, or there is other evidence that some degree of anaphylaxis is present. Under these circumstances it has been shown that the coagulation time is much diminished and may become normal for a time. The same is the case after blood transfusion, and it has, in fact, been stated that every hæmophilic should be tested against five or six possible donors, so that one of them at least would always be available in a sudden emergency. It is not necessary to use much blood. In case an operation becomes necessary, a state of active anaphylaxis should be produced in the patient by injecting 10 c.c. horse serum subcutaneously, ten days previously, if he has not had serum before. The coagulation time should be tested and the presence of anaphylaxis is indicated by the *local* reaction following an intradermal injection of 1 minim of horse serum.

An allied method which is reported to have brought about most satisfactory results is to produce a condition of passive anaphylaxis in the patient. Rabbits were injected with many minute doses of horse serum, so that anaphylaxis was produced in them. The rabbits' serum was then injected into the patient.

For the stiff joints hot-air baths and gentle massage may be employed: breaking down the adhesions under an anæsthetic is generally avoided, from fear of starting a fresh hæmorrhage, but it has been done without this accident following. Considering the serious nature of this disease and the manner in which transmission takes place through the female sex, it is clear that women who belong to bleeder families, even though themselves not the subjects of hæmophilia, should not marry.

## BLOOD TRANSFUSION

By this term is meant the taking of blood from a healthy person (" the donor ") and injecting it into the circulation of a patient (" the recipient ") for therapeutic

reasons. The indications for this treatment in medicine are—(1) simple hæmorrhage, *e.g.* in gastric and duodenal ulcer, dysentery, typhoid, ectopic gestation, melæna neonatorum; (2) diseases of the blood, *e.g.* severe purpura, hæmophilia, anæmia, leukæmia; (3) severe infections, *e.g.* infective endocarditis; (4) possibly certain intoxications, such as threatening uræmia.

Methods which necessitate cutting down on the artery or vein of the donor are rather undesirable, owing to the slight, but definite, risk to the donor from sepsis. The donor runs no risk if the blood is taken through a wide, hollow needle plunged directly into the vein. In the "citrate method" the blood is withdrawn from the vein into a warm, sterile bottle by suction, and is mixed in the collar of the needle with a 3 per cent. sterile sodium citrate solution, running slowly from a burette and being delivered into the needle through a side tube. The whole apparatus and tubing must be washed through with citrate first of all. A volume of blood up to 600 c.c. or 1 pint may be taken, and 100 c.c. of the citrate solution are added, so that 0·5 gram of citrate is added to each 100 c.c. of blood. If the donor becomes faint or pale or sweats, or if the pulse falls below sixty, the withdrawal of blood must be stopped. The citrated blood is then passed through another needle directly into the vein of the recipient, under slight positive pressure. Recently more favourable results have been obtained, especially in infections, by the use of defibrinated blood, which appears to retain its antitoxic or antibacterial properties to a greater extent than citrated blood. Instead of adding citrate the blood is agitated as it flows into the bottle and the fibrin becomes deposited on a bent glass tube which passes down from the stopper to the bottom of the bottle, and up again. Before injecting the blood it must be filtered through sterile gauze to remove small tags of fibrin. In "immuno-transfusion" the donor is immunised beforehand, or the blood is immunised *in vitro*.

Certain precautions must be taken. The Wassermann reaction of the donor should be investigated, and the "compatibility" of the bloods of donor and recipient must be determined.

*Compatibility.*—It is sufficient if the donor's cells are not agglutinated by the recipient's serum. The reverse condition need not hold, because the donor's serum rapidly becomes diluted in the circulation of the recipient. To test compatibility a drop of blood from the donor's finger is allowed to fall into 1 c.c. of 1·5 per cent. sodium citrate solution, and one drop of the resulting suspension is mixed on a microscope slide with one drop of the recipient's serum and covered with a cover slip. After the lapse of a few minutes the slide is examined macroscopically for agglutination. Individuals are arranged in four groups, thus:—

| | | Serum | | | |
|---|---|---|---|---|---|
| | | Gr. I. | Gr. II. | Gr. III. | Gr. IV. |
| Cells | Gr. I. | — | + | + | + |
| | Gr. II. | — | — | + | + |
| | Gr. III. | — | + | — | + |
| | Gr. IV. | — | — | — | — |

It will thus be seen that, if the sera of groups II. and III. are kept in stock, the cells of any given individual can be assigned to their group. The cells of group IV. are agglutinated by no one, and, therefore, the members of group IV. can always act as donors, but it is generally safer to have both donor and recipient of the same group.

## REACTION OF THE BLOOD AND ACIDOSIS

The reaction of any solution depends on its concentration of hydrogen ions ($C_H$) and hydroxyl ions ($C_{OH}$). When the reaction is neutral, the concentration of hydrogen and hydroxyl ions is equal; when it is acid, the $C_H$ is increased and the $C_{OH}$ is diminished; when it is alkaline, the $C_H$ is diminished and the $C_{OH}$ increased. The product of the two is always constant. At the neutral point and at body temperature the $C_H$, expressed in grams of hydrogen per litre, is $1{\cdot}83 \times 10^{-7}$, which is the same as $10^{-6{\cdot}74}$. The $C_H$ of the arterial blood is about $10^{-7{\cdot}40}$, which, being smaller than $10^{-6{\cdot}74}$, is slightly on the alkaline side of neutrality. The method usually adopted of expressing these facts is to say that at the neutral point $p_H$ (the logarithm of $C_H$) is 6·74, leaving out the minus sign for the sake of simplicity.

The $C_H$ of blood may be regarded as the resultant of all the acid and basic substances of the blood. Among the acid substances of importance are the $CO_2$ dissolved, the $CO_2$ combined with bases as bicarbonate, oxyhæmoglobin and various fixed or non-volatile acids which are produced in metabolism. Acid is excreted by the kidney in two ways: (1) the urine is made more acid than the blood by the predominance of acid over basic phosphate; (2) ammonia is formed, and this combines with the acids to form neutral salts, which are excreted. The ammonia of the urine is thus increased.

At times the production of fixed acids is greatly increased, or abnormal acids may appear, such as β-oxybutyric and aceto-acetic acids. If these extra amounts were accommodated in the blood, the $C_H$ would rise; but this is prevented by a corresponding diminution in the acid $CO_2$. This means that both the $CO_2$ dissolved and the $CO_2$ combined as bicarbonate in the blood are diminished. This is expressed by the equation

$$C_H = \text{constant} \times \frac{CO_2 \text{ pressure}}{\text{bicarbonate concentration}},$$

the $CO_2$ pressure being proportional to the $CO_2$ dissolved in the blood. In normal people the $CO_2$ pressure of the arterial blood is the same as the $CO_2$ pressure in the alveoli of the lungs. It will be noticed from the equation that, if the $CO_2$ pressure is not diminished in proportion to the bicarbonate, the $C_H$ will be increased; in other words, the blood will be abnormally acid. This is known as *acidæmia*. The term *alkalæmia* is used for the opposite condition, when the $C_H$ is abnormally diminished. This occurs to a slight extent at high altitudes, and after taking large doses of alkali, and after washing out $CO_2$ from the body by forced breathing (acapnia), and it is sometimes associated with tetany (*see* p. 585).

Besides a knowledge of the $C_H$ of the blood in pathological conditions, it is important to be able to measure the amount of the fixed acids in the blood. For this purpose the amount of $CO_2$ in the form of bicarbonate in the blood is measured at a fixed pressure of $CO_2$, viz., 40 mm. This is defined as the *alkali reserve* of the blood, the *fixed* $CO_2$, or the *blood bicarbonate*. The normal value for this lies between 40 and 54 c.c. per 100 c.c. of blood. When the fixed acids are increased the alkali reserve is diminished.

The term *acidosis* was first of all introduced to indicate the production of aceto-acetic and β-oxybutyric acids in the body and their excretion in the urine in diabetes. Since these bodies circulated in the blood before excretion, the term was used to describe their presence in the blood.

More lately *acidosis* has been used as equivalent to diminution in alkali reserve, in whatever way this has been brought about. Other authors, however, have used the term to mean increase in the $C_H$ of the blood, a totally different thing. Owing to this confusion in terminology, it is best to use the term *acidosis* to indicate the process of acid production in the body and *alkalosis* to indicate the process of alkali production.

The foregoing remarks are important when the cause of certain common types of dyspnœa is considered. In emphysema, asthma and bronchitis, and bronchial obstructions due to growths, when these conditions are associated with dyspnœa, there is marked acidæmia, owing to accumulation of $CO_2$ in the blood. The amount of fixed acid in the blood remains about normal. In such cases, owing to the condition of the lungs, the $CO_2$ fails to escape in spite of the increased breathing. Want of oxygen may also play a part in the dyspnœa of some of these cases.

In the dyspnœa of mitral disease, the $CO_2$ is lowered from increased respiration and there is alkalæmia. The alkali reserve is normal, provided there is no very extensive generalised œdema. In the latter case it may be diminished.

Acidæmia may be present in three other conditions of clinical importance: ketosis, chronic or "asthenic" uræmia, and eclampsia. Here the acidæmia is associated with a lowered alkali reserve and a low $CO_2$ pressure. In ketosis the accumulation of aceto-acetic and $\beta$-oxybutyric acids in the blood is the primary cause of the acidæmia. They stimulate the respiratory centre, so that the respiration is increased and $CO_2$ is washed out of the blood, with lowering of the alkali reserve; but, to begin with, the $CO_2$ is diminished almost in proportion, so that the $C_H$ does not rise very much. It is only in the later stages, towards the onset of coma, that a measurable degree of acidæmia may occur. Ketosis will be considered further in the section on diabetes. The acidæmia of asthenic uræmia and eclampsia is also probably due to accumulation of fixed acids, as in ketosis, but the nature of these acids is unknown. In normal pregnancy and in secondary contracted kidney long before uræmia supervenes, there is a measurable lowering of the alkali reserve. In polycythæmia and in gas gangrene the alkali reserve is also lowered, and probably also in various febrile conditions.

In health the various mechanisms for combating acidæmia in the body succeed in keeping the $C_H$ of the blood and body fluids within comparatively narrow limits. In the later stages of ketosis and in uræmia a measurable increase in the $C_H$ of the arterial blood takes place. In a case of uræmic coma the $p_H$ was 7·27 shortly before death. In a case of emphysema with $CO_2$ retention the $p_H$ was 7·24 some weeks before death, when the patient was walking about perfectly conscious. It is thus very doubtful whether the acidæmia by itself is the cause of uræmic coma, since the blood of the emphysematous patient was more acid. Coma is more probably due to the poisonous character of the acid substances retained in the blood. The same theory may apply in ketosis, for in this condition there is good evidence from the structure of its molecule that aceto-acetic acid is itself a poison (Hurtley and Trevan).

**Diagnosis.**—Breathlessness is a valuable indication of acidæmia; but this may be due to (*a*) want of oxygen, (*b*) reflex action, as is possibly the case in primary heart disease, where the respiratory pump helps the circulation, (*c*) local irritation of the respiratory centre, (*d*) acidæmia, increase of $CO_2$ or fixed acid in the blood. (*a*) The patient will probably show lividity or cyanosis. (*b*) The respiration is rapid, and there will be evidence of heart disease. (*c*) There will be evidence of intra-cranial disease, such as cerebral hæmorrhage, and the patient will probably be unconscious. (*d*) Where there is no primary lung lesion, which causes acidæmia by increase of $CO_2$, the increased respiration will probably be due to increase of fixed acid in the blood. The respirations are often slow and deep. The most accurate measure of the amount of fixed acid is to determine the alkali reserve directly, but this is hardly a clinical method. Three other methods may be used: (1) The ratio of the ammonia nitrogen to the total nitrogen in a specimen of urine may be estimated. Normally this is 3 to 5 per cent. In severe cases of acidæmia values of 20 to 40 per cent. may be obtained. (2) The alveolar $CO_2$ may be measured by some apparatus, *e.g.* Fridericia's $CO_2$ tensimeter, a determination taking about ten minutes. (3) The amount of sodium bicarbonate which is required, when given by mouth, to make the urine alkaline to litmus, may be determined. Five grammes are enough for this in normal

people. When the alkali reserve is diminished a larger amount will be necessary, and gradually increasing doses at three or four hourly intervals may be given to test this (Sellards).

It is very necessary to make a sharp distinction between ketosis where the nature of the fixed acids is known and other conditions of lowered alkali reserve and acidæmia. Ketosis can readily be diagnosed by the mahogany brown colour of the urine when ferric chloride is added to it and the purple colour with Rothera's or Legal's tests, and by the smell of acetone in the breath. The presence of ketosis is not always due to lack of available carbohydrate in the body, tending to acidæmia (*see* p. 568). It is produced by giving large doses of sodium bicarbonate, which tends to alkalæmia. In this case these acids are probably formed as part of a compensating mechanism to combat the alkalosis.

**Treatment.**—It has already been stated that it is doubtful how far acidæmia itself is a cause of death; but when it is present accompanied by a lowered alkali reserve, it is reasonable, in the present state of our knowledge, to counteract it. For this purpose sodium bicarbonate or sodium citrate may be given in drachm doses by the mouth every two hours. In acute cases an intravenous injection of 2 per cent. sterilised sodium bicarbonate may be given.

Where it is believed that the clinical condition is due to poisons in the blood the formation of the latter must be arrested (*see* under Ketosis), and their excretion must be facilitated. The latter will be helped (1) by giving sodium bicarbonate; (2) by intravenous injections so as to keep up the blood pressure and the flow through the kidneys; (3) by producing watery evacuations or washing out the colon; (4) most important of all, by giving copious draughts of water by the mouth, if necessary by means of a nasal tube.

# DISEASES OF THE SPLEEN

The spleen lies in the upper part of the abdomen on the left side, and is entirely concealed by the ribs. In health its position and size can only be estimated by percussion. There is dulness in the left infra-axillary region over the ninth, tenth, and eleventh ribs, and the included spaces. In front, this dulness is limited by a line drawn from the left nipple to the tip of the eleventh rib; behind, it reaches nearly to a line continuous with the anterior margin of the latissimus dorsi. If the spleen becomes enlarged, it extends downwards and forwards, the dulness passes in front of the line above mentioned, and if the fingers be placed under the ninth and tenth costal cartilages while the patient takes a deep breath, the margin of the spleen can be felt to impinge against them. With greater enlargement, it comes distinctly below the ribs at this point, so that it can be readily felt, and occupies more or less of the left upper quarter of the abdomen. In extreme cases of leukæmia the spleen reaches down to Poupart's ligament and crosses the middle line below the umbilicus, though it may remain on the left side above. An enlarged spleen is always dull to percussion, is continuous with the lower ribs, and is never overlaid by bowel or stomach. It descends along, and clings to, the abdominal walls throughout. The anterior margin is irregular, and presents one or two distinct notches.

The majority of the disorders of the spleen are secondary to other lesions elsewhere, and frequent reference has been made to its implication in different forms of fever, in malaria, in Kala-azar, in diseases of the blood, and in malignant endocarditis. Unless the enlargement is very considerable (leukæmia, splenic anæmia, and hydatid) the lesion of the spleen is not generally a source of much trouble to the patient. Pain may be present from the formation of infarcts, and from the resulting perisplenitis or abscess, but is not a marked feature in the enlargements which accompany fevers. If the organ attains a great size, as in

cases of spleno-medullary leukæmia, there may be a sense of weight, or dragging, on the left side.

An important function of the spleen is the destruction of worn-out red blood corpuscles; but it has been recently discovered that the spleen also acts as a regulator of the blood volume, turning out its corpuscles into the circulation as occasion requires. Thus it may shrink to one-fourth or one-sixth of its normal size during hæmorrhage, or during exercise or exposure to heat, when extra blood is required in the skin for cooling purposes or during asphyxia (**11**). In disease the size of the spleen is very variable, probably owing to this regulating function.

The pathological changes to which the spleen is liable will now be summarised.

**Active Congestion.**—The spleen is enlarged in many acute infectious processes, and this is most prominently the case in enteric fever, in relapsing fever, in ague and other malarial fevers, in pneumonia, pyæmia, malignant endocarditis, phthisis, and acute tuberculosis, and less so in puerperal fever, erysipelas, and syphilis. The capillaries and veins are distended with blood. The splenic pulp is swollen, and the capsule of the organ is distended. After death the spleen is found to be of dark red or purple colour, and very soft; and the pulp is readily washed away by a current of water. The histological changes resulting from infective processes in the spleen are, according to Muir, as follows: great phagocytic activity of the cells of the pulp, especially non-granular hyaline cells and endothelial cells, which may be seen to contain numerous red cells, and neutrophil leucocytes; the presence of myelocytes in the pulp; apparent enlargement of the Malpighian corpuscles due to proliferation of cells around them.

**Splenitis and Perisplenitis.**—In some of these infective conditions the process goes beyond the stage of hyperæmia into one of acute inflammation, as shown, according to Ziegler, by the excessive quantity of white cells found within the vessels and pulp. Abscess is a very rare result of general splenitis. It more often results from the breaking down of infarcts in malignant endocarditis (*see* below). Accompanying the splenitis there may be inflammation of the capsule, *capsulitis*, or *perisplenitis*, with resulting adhesions to adjacent organs, or to the abdominal parietes. Acute or chronic capsulitis is very frequently found at post-mortem examinations; and its occurrence can often be traced, especially in the acute form, to infective processes.

**Chronic Enlargement.**—Splenitis may subside entirely, or go on to hyperplastic changes in the pulp, trabeculæ, vessels or capsule. The *ague cake* already mentioned (*see* p. 154) is an example of this. A moderate enlargement is also seen in rickets, tuberculosis, congenital syphilis, and Hodgkin's disease; a much greater enlargement is often seen in ordinary cirrhosis of the liver, in malignant endocarditis, and in erythræmia; but the greatest size is reached in leukæmia, in splenic anæmia, especially the Gaucher variety, in infantile pseudo-leukæmic anæmia, in splenomegalic cirrhosis, and in Kala-azar, in all of which the organ may occupy a large part of the abdomen. These conditions have been already described.

**Passive Congestion.**—The usual causes of venous congestion of the spleen are mitral valvular disease and cirrhosis of the liver; but in old heart disease the organ is often small and very hard.

**Embolic Infarcts.**—These are the results of the impaction of fibrinous particles, detached from the valves of the heart or from thrombi in its cavities. The infarcts form wedge-shaped or conical masses, which may reach a large size, and occupy one-half or two-thirds of the organ. They go through the changes of colour elsewhere described (*see* p. 379), and in septic cases, such as pyæmia and malignant endocarditis, they become purulent, from the presence of pyogenic bacteria. Infarcts also occur in the spleens of leukæmia and splenic anæmia.

**Lardaceous Degeneration.**—This change is similar to that which is seen in the liver and kidneys. It affects the splenic vessels and the Malpighian follicles, which last appear as grey specks upon the surface (*sago spleen*); in other

cases the lardaceous material is deposited between the cells of the pulp, and the organ is more uniformly pale. The diseased parts are coloured brown-red by the addition of tincture of iodine. The lardaceous spleen is enlarged, hard and smooth; the liver and kidneys are often affected at the same time.

**Tubercle.**—This appears in the spleen as a part of general tuberculosis, in the form of grey or often bright yellow nodules, which may reach the size of small peas, scattered throughout the substance and on the surface. Cases of primary tubercle of the spleen have been recorded. *Syphilitic* gumma is rare; but the spleen is often enlarged in congenital syphilis.

**Parasites.**—Exceptionally a hydatid cyst develops in the spleen and forms a large tumour.

**Diagnosis.**—From the frequency with which the lesion of the spleen is secondary to, or closely associated with, a disorder of some other organ or of the blood, it is obvious that the diagnosis of its disorders requires a careful investigation of the other organs, and especially a complete examination of the blood.

The **Treatment** of splenic lesions is dealt with under the various diseases with which they are associated.

# DISEASES OF THE LYMPHATIC SYSTEM

The majority of the diseases to which the lymphatic system is liable arise as the result of the passage into the lymph vessels of some substance foreign to them, such as micro-organisms, tumour cells, or other solid particles, and the poisons of certain diseases which may ultimately prove to be of microbic nature. These either set up acute inflammation or cause a change in the gland of the same nature as the source from which the foreign substance has come. Thus we see the inguinal lymph glands inflamed in syphilis, the glands of the jaw in diphtheria, the axillary glands in poisoned wounds of the arm, the bronchial glands in pneumonia, and the mesenteric glands in enteric fever. Again, the bronchial glands become tuberculous as the result of phthisis; and the axillary glands are cancerous in consequence of carcinoma of the breast. Inflammation of the lymph glands either goes on to suppuration, or subsides as the primary cause is removed, or becomes a chronic induration. Tubercle and cancer run the same course as they do in other parts. The relation of the lymph glands to leukæmia has been mentioned; Hodgkin's disease, or lymphadenoma, and tuberculosis of the mesenteric glands will be separately described. The inflammation and suppuration of the cervical glands, which is often, but not always, a tuberculous process and probably depends on infection from the tonsils, is generally dealt with in surgical works.

The lymph vessels are inflamed as a result of septic poisons, as, for instance, in *lymphangitis*, or absorbent inflammation, which involves the lymphatics between a wound and the nearest gland. Obstruction of lymph vessels occurs under certain circumstances in tropical climates, especially by the nematode worms, *filariæ*. These conditions will be described under the name *filariasis*.

## HODGKIN'S DISEASE

(*Lymphadenoma*)

Dr. Hodgkin first described, in 1832, a series of cases of enlargement of the lymphatic glands with a peculiar deposit in the spleen; and similar cases were recorded by other writers as progressive multiple hypertrophy of the lymph glands (Wunderlich), multiple malignant lymphoma (Billroth), and *adénie* (Trousseau). The name *lymphadenoma* indicates the nature of the new growths which occur in the disease; but German writers, while often speaking of it as Hodgkin's disease, mostly use the term *pseudo-leukæmia*, from the superficial

resemblance it bears to lymphatic leukæmia. It has also been called *anæmia lymphatica*.

**Ætiology.**—Very little is known of its causation. It occurs at all ages, but is rare after sixty; one-half of the cases occur between the ages of twenty and forty, and one-third from infancy to twenty years. Men are the subjects of it twice as often as women. In a few cases depressing causes, such as intemperance, insufficient food, exposure to cold, and parturition, have been noted. More often, however, no cause whatever can be discovered.

**Anatomical Changes.**—On post-mortem examination, it is found that there are two important changes: one the enlargement of the ordinary lymph glands; the other the growth of tumours having a similar structure in different organs of the body, and in subserous and other tissues. The glands are light grey, or greyish white on section. Microscopic examination shows that the enlargement of the glands consists essentially in the proliferation of endothelial cells and fibroblasts. The presence of small giant cells with one, two or more nuclei is very characteristic. Numerous eosinophil leucocytes are seen in the earlier cases. Lymphocytes are, of course, also present. The glands, which are soft to begin with, become harder from the deposition of fibrous tissue as the disease progresses.

The lymphadenoid growths which are independent of the glands proper occur in the spleen, kidneys, liver, lungs, and other parts. The spleen is generally larger than normal, and may weigh even 30 ounces; it is moderately firm, and presents on section a number of white or yellowish tumours, from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter, scattered through its substance, giving it an appearance which has been compared, not inaptly, to "hard-bake." These tumours arise from the Malpighian corpuscles. Similar tumours occur scattered through the liver or the kidneys, or in the tonsils, and in the follicles of the pharynx, stomach, and intestine. Nodules of growth are also present in the lungs, and soft pinkish-grey, flat masses have been found under the pleura and other serous membranes. In one case under Sir Frederick Taylor's care, growths the size of a pea had formed in pleural adhesions, which stretched across the fluid of a hydrothorax. The epididymis and testicle have been invaded; and the bone marrow is often affected, being converted into a reddish-grey, semi-diffluent matter, or presenting yellow, grey, or white nodules.

**Symptoms.**—The chief clinical features of the disease are enlargement of the lymphatic glands and anæmia. Generally, the *lymphatic enlargement* occurs first, the change beginning in the cervical glands in most cases, and subsequently involving those of the axilla and the groin. The glands form irregular and nodulated masses of different sizes, extending, perhaps, from the clavicle to the jaw, or of such a size in the axilla as to prevent the arm being applied to the side. The individual glands are as large as a pigeon's egg or a hen's egg, are either soft or firm, usually painless, and at first freely movable upon one another under the skin. Subsequently they may become adherent, but rarely caseate or suppurate. Other smaller groups of glands are also involved, such as the occipital; and the change affects the glands in the interior of the body—namely, the bronchial, mediastinal, mesenteric, and retro-peritoneal. In many of these regions the growth of the glands may be such as to cause serious pressure on the neighbouring parts. These are—in the neck, the larynx, trachea, and œsophagus; in the thorax, the large veins and the recurrent nerves; in the abdomen, the nerves of the solar plexus; and in the axillæ and groins, the nerves supplying the limbs.

The *spleen* is, as a rule, only moderately enlarged; it projects a little below the left costal margin, or occupies the left upper quarter of the abdomen; it rarely attains the same size as that seen in myelocytic leukæmia.

*Anæmia* is a prominent symptom, even comparatively early, in Hodgkin's disease. The red corpuscles are diminished to 60, 50, or 40 per cent. of the normal; the hæmoglobin is diminished to a still greater extent (chlorotic type); and in severe cases poikilocytes and nucleated red cells (normoblasts) are seen.

The leucocytes are generally not increased in number, but when the glands are soft (or inflamed) they may be as many as 15,000 or 20,000 per cubic millimetre. Of these the polymorphonuclears form about 70 per cent.; lymphocytes are not numerous, but eosinophils are sometimes in excess, both relatively and absolutely. If, as may happen, a pronounced lymphocytosis develops, the case must be regarded as a lymphocytic leukæmia (*see* p. 535).

*Pigmentation of the skin* sometimes occurs, and has been ascribed to pressure on the solar plexus by enlarged glands; occasionally there is intense *pruritus*, with the formation of papules, vesicles, or bullæ.

*Pyrexia.*—Some cases of lymphadenoma show a relapsing pyrexia (*see* Fig. 54), and in this feature the disease resembles relapsing fever, malaria, trench fever, and rat-bite fever, because the periodicity is regular. There is a gradual ascent or descent of the temperature in steps from day to day, and there is apparent complete recovery in the apyrexial periods. It differs, however, in the greater length of the span, which in 85 per cent. of cases lies between fifteen and twenty-five days, and is fairly constant for each individual case (**14**). Some of these cases have been called *Pel-Ebstein's disease*, or *recurrent pyrexia*. There may also be continuous pyrexia.

A certain amount of weakness is soon observed, and as the disease progresses

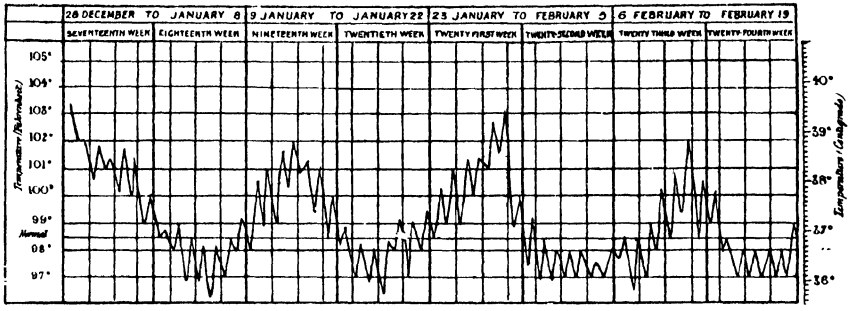



FIG. 54.—Chronic Relapsing Pyrexia in Hodgkin's Disease.

the effects of the anæmia become more pronounced. There is generally a good deal of dyspnœa, partly from the anæmia, and partly from mechanical interference with the trachea, bronchial tubes, or lungs. In time, also, œdema of the lower extremities takes place, with, perhaps, ascites, pericardial effusion or hydrothorax; and hæmorrhage from the nose or gums, or under the skin, may occur as in other severe blood diseases. Finally, death is caused by exhaustion, suffocation, or starvation from the pressure of enlarged glands, by hæmorrhage, by cerebral disturbance, coma or convulsions, or by pneumonia, pleurisy, or œdema of the lungs. The duration is generally several months, or one or two years; occasionally the cases are much more rapid.

**Diagnosis.**—It may be difficult to distinguish the early stage of Hodgkin's disease of the glands from tuberculous enlargement, especially when the growth is confined to one set of glands. However, tuberculous glands tend to be matted together; in Hodgkin's disease the glands are usually discrete. Tubercle usually affects one group of glands; in Hodgkin's disease the changes are eventually widespread. Malignant growth in the glands may resemble Hodgkin's disease. A general glandular enlargement in *syphilis* can usually be distinguished by the history and by the influence of drugs. If a leucocytosis develops, its extent and the characters of the differential count will require careful consideration (*see* Leukæmia). A relapsing temperature of the kind described is a strong point in favour of Hodgkin's disease.

**Treatment.**—Arsenic has been of great benefit in some cases; it should

be given in increasing doses until as much as 15 minims of the liquor arsenicalis three times daily is being taken. The more usual tonics—iron, cod-liver oil, quinine, and others—have no influence. In cases where the enlargement has been confined for some time to one set of glands, their excision has sometimes appeared to postpone the end, but in most cases the disease has spread in spite of it. Great improvement has been recorded in a few instances from exposure to the Röntgen rays, which appear to act prejudicially upon lymphoid tissues; but relapses are almost certain. And, in fact, few cases of complete recovery are known to have occurred. Deep X-ray therapy is an improved method of treatment.

## FILARIASIS

Several species of the nematode worms Filariæ and allied genera occur as parasites in the human body, infesting the connective tissues and lymphatics. They occur extensively in tropical and sub-tropical countries, especially in West Africa, India, the West Indies, and the South Pacific islands. The most important is the *Filaria bancrofti*, of which the embryo is conveniently known as microfilaria nocturna on account of its habit of appearing in the blood stream by night-time only. Filarial infection is usually accompanied by a marked increase of the eosinophil cells in the blood.

**Life History of Filaria bancrofti.**—The adult female is filiform, from 3 to 3½ inches long, and $\frac{1}{100}$ to $\frac{1}{90}$ inch in thickness; the male is much smaller and has a corkscrew-like tail. They are usually found in dilated lymphatic vessels and lymphatic glands, and there the female discharges an innumerable progeny of embryos, which are sufficiently minute to pass through the lymphatic channels into the thoracic duct, and thence into the blood vessels, where they are found during the life of the host in extraordinary numbers. These embryos (microfilariæ) are also filiform, about $\frac{1}{90}$ inch long and $\frac{1}{3200}$ inch broad; they are contained in a sort of sheath in which they elongate and shorten themselves, and which commonly projects for some little distance beyond the caudal extremity. This covering is the chorionic envelope of the egg which has been stretched by the growth of the embryo. The embryos are extremely active, twisting and wriggling amongst the blood corpuscles as they are seen on the slide of the microscope.

The microfilaria nocturna can only be found in the peripheral blood vessels during the night, from 6 or 7 p.m. to 8 or 9 a.m., whereas during the day-time it occurs in small numbers or is entirely absent. It is conjectured that these embryos must during the day-time occupy the blood of the deeper vessels; and it has been shown that this diurnal variation depends on the habit of the host with regard to waking and sleeping, and not upon light and darkness. In the tropics, the presence of the embryos in the cutaneous vessels at night-time is of importance to their future development, as, like some other entozoa, they require an "intermediate host" in order that the embryo may complete its development outside the human body. Certain species of mosquito act as intermediate hosts, and, on sucking the blood of the human victim, draw up the microfilariæ into the stomach. Within a few hours of their ingestion the embryos escape from their sheaths, and pass from the stomach of the mosquito to the thoracic muscles; and here they both develop their organs and grow to a considerable size ($\frac{1}{16}$ inch); they then migrate to the proboscis of the mosquito, and pass again into their human host, when the mosquito next proceeds to bite its victim. Ultimately the filariæ establish themselves in the lymphatic vessels of their host, become sexually mature, and produce the embryos which are found in the blood.

In most tropical countries the mosquito intermediary is the brown gnat *Culex fatigans*, but in the Pacific Islands it is quite a different kind—*Aedes variegatus*—and wherever this latter insect occurs, there the filarial embryos have a

different habit altogether and appear in the blood stream in large numbers both by day and by night, thus differing entirely from the variety of *F. bancrofti* found elsewhere. No explanation of this peculiarity has so far been forthcoming.

**Pathological Relations.**– The microfilariæ may be found in the blood of perfectly healthy persons, and no symptoms appear to attach to their presence therein. However, it is believed that these people may ultimately develop some signs of lymph stasis. They may be readily seen with a low power of the microscope. A film of the blood may be spread on a microscope slide, and examined at once or allowed to dry, dehæmoglobinised and stained. About 20 c.c. of blood are necessary.

It is the presence of the adult parasites in the larger lymphatic vessels which gives rise to definite pathological conditions; there is narrowing of the lumen of the containing vessel and fibrosis of the wall, which tends to cause lymph stasis. The narrowing also prevents the free passage of the microfilariæ into the blood stream, so that they are held up and die in the lymph glands, causing fibrosis, which tends still further to increase the stasis. After the death of the parasite its body becomes cretified, but secondary streptococcal lymphangitis also occurs, and often causes the death of the parent worm; but by the time this happens permanent interference with the lymph flow has been caused.

Thus are produced obstruction of the thoracic duct or other lymph vessels and in consequence varicosity of more remote vessels and the solid œdematous condition of which *elephantiasis* is probably an example. The obstruction to which the thoracic duct may be subject was well shown in a case recorded long ago by S. Mackenzie. He found a large mass of dilated lymph sinuses and glands, extending from the bifurcation of the aorta to the diaphragm, and occupying all the space between the two kidneys. The lower part of the thoracic duct was sinuous and pouched; and above the diaphragm it became impervious, and was lost in the tough, dense material, apparently of inflammatory origin. If the thoracic duct is obstructed, the chyle vessels and lymph vessels below it become dilated and varicose; and the chyle, unable to pass by its usual course, will go along new channels, and thus come into relation with such parts as the walls of the bladder, the tunica vaginalis, or the pleura. By the rupture of dilated lymph vessels in connection with these parts chyle or a chylous fluid is extravasated into the hollow cavities, and thus is explained the occurrence of *chyluria, chylous hydrocele* or *chylocele, chylous pleurisy* or *chylothorax* (*see* p. 279), and *chylous ascites* (*see* p. 478).

Subcutaneous and intramuscular *abscesses* are extraordinarily common in filarial subjects. Should they occur in the thorax or abdomen, the result may be fatal, and a form of fatal peritonitis is now recognised. Septicæmia is quite common. Enormous glandular enlargement frequently occurs, forming when in the vaginal region what are known as *varicose groin glands*. Filarial *orchitis*, endemic *funiculitis* and *hydrocele* result when the filaria worms infest the genital tract in the male. Finally, the joints may be affected, and filarial synovitis results, which, when affecting the hip joint, may result in ankylosis and resemble tuberculous disease.

The connection of elephantiasis with *Filaria bancrofti* is indicated by the fact that the geographical distribution of both is the same, and that elephantiasis is most prevalent in places where microfilariæ can be found most commonly in the blood of the population. In many cases of elephantiasis microfilariæ are not found in the blood, but this may be due to their not being able to get there owing to obstruction or to the previous death of the adult worms.

Some of the more important results of filarial obstruction of the lymphatic vessels will now be described.

### CHYLURIA

The urine is opaque, whitish or milky in appearance, and has an odour of milk. On standing a layer of fat may collect on the surface; and generally

also a soft coagulum forms, which is either transparent or opaque. If it is shaken up with ether and placed under the microscope, the turbidity is seen to be due to minute oil globules and granules. It contains also a small quantity of albumin. Sometimes blood is present, and gives the urine a pink or darker red colour. Careful microscopical examination of the sediment will often detect the embryo filariæ, which have been already described as being present in the blood, and which have obviously escaped with the lymph or chyle into the urinary passages, and it is interesting to note that they were first found in this situation by Demarquey long before they were known to occur in the blood.

Chyluria, when once established, is not constant; it may disappear and again recur. When present, it is generally more marked after a meal.

There is not necessarily any disturbance of the health; the patient may seem perfectly well. On the other hand, there is often some uneasiness or pain in the back, loins or perineum; and it may be febrile symptoms, debility or mental depression. Retention of urine, from blocking of the urethra by fibrinous coagula, is sometimes the first sign. In prolonged cases—and it may last twenty or thirty years—there are emaciation, craving appetite, and severe thirst. The disease occurs at all ages, and equally in males and in females.

**Diagnosis.**—Chyluria is readily recognised, and if it occur in persons from the tropics, the microfilariæ may be looked for in the blood and the urine But chyluria and the other lymphatic lesions are occasionally seen in persons who have never resided abroad, and may be exceptionally due to obstruction or rupture of lymphatic vessels produced by malignant growths. For instance, Whitla records a case of chylous ascites from invasion of the thoracic duct by tubercle.

**Treatment.**—When once established, little can be done beyond supporting the patient's strength as much as possible, so as to meet the drain of fat, fibrin, and albumin through the urine.

Manson relies on rest in bed, elevation of the pelvis, restriction of food and drink, and gentle purgation, for at least temporary relief.

### LYMPH SCROTUM

Numerous clear vesicles appear on the scrotum, which is enlarged, soft, and spongy; the vesicles may give way and discharge a fluid, which is obviously lymph. The inguinal glands are enlarged, and there is a liability to the erysipelatoid attacks which occur in elephantiasis. Embryo filariæ may be found both in the blood and in the lymph from the vesicles.

### ELEPHANTIASIS

The legs and the scrotum are the parts most commonly affected. It is not necessarily symmetrical; it may affect one leg alone (*Barbados* leg), or the leg below the knee only, or the scrotum or vulva, ears, lips, arm, mamma, or the lower part of the abdominal wall. If the leg is affected, it becomes enlarged to two or three times its natural size. The skin looks œdematous, but it does not pit on pressure; it is obviously greatly thickened. Where folds naturally occur, as about the knee or ankle, there are deep sulci, and these may be moist from retained sweat or sebum. Pigment is increased in the limbs; the surface becomes rough and scaly; here and there are patches of hypertrophied papillæ, and, in other parts, vesicular or moniliform prominences due to dilated and varicose lymphatic vessels. Sometimes these burst and discharge a more or less turbid lymph. The scrotum has sometimes grown to a tumour weighing more than 100 lb. In general the change affects the subcutaneous layer, which is enormously thickened by the growth of new connective tissue.

In tropical countries the disease often begins by attacks of lymphangitis with swelling and redness, like erysipelas, accompanied by fever (*elephantoid*

*fever*), after the subsidence of which the leg is left bigger than normal. A fresh attack of fever and local inflammation after some months again leaves the leg worse than it was after the first, and the evil goes on increasing. Sometimes, however, the enlargement is gradual, and not at any time associated with febrile attacks. The lymphangitis and inflammatory reaction are now believed to be due to secondary streptococcal invasion.

In England erysipelas and " white leg " in pregnancy are occasionally causes of a permanent elephantoid change, and a septic lymphangitis can bring about the same result. Local pressure on veins and lymphatics may cause it, and it may occur as a part of extreme obesity.

**Treatment.**—Very little can be done, except surgically; thus a large scrotum can be removed. But in any other part of the body such a measure is too serious. Bandaging, especially with rubber bandages, and elevation of the limb, may give temporary relief. Implantation of the lymphatic trunks into the artery has been tried, and failed.

### OTHER FORMS OF FILARIASIS

*Filaria loa* (*Loa loa*), about 2 inches in length and $\frac{1}{70}$ inch in thickness, is found under the skin of the finger, neck, breast and eyelids, and is the cause of the tumours known as *Calabar swellings*, painless swellings half the size of a goose's egg, occurring in different parts of the body and lasting but a few days. The embryos, microfilaria diurna, are found in the blood in the day-time, and disappear at night. Their intermediate host is a fly of the genus *Chrysops*, which bites only by day. Another nematode is *Filaria* (*Acanthocheilonema*) *perstans*, of which the larval form, microfilaria perstans, is found in the blood both day and night.

*Dracunculus medinensis* is well known as the *Guinea worm*, which infests the subcutaneous tissues of the legs in certain parts of India and tropical Africa. This, the female worm, is from 12 to 30 inches in length, and only $\frac{1}{10}$ inch in width. It discharges its embryos into water, and they find an intermediate host in the small crustacean, *Cyclops quadricornis*. It appears probable that it is transferred to man by means of drinking water. The adult worm usually burrows down and presents itself at the ankle or lower part of the leg, where, by extruding its head, it can come into contact with water. A blister forms at the site from which the embryos are ejected. If the parent worm becomes ruptured in the tissues and the embryos escape, sepsis invariably takes place, leading to extensive cellulitis of the tissues.

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# DISEASES OF METABOLISM INCLUDING DISEASES OF THE ENDOCRINE ORGANS

BESIDES the racemose secreting glands provided with ducts and the glands or organs connected with blood formation (spleen, lymph glands and bone marrow), there are certain bodies with special histological structure the diseases of which are accompanied by very striking changes in the metabolism of the body. The evidence derived from these diseases and from experiments points to the conclusion that these organs elaborate and pour into the blood a substance or substances, forming an *internal secretion;* hence they have been named *endocrine* glands or organs (ἔνδον, within; κρίνω, to separate). These internal secretions have an important influence upon metabolism, supplying something which is necessary to the economy, or neutralising or destroying other bodies which are harmful, or conveying substances, called hormones (ὁρμάω, I rouse), which excite the secretion of other glands or the function of other parts.

The endocrine organs are the thyroid and parathyroid glands, the islands of Langerhans in the pancreas, the suprarenal capsules, or adrenals, the pituitary gland, or hypophysis cerebri, the thymus, the pineal gland, or epiphysis cerebri, the carotid and coccygeal bodies, and the *gonads*, as the testes and ovaries are now called in relation to this subject. Several diseases formerly quite obscure are recognised as being the results of excessive, deficient or disturbed functions in these glands.

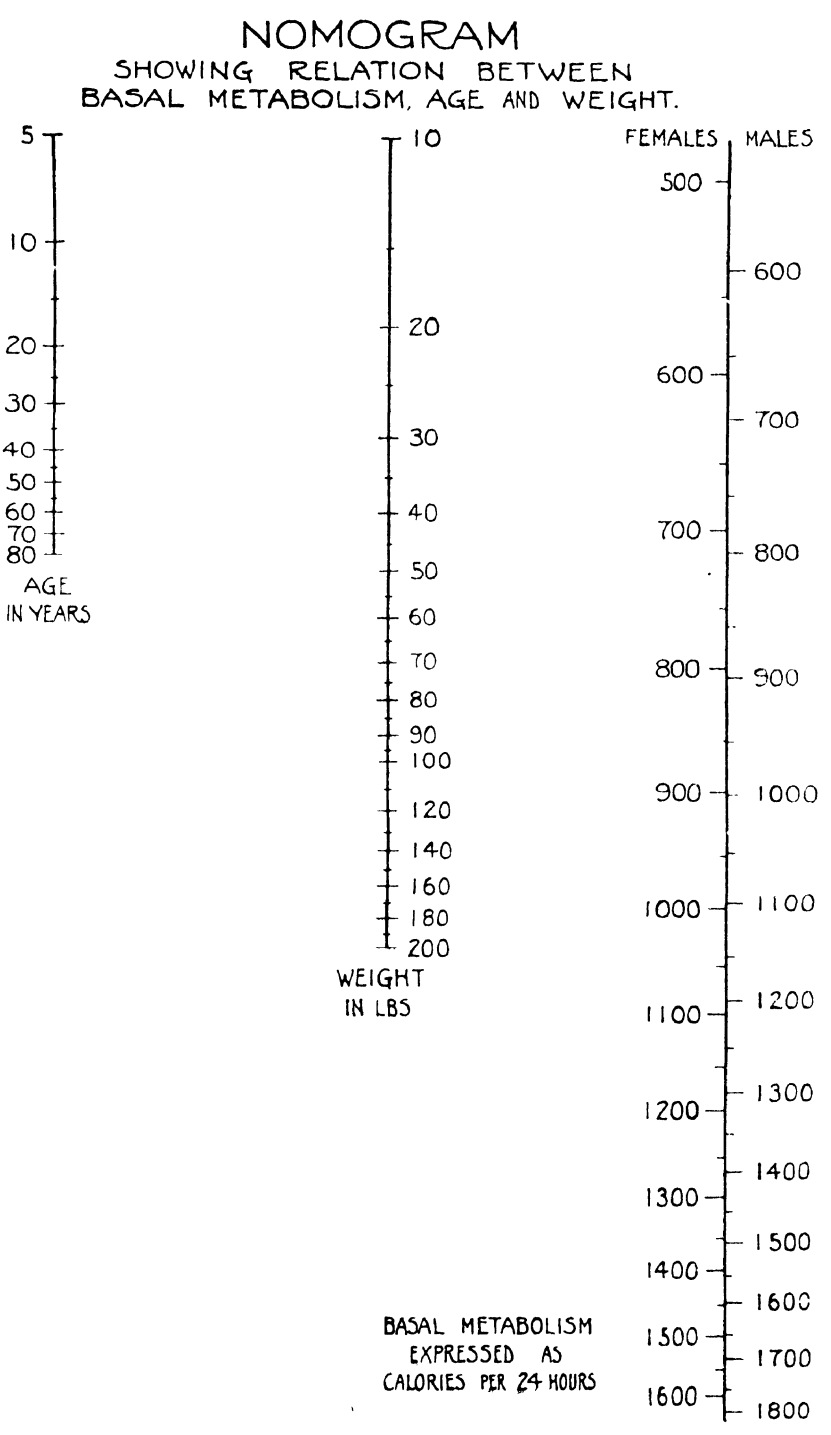
Further, cases of disease occur in which several of the endocrine glands appear to be simultaneously involved, for instance the hypophysis, thyroid, adrenals and ovary in one case; in another the hypophysis, adrenals, testes and thymus; in another the hypophysis, adrenals, ovaries, and pineal body. These may be called *pluriglandular syndromes.*

An important outcome of the recognition of internal secretions is the use of extracts and chemical substances from the same glands in animals to supplement the defect in the patient (*opotherapy*). This has had a conspicuous success in the case of the thyroid gland, and the islands of Langerhans.

## THE BASAL METABOLISM

(*Basal Metabolic Rate*)

This subject is considered here because of its increasing importance in medicine. A description of the various apparatuses used will not be given, but enough will be said to enable the reader to apply the measurements to any individual case. At present the method is principally used in cases of exophthalmic goître (*see* p. 578), both for purposes of diagnosis and as a guide to the progress of a case and the effect of treatment. The basal metabolism means the heat given out per unit of time by the individual under standard conditions, *i.e.* when he is resting quietly on his back without making any muscular movements, at least twelve


NOMOGRAM
SHOWING RELATION BETWEEN
BASAL METABOLISM, AGE AND WEIGHT.
5
10
20
30
40
50
60
70
80
AGE
IN YEARS
10
20
30
40
50
60
70
80
90
100
120
140
160
180
200
WEIGHT
IN LBS
FEMALES
MALES
500
600
600
700
700
800
800
900
900
1000
1000
1100
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1200
1200
1300
1300
1400
1400
1500
1500
1600
1600
1700
1800
BASAL METABOLISM
EXPRESSED AS
CALORIES PER 24 HOURS

FIG. 55.

hours after the last meal. For clinical purposes this value is always determined *indirectly* from the amount of oxygen taken in and the amount of $CO_2$ given out. One method consists in getting the patient to breathe in and out of a closed system; the $CO_2$ is absorbed by soda-lime and the diminution in volume of the gas in the system represents the oxygen taken in (Benedict, Krogh). In the other method, which is often employed in this country, the patient breathes from the atmosphere through a mouthpiece containing inspiratory and expiratory valves into an empty Douglas bag so as to fill it. From the amount and composition of air in the bag the volume of oxygen taken in and $CO_2$ given out can be calculated.

The observation is carried out with the patient at rest in bed in the morning before breakfast, after an early light supper the night before. The output of heat in calories is calculated from the amount of oxygen taken in; but it also varies a little with the $CO_2$ given out, as is shown by the following figures of Zuntz and Schumburg, the assumption being made that the diet contains the usual amount of protein (**10**):—

| Respiratory Quotients. | Calories per 1 litre of Oxygen. |
|---|---|
| 0·72 ... | 4·67 |
| 0·75 ... ... ... | 4·71 |
| 0·80 ... ... ... | 4·77 |
| 0·85 ... ... ... | 4·83 |
| 0·90 ... ... ... | 4·89 |

The respiratory quotient is the ratio of the volume of $CO_2$ given out to the volume of oxygen taken in. From these figures it is possible to calculate the output of heat per hour or per twenty-four hours for the individual. The output of heat actually found must be compared with the average heat production of normal people of the same size. Two methods are available to find this:

1. By the use of Dreyer's formula (**1**) the nomogram in Fig. 55 has been constructed by Dr. W. A. M. Smart, from which it is possible to tell the basal metabolism of either sex according to the weight and age of the individual by connecting the points with straight lines.

However, the nomogram must not be used before considering whether the patient's weight is about the average for his size, because in very fat or very wasted patients too high or too low values for the metabolism would be obtained. Advantage may be taken of Ainley Walker's measurements (**2**) of the body length and body weight to find out what the average weight would be (*see*

TABLE I.—BODY WEIGHT (POUNDS).

| Body Length (inches) | MALE | | | FEMALE | | |
|---|---|---|---|---|---|---|
| | Max | Average | Min | Max | Average | Min |
| 20 | 31 | 25 | 21 | 31 | 25 | 21 |
| 22 | 41 | 34 | 27 | 41 | 34 | 27 |
| 24 | 54 | 44 | 36 | 55 | 45 | 37 |
| 26 | 68 | 56 | 46 | 69 | 57 | 47 |
| 28 | 86 | 70 | 57 | 88 | 72 | 59 |
| 30 | 105 | 86 | 70 | 109 | 90 | 74 |
| 31 | 117 | 94 | 78 | 121 | 98 | 82 |
| 32 | 128 | 104 | 85 | 133 | 109 | 90 |
| 33 | 141 | 114 | 94 | 146 | 119 | 99 |
| 34 | 154 | 125 | 103 | 160 | 131 | 109 |
| 35 | 169 | 136 | 112 | 175 | 142 | 118 |
| 36 | 183 | 148 | 122 | 190 | 155 | 129 |
| 37 | 200 | 161 | 134 | 208 | 169 | 142 |
| 38 | 217 | 174 | 144 | 226 | 183 | 153 |

Max. and min. represent the range of variation for approximately 90 per cent. of normal subjects. It will be noticed how wide this is in the case of larger people (**3**).

Table 1). The patient sits on the floor with his back pressed firmly against the wall and his knees bent. The body length is measured from the floor to the top of the head in inches and compared with the body weight without clothes in pounds. If the patient is too fat or too thin owing to disease, then his normal metabolism would be best obtained by using in Fig. 55 the average weight corresponding to his body length and not his actual weight. Table 1 is of more general use to indicate at any time whether a patient is too fat or too thin; this is of special value in the case of children.

2. Since in healthy people of the same age and sex the output of heat is proportional to the body surface, it is possible to calculate in any individual case

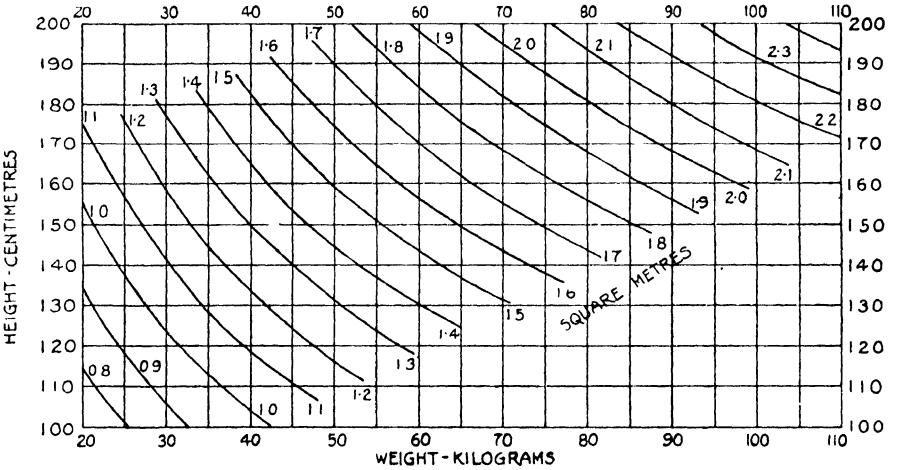


FIG. 56.—Diagram for determining the Body Surface in Square Metres when the Height and Weight are known. (Du Bois.)

the heat output per hour per square metre of body surface. The surface area in square metres can be obtained from the diagram in Fig. 56, after measuring the height in centimetres and the weight in kilograms.

Finally, Table 2 gives the calorie output per hour per square metre of body surface for normal men and women of various ages.

TABLE 2.—STANDARDS OF NORMAL METABOLISM.

*Average Calories per hour per square metre of Body Surface (Du Bois)* (4).

| Age. Years. | Males. Calories. | Females. Calories. |
|---|---|---|
| 14 to 16 | 46·0 | 43·0 |
| 16 „ 18 | 43·0 | 40·0 |
| 18 „ 20 | 41·0 | 38·0 |
| 20 „ 30 | 39·5 | 37·0 |
| 30 „ 40 | 39·5 | 36·5 |
| 40 „ 50 | 38·5 | 36·0 |
| 50 „ 60 | 37·5 | 35·0 |
| 60 „ 70 | 36·5 | 34·0 |
| 70 „ 80 | 35·5 | 33·0 |

Whether Dreyer's or Du Bois' method is used for calculation, the metabolism may be considered normal if the value found does not differ from that calculated by more than 10 per cent.

The basal metabolism is diminished during starvation or in under-nutrition from diminished food supply.

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| 35 | 169 | 136 | 112 | 175 | 142 | 118 |
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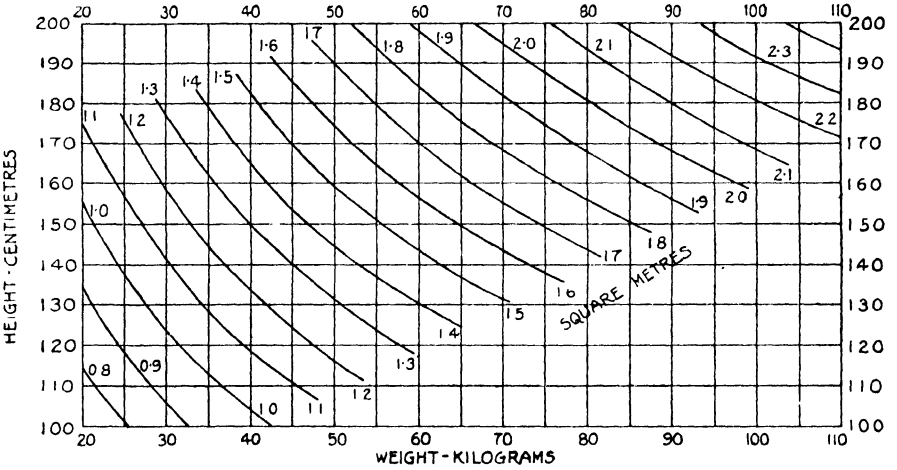


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The basal metabolism is diminished during starvation or in under-nutrition from diminished food supply.

## DIABETES MELLITUS

Diabetes mellitus is a disease characterised by the persistent passage of sugar (glucose or dextrose) in the urine. Defective action on the part of the islands of Langerhans is the cause of the disease, and the regular administration of *insulin* is the appropriate remedy for it. The passage of sugar, or *glycosuria*, is often accompanied by the passage of large quantities of urine or *polyuria*, to which, as the prominent symptom, we owe the name *diabetes* (διαβαίνω, I go through). Polyuria without glycosuria arises in many conditions, and a special form known as diabetes insipidus is described later (*see* p. 595).

**Ætiology.**—In many cases of diabetes mellitus no ætiological factor can be found. There are, however, some definite factors that favour its onset. It is prevalent among the Jews. It may be hereditary, or may occur in brothers and sisters of the same family. Hereditary diabetes may be very mild, but often shows a tendency to become more serious and to begin at an earlier age in successive generations. Diabetes often attacks fat people who eat plenty and take little exercise. Hence it is especially a disease of the well-to-do. This fact may be explained by the increased metabolism that follows excessive eating, which makes greater demands on the pancreas and other organs. The association of diabetes with gout is also probably explained by both diseases occurring in overfed people. In the Central Empires war diet had a profound influence on the disease, causing the glycosuria of fat elderly people to disappear. This fact had been observed by Bouchardat during the siege of Paris. Probably the most important factor was the reduction of the meat ration. There is a very general impression that diabetes is apt to occur in people who eat sugar and sweets to excess, but this lacks statistical proof. The great prevalence of a mild form of diabetes in India is probably associated with obesity, to which the excessive carbohydrate diet and the lack of exercise contribute. A strenuous life, nervous strain and emotional shock may play a pronounced part in the onset of the disease. This is of special interest owing to Cannon's observations that emotion in animals caused hyperglycæmia from excitation of the suprarenals, and that students who were up for examinations, or about to undergo some important physical test, such as playing for their college, often had glycosuria. Graham found in his own case that the blood sugar rose to 0·15 per cent. thirty minutes after taking 100 grammes of dextrose, but that when the same test was carried out after a period of hard work, when he was in need of a holiday, the value was 0·185 per cent., and this persisted for half an hour. True diabetes has followed exophthalmic goitre. Acute infection is a predisposing cause, whether this is general or localised particularly to the neighbourhood of the pancreas, giving rise to a pancreatitis. The diminished sugar tolerance and glycosuria found in septic conditions, sometimes called *sapræmic* glycosuria, must also be mentioned. The glycosuria disappears with relief of the condition. Syphilis is also a possible cause. Diabetes of a mild kind is common in the later decades of life. It is possible that atheroma or senile arterial changes may lead to some defect in the islands of Langerhans, similar to the slight defect in excretory power that often accompanies senile arterio-sclerosis of the kidney (*see* p. 633). In diabetic gangrene both the diabetes and the gangrene may have a primary vascular cause; but the gangrene undoubtedly increases the glycosuria, which often clears up after operation. The reverse also holds, since the gangrene may clear up with insulin. Diabetes has followed trauma, not only over the site of the pancreas, but in places far removed, *e.g.* a fractured limb. Head injuries may cause glycosuria; these are probably analogous to Claude Bernard's "puncture diabetes" (*see* later).

**Physiology of Carbohydrate Metabolism.**—The carbohydrate reserves of the body are stored in the form of *glycogen*, which is about equally divided between the liver on the one hand and the muscles on the other. Glycogen is

formed from the carbohydrates and proteins of the food. The latter are absorbed from the intestine in the form of amino-acids, which may be used to synthesise the proteins proper to the organism, or after deamination may be oxidised at once, or built up into glycogen. Carbohydrates are also broken down by digestion, and may be absorbed in the form of *dextrins* and of comparatively simple substances like *dextrose* and *lævulose*. These bodies all pass to the liver by the portal vein. The dextrins and lævulose are entirely taken up by the liver and formed into glycogen there. If very large amounts of lævulose are eaten some of it manages to get through the liver into the general circulation, and is then promptly excreted by the kidney, producing *lævulosuria*. This condition also occurs in disease of the liver, which under these circumstances is unable to hold back the amount of lævulose, which normally can be dealt with quite readily. Dextrose *may* be partly kept back by the liver, but some of it certainly gets through into the general circulation, because the amount of sugar in the blood of a healthy person shows an increase immediately after a meal (*see* Fig. 57). The systemic blood is thus supplied with dextrose from two sources: (1) the food, which forms a variable supply; (2) the glycogen of the liver, which probably forms a fairly constant supply, the glycogen being broken down by an amylolytic ferment there. Before breakfast the concentration of dextrose in the blood usually lies between about 0·08 and 0·12 per cent. This constancy is preserved by means of a steady production of dextrose from the liver on the one hand, and its disappearance into the tissues on the other hand, where it is either oxidised to form $CO_2$ and water, or elaborated into more complicated compounds, one of these being the glycogen of the muscles.

*Insulin*, which is the hormone secreted by the islands of Langerhans, plays an essential part in these processes, though its action is complicated. It is best regarded from two points of view: (1) In the *periphery* insulin causes the disappearance of dextrose from the blood stream, since if administered to a diabetic the percentage of sugar in the venous blood of the arm is found to be less than the percentage in the arterial blood, whereas before administration the two values are nearly the same (**5**). The sugar that disappears is partly oxidised by the muscles, skeletal and cardiac, and partly built up into glycogen in the muscles (**6**). (2) Its *central* or *visceral* action is manifested on the liver in two ways: (*a*) In acute diabetes there is lipæmia and increase of fat in the liver, probably because, in default of carbohydrate, fat is mobilised from the depôts and assembled in the liver to be utilised (**7**). Here it is probably converted into carbohydrate, which explains the low respiratory quotient found in severe cases, and this conversion involves the production of aceto-acetic and $\beta$-oxybutyric acid (*ketosis*) (**8**). Insulin arrests this process, the ketosis and lipæmia vanish, and the fat disappears from the liver, and the respiratory quotient rises. (*b*) The diabetic liver contains less glycogen than normal, since this is discharged into the blood and causes the well-known hyperglycæmia. It is not uncommon to find a concentration of 0·4 or 0·6 per cent. dextrose in the blood in severe diabetes. Insulin causes in the liver a retention of glycogen, which increases *pari passu* with the diminution in the liver fat. This result is possibly obtained because the insulin inhibits the action of the amylolytic ferment of the liver, which becomes excessive in its absence. The sudden fall in blood sugar, which is the most striking clinical effect of insulin, is thus due partly—probably only slightly—to oxidation and partly to storage in the muscles and to the fact that the excessive supply of sugar from the liver is stopped. At the same time any carbohydrate that is taken as food is partly stored in the liver as glycogen and probably partly converted into fat; the reason for the latter statement is that several observers have found a rise in respiratory quotient without any marked increase of combustion, which would occur if most of the sugar taken had been burnt. The following observation shows that storage of sugar by means of insulin may be only a temporary matter. A patient was given by mistake

the enormous single dose of 100 units of insulin, and to counteract this was given by mouth immediately 110 grammes of carbohydrate in the form of dextrose and bread. This was retained in the tissues for two days and then largely excreted in the urine after the effect of the insulin had worn off (**9**).

The early stage of pancreatic deficiency, in which dextrins and amylolytic ferment enter the blood from the liver, is mentioned elsewhere (*see* p. 504). Three other ductless glands, the suprarenals, thyroid and pituitary, act in the opposite direction to the islands of Langerhans, since their excitation increases the sugar in the blood. The suprarenals pour adrenin into the circulation, and this travels to the liver and causes the breakdown of glycogen into dextrose. For this action to occur it appears to be essential for the hepatic plexus to be intact. The thyroid probably acts by exciting the suprarenals; the mode of action of the pituitary is unknown. Claude Bernard's puncture of the fourth ventricle of the brain causes hyperglycæmia by exciting the suprarenals through the splanchnic nerves.

**Pathology of Diabetes Mellitus.** Diabetes mellitus has been thought to be associated with pancreatic disease for over 200 years, because observation showed that in certain cases the pancreas was obviously pathological. In 1889 von Mering and Minkowski by removal of the pancreas in an animal produced the disease experimentally. Recently Allen has shown that if seven-eighths of the gland are removed from a dog a mild diabetes results, while if nine-tenths are removed the diabetes is severe, and further that it is the island, and not the acinar, tissue that is of importance. The clinical features of the experimental disease closely resemble human diabetes mellitus, and in severe cases *ketosis* is present when there is much fat in the diet.

In experimental diabetes the histological changes in the remaining islands are quite characteristic. There is hydrops of the cells (Weichselbaum), and the latter no longer contain their characteristic granules (Bensley); they have a washed-out appearance, because, owing to the deficiency of island tissue, they have been overworked. In human cases the pancreas often looks normal at post-mortem, and there are no obvious histological changes. The difficulty is that the pancreas rapidly decomposes after death, and further that the characteristic histological changes in the islands which are seen in acute cases tend to disappear, if the case is prolonged, because the cells gradually die. However, Allen states that by taking adequate care he can always differentiate the diabetic from the non-diabetic pancreas by microscopical examination, if the tissue is fresh, so that the connection between diabetes and the islands of Langerhans is clear.

This view of the pathology received remarkable confirmation from the epoch-making work of F. G. Banting in collaboration with C. H. Best in 1922. Insulin was first isolated from the dog's pancreas some weeks after tying the pancreatic duct, a procedure which causes the acinar cells to become degenerated, while the island cells remain intact. Finally, a method of preparing insulin from ordinary pancreas from the slaughter-house by means of fractional precipitation with alcohol was carried out, and this method with modifications is at present in use.

In the healthy person, no sugar can be detected in the urine by ordinary tests; but by special methods the urine has been found always to contain up to 0·10 per cent. of sugar. When the blood sugar rises to about 0·18 or 0·20 per cent. the kidney excretes dextrose in amounts that can be readily detected. This is called the *threshold* of the kidney. Cases of true diabetes may have a low or high threshold. In the former case sugar is still excreted even though the percentage in the blood has been lowered by treatment to the normal. In the latter case the sugar excretion stops when the percentage is still high, 0·3 per cent. for instance.

Jacobsen's blood sugar tolerance curves are becoming of increasing importance. A dose of dextrose is given in the early morning on an empty stomach, the blood sugar being estimated beforehand and at stated intervals afterwards. The urine is collected, and any sugar in it is estimated. Results obtained by Dr. W. W.

Payne from eleven normal students after 50 grammes of dextrose are shown in Fig. 57. A is the average curve for the whole series, B is the lowest curve, and C the highest curve. In the latter case a trace of sugar was passed in the urine. In all three curves the blood sugar had nearly fallen to the starting point in one and a half hours; but secondary rises are seen. This method is used to separate the group of diabetes innocens cases (*see* p. 570) from true diabetes mellitus (**29**). In this group the sugar tolerance curve is normal although the patients continually pass sugar. When suspected cases of diabetes mellitus are examined, a smaller dose of dextrose is given—10, 20 or 25 grammes, or occasionally 50 grammes. The blood sugar is often found to rise more quickly than normal, and the rise continues for a longer time, and the return to the original

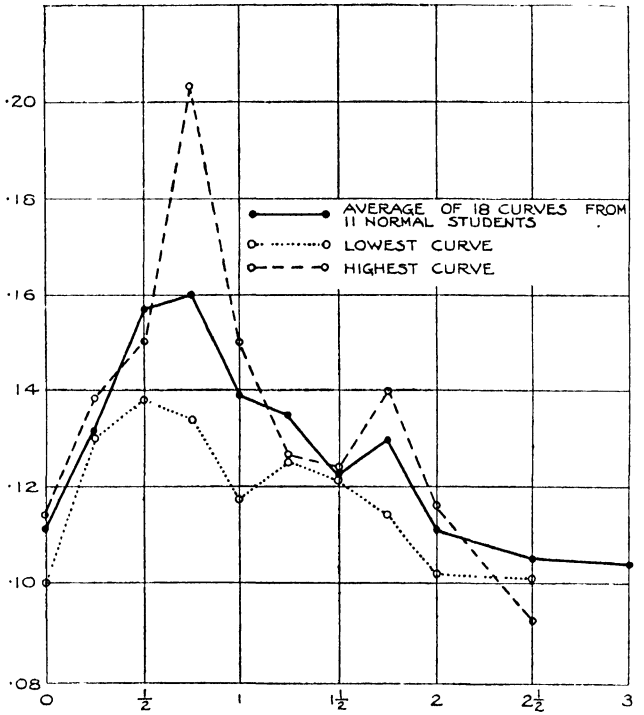


Fig. 57.—Normal Blood Sugar Tolerance Curves.

level is much delayed. This is illustrated by curves B, C, and D in Fig. 58, which were obtained from cases of diabetes mellitus of varying severity. For the sake of comparison, curve A is included, which is the curve of the normal student which most closely resembles the average curve in Fig. 57. It will be noticed that in diabetes the curves tend to rise higher; but a more characteristic feature is the long time required for them to fall to the starting point. It is always more than one and a half hours.

In mild cases of diabetes, sometimes called *alimentary glycosuria*, sugar is only passed in the urine after a carbohydrate meal: but in more severe cases it is passed continuously, even though all carbohydrates are excluded from the diet, because it is produced from protein, and also probably from fat.

**Clinical Tests for Sugar.**—*Fehling's test* consists of (1) a cupric sulphate solution of 34·63 grammes in ½ litre of distilled water, and (2) caustic potash 100 grammes and sodium potassium tartrate 173 grammes in ½ litre of

water. The two solutions are mixed. After some weeks decomposition takes place, and renders the test uncertain. This may be prevented by keeping the two solutions separate, and only mixing them for use, or at short intervals; or the altered mixture may be again rendered fit by the addition of a piece of caustic potash.

When ½ drachm of the reagent is boiled with an equal quantity of urine containing dextrose, the cuprous oxide is thrown down as a yellow precipitate, changing to orange and red-brown. If the quantity of glucose is small, the precipitate is at first bluish green, and then becomes yellow; or if smaller still,

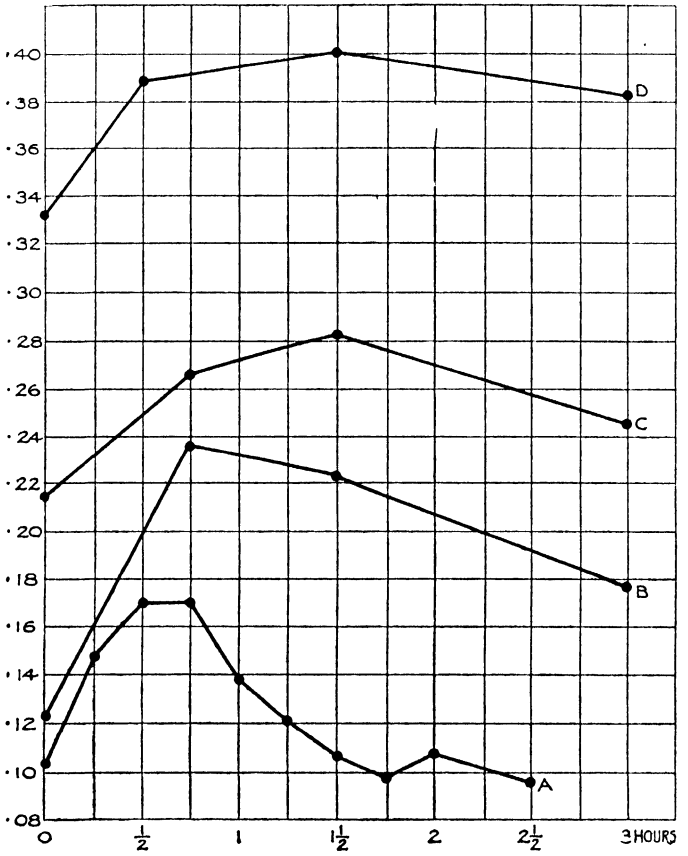


FIG. 58.—Three Blood Sugar Tolerance Curves in Diabetes (for description see text).

the precipitate is slight, and remains bluish green from the presence of unreduced cupric salt.

*Benedict's Qualitative Test.*—The solution is made up as follows: Sodium (or potassium) citrate, 173 grammes; anhydrous sodium carbonate, 100 grammes (or the crystallised salt, 200 grammes); distilled water, about 700 c.c. This is filtered, if necessary. Crystallised copper sulphate, 17·3 grammes, is dissolved in about 100 c.c. of distilled water, and the solution poured slowly with constant stirring into the saline solution. The mixture is then cooled and made up to 1,000 c.c.

To test the urine 5 c.c. of the solution are placed in a test tube, and 8 to 10 drops only of the suspected urine are added. The mixture is heated to vigorous

boiling, kept so for one or two minutes, and then allowed to cool spontaneously. If glucose is present, the whole body of the mixture will be filled with a precipitate which may be red, or yellow, or greenish. If the quantity is less than 0·3 per cent., the precipitate forms only on cooling. If there is no sugar, the solution either remains quite clear, or shows a faint turbidity which is blue in colour, and consists of urates.

Benedict's solution is not appreciably reduced by creatinin, uric acid or chloroform, but it reacts with lactose, greatly increased amounts of glycuronic acid, and homogentisic acid. On the whole it is a better test than Fehling's.

*Phenylhydrazine Test.*—A test tube is filled for about ½ inch with phenylhydrazine hydrochloride, and for another ½ inch with sodium acetate. The test tube is then half filled with urine, and the whole is heated in a water bath for some fifteen to sixty minutes, depending on the amount of sugar present. It is allowed to cool slowly. The yellow sediment is examined, when it will show, under the microscope, sheaf-like clusters of fine crystalline needles (phenylglucosazone), which melt at 205° C.

*Fermentation Test.*—If a small quantity of yeast (washed free from any starch or sugar) be added to the urine, and this be set aside in a warm place for some hours, the glucose will be converted by fermentation into alcohol and carbonic acid. If now the specific gravity be taken, and compared with that of a duplicate specimen placed under similar conditions except for the presence of yeast, it will be found that there is a loss of density corresponding to the glucose destroyed. The difference in specific gravity multiplied by 0·23 gives the percentage. If the test tube be filled entirely and inverted in a saucer, the carbonic acid gas, as it forms, will collect in the upper part and displace the urine.

*Polariscope.*—Dextrose turns the plane of polarisation to the right. This is interfered with by the presence of β-oxybutyric acid, which is lævo-rotatory.

**Fallacies.**—With Fehling's test a small amount of reduction may take place from the presence of glycuronic acid, uric acid, hippuric acid, and creatinine when no sugar is present. Benedict's solution is not so apt to be reduced by these substances.

*Glycuronic acid* is always paired with some other substance which is excreted in the urine. It may appear after the administration of morphine, chloroform vapour, chloral, butyl chloral, camphor, copaiba, cubebs, salicylic acid, and tannic acid. The fresh urine shows slight reduction and fermentation, and is lævo-rotatory. The reduction is increased on boiling for three-quarters of an hour with 5 per cent. sulphuric acid, and the urine often becomes dextro-rotatory, and gives the orcin test.

Sugars other than dextrose may appear in the urine. *Lævulose* may be excreted with dextrose, and also alone. Its presence is suggestive of hepatic deficiency. It gives the reduction and fermentation tests, but can be distinguished by its lævo-rotation and by Seliwanow's test. A mixture of 4 c.c. of the urine and 1 c.c. of Seliwanow's reagent (resorcin, 0·5 gramme; hydrochloric acid, specific gravity 1·195, 30 c.c.; distilled water, 30 c.c.) is heated to boiling in a water bath for a few minutes. If lævulose is present the solution assumes a purple-red colour, but dextrose alone gives no colour change. *Pseudo-lævulose* (iso-glycuronic acid) also gives this colour. *Lactose* occurs in the urine of nursing women and in infants with gastro-enteritis. It gives the reduction, but not the fermentation, test and is dextro-rotatory; with the phenylhydrazine test it gives crystalline needles in spherical clumps, which melt at 200° C. *Pentose* may appear in the urine after eating fruits containing arabinose, *i.e.* cherries, plums and apples. Spontaneous pentosuria is a rare error of metabolism, and is lessened by restricting the protein intake. It reduces copper, but does not ferment, and gives Bial's orcin test.

**Morbid Anatomy.**—In a certain proportion of cases the *pancreas* is obviously diseased to the naked eye. Most frequently it is the subject of atrophy

or fibrosis, or the two combined ; and other changes found in these circumstances are fatty transformation of large parts of the gland, suppuration, hæmorrhage, carcinoma, calculus in the ducts, and cysts. The histological changes have already been considered. In many cases, especially those of short duration, the post-mortem appearances of other organs differ very little, if at all, from the normal ; in older cases the pathological lesions due to the complications are found. There is often enlargement of the *kidneys*, which otherwise may look perfectly normal. The *liver* presents nothing abnormal to the naked eye, except in the rare cases of hæmochromatosis or diabète bronzé (*see* p. 484). The *blood* sometimes presents a peculiar pink or strawberry colour, and on being placed aside a creamy layer collects on the surface. This condition has been called *lipæmia ;* but the granules constituting the creamy layer certainly differ from those of true fat, and are composed of lecithin and globulin (*see also* p. 1018). The condition of the blood has been recognised during life in the retinal vessels, which, both arteries and veins, are salmon-coloured at the centre of the fundus and creamy-coloured at the periphery (*lipæmia retinalis*) ; they are dilated and resemble one another closely.

**Ketosis.**—When there is a deficiency of carbohydrate, or the latter cannot be utilised, there is probably a conversion of food fat and body fat into carbohydrate, and this is accompanied by the formation of *aceto-acetic acid*, $CH_3\ COH = CH\ COOH$ (incorrectly called diacetic acid), from the fat (**8**). It is a poisonous substance, and is largely converted in the liver, by reduction, into the harmless *β-oxybutyric acid*, $CH_3\ CHOH\ CH_2\ COOH$. A little of it is converted into *acetone*, $CH_3\ CO\ CH_3$, by the loss of a molecule of $CO_2$. All these three substances appear in the blood and urine, and in addition acetone escapes in the breath. This condition is called ketosis (*see* p. 547). It appears during starvation, and when there is lack of carbohydrates in the diet, especially if the fat is in excess ; in severe vomiting, such as cyclical vomiting and pernicious vomiting of pregnancy ; febrile and cachectic conditions ; and post-anæsthetic or "delayed chloroform poisoning," phosphorus poisoning, acute yellow atrophy and eclampsia, which all show focal necrosis of the liver with fatty changes ; in healthy persons after taking large doses of sodium bicarbonate ; and in diabetes mellitus. When the aceto-acetic acid reaches a high concentration in the blood it may cause death by producing coma. This may occur in severe diabetes or after prolonged vomiting (*see* p. 418). In a very prolonged case of severe diabetes the body fat largely disappears as the patient wastes, and the ketosis may almost vanish, while the patient dies of inanition.

**Clinical Tests for Ketosis.**—There is no colour test for β-oxybutyric acid in the urine. Gerhardt's test, which is used for aceto-acetic acid, consists in adding ferric chloride to the urine when a port-wine colour is obtained. It disappears on heating. It is not a very sensitive test. It must be distinguished from a similar reaction after taking salicylates, but in this case the colour is not discharged by heat. Two tests with sodium nitroprusside are given both by aceto-acetic acid and acetone, but they are about twenty times as sensitive for the former as the latter. In Legal's test a small crystal of sodium nitroprusside or a few drops of a freshly prepared solution of it are dropped into the urine, and then a little caustic soda. A cherry-red colour is developed which soon fades ; an excess of acetic acid now added produces a carmine-red or deeper purple colour. In Rothera's test solid ammonium sulphate is added to the urine with a crystal of sodium nitroprusside and ammonia in excess. A purple colour gradually develops. This is much the most sensitive test there is for aceto-acetic acid.

These tests are excellent for indicating the presence of ketosis ; but they are not of much use in indicating its amount, in order to be able to determine whether a diabetic patient is on the verge of coma or not. One reason is that when coma approaches the excretory power of the kidneys begins to fail with fall of blood pressure, so that the amount of these substances in the urine is diminished, and

inversely they accumulate in the blood. It is this accumulation that gives the measure of the danger. Three methods are available (*see* p. 548). The determination of the ratio of ammonia nitrogen to total nitrogen and the test of giving 5 grammes of sodium bicarbonate by the mouth have already been sufficiently described. The alveolar $CO_2$ method depends on the fact that the normal $CO_2$ values lie between 4·5 and 6·2 per cent., being rather lower in women than in men. In diabetes a value of 2 per cent. means that coma may supervene within twenty-four hours, if no improvement takes place. A patient may go on living for many days or even some weeks with the alveolar $CO_2$ between 3 and 4 per cent. In the worst evert coma will not supervene before three or four days. It has already been stated that the lowering of the $CO_2$ which is caused by increased respiration is a mechanism compensating for the increase of fixed acid in the blood, and this prevents the hydrogen ion concentration of the blood rising too much.

**Symptoms.**—The onset of diabetes is often insidious; the patient only gradually notices that he drinks more fluids and passes more urine than normal; or he may complain of debility and loss of flesh rather than of any alteration in his urine. In some cases the onset is acute, the patient being able to remember the exact day when he first noticed he was thirsty.

In the more severe type of case, which may come on acutely or develop from a milder type, the characteristic symptoms, in the absence of treatment, soon become unmistakable—namely, frequent and abundant micturition, great thirst, generally a very large appetite, physical weakness, and loss of flesh. The appetite is sometimes enormous, but in other cases it is but little affected, and often fails towards the end. The mouth and lips are dry, the tongue red, raw, and "beefy"; and there is generally a sweet taste in the mouth. The digestion is, as a rule, good, and patients may have no difficulty in disposing of large quantities of food. The bowels are generally confined. The skin is harsh and dry. At the same time, nutrition is profoundly affected; the patient rapidly loses flesh, and becomes excessively weak; he is indisposed to make any mental effort, and is depressed and irritable. The teeth become loose from pyorrhœa alveolaris. There is often loss of virility in men, and in women the menses may cease.

The urine is increased to 5 or 10 litres in the day, the amount of sugar passed being over 500 grammes and the concentration up to 8 per cent. Owing to the presence of so much sugar, the specific gravity is raised to 1,040 or 1,045. The urine is generally pale yellow, or almost like water; it has a sweetish odour like hay and a sweet taste. The reaction is acid. It contains acetone, aceto-acetic and $\beta$-oxybutyric acids.

Mild cases of diabetes are sometimes described as "alimentary glycosuria"; but blood sugar tolerance curves show that this is a type of true diabetes, although the glycosuria may be found only after a heavy meal containing much starch. There is no thirst, and the amount of sugar passed in the day may be under 50 grammes. There may be no symptoms, but often the patients notice that the volume of urine is increased. They may suffer from various complications. This type occurs in elderly people particularly.

**Complications.**—In the course of diabetes a number of complications are liable to occur. The irritation of the saccharine urine may excite in women a troublesome *pruritus* of the vulva, and in men *balanitis*. There may be a general pruritus of the skin. *Carbuncles* and *boils* are liable to occur in various parts of the body, and the former may be the cause of death. A form of *xanthoma* has also been seen in diabetes. There is sometimes *gangrene* of the toes or of an entire limb, but this is associated with atheromatous arteries. *Albuminuria* may be present, indicating coincident renal changes. The Achilles jerks and knee jerks are commonly absent in diabetes; this may be due either to *peripheral neuritis* or to degeneration in the fibres of the *posterior nerve roots* between the pia mater and grey matter, which leads to changes in the posterior columns of the cord (**11**). *Neuralgia* may be severe, especially sciatic, occipital and trigeminal. There

may be cramps. Œdema of the feet and legs may be seen in very wasted individuals (*cachetic œdema*), and it can be readily produced by administering too large doses of sodium bicarbonate. *Phthisis, pneumonia* and other infectious diseases do not occur more commonly among diabetics than among the general population; but the outlook is worse, although it has improved with modern methods of treatment.

Vision is affected in diabetes in several ways. The most important is the formation of *cataract*, which is always symmetrical, and develops rapidly in the young and middle-aged, more slowly in old people. Other changes are: *Iritis* and *defects of accommodation*, from paralysis of the ciliary muscle sometimes coming on quite suddenly; *atrophy* of the optic nerve and *retrobulbar neuritis*; and *retinitis*, resembling that of albuminuria, with white spots and hæmorrhages, and probably secondary to arterial changes. *Amblyopia*, without visible ocular changes, like that of uræmia, also occurs.

**Diabetic Coma.**—This name has been given to a group of symptoms due to the accumulation in the blood of aceto-acetic acid, which acts as a poison both on the circulatory and on the central nervous systems, and with a fatal result. Deficiency of $CO_2$ in the blood may also play a part in the causation of this symptom complex. The causes predisposing to coma are: (*a*) a diet rich in protein and fat; (*b*) excitement or emotional shock; (*c*) general anæsthesia: probably gas with oxygen is the least harmful, but it is very important that the patient should not get blue; (*d*) acute infections; (*e*) impaired function of the kidneys, so that aceto-acetic acid is imperfectly excreted; (*f*) constipation; (*g*) failure to continue the use of insulin in a young patient, who has put on fat by its help. The onset is often gradual, but may be indicated by loss of appetite, by a rapid fall in the quantity of urine and of sugar passed in the day, and by obstinate constipation. Sometimes there is severe abdominal pain. The patient then rather rapidly falls into a condition of collapse and coma. The pulse is quick and feeble, the surface cold, the features pinched, and the extremities livid. He lies with the eyes half open, taking no notice of his surroundings; and though he can be roused by a question, he answers, if at all, in a dazed manner, as if only half comprehending it. The breathing in these cases is peculiar; it is slow, deep, and sighing in character; the movements of the chest are very extensive; the respirations become rather more frequent towards the end. At the same time, examination of the chest reveals nothing abnormal. This form of breathing has been called *air hunger*. In many cases a sweetish, fragrant, or ethereal odour, likened to the smell of apples by some, may be noticed about the bed of the patient; it has been attributed to acetone. This condition may last from one to three days, when the pulse gets more and more feeble, though the heart may be beating forcibly, the patient more apathetic, and finally quite comatose; and death ends the scene. Occasionally there is a little muttering delirium. In some cases the symptoms are much more rapid; without any warning the patient becomes collapsed, with a quick, feeble pulse and livid extremities; air hunger develops, and he dies in coma after twenty-four or thirty-six hours.

**Renal Glycosuria.**—*Benign Glycosuria* (*Diabetes Innocens*).—These terms are applied to a condition, of which a number of cases have now been described, where the patients pass sugar continuously for years, but remain in perfect health and have none of the symptoms of diabetes. The output of sugar is small, being usually not more than 30 grammes in the day. A dose of carbohydrate makes little difference to the output. In some cases it is difficult to get rid of the sugar by treatment; in others it disappears rapidly with starvation. The blood sugar tolerance curve is normal (*see* p. 565). The blood sugar on an empty stomach is normal, and yet sugar is passed, showing that the threshold of the kidney is low. The ætiology of the condition is unknown, but occasionally it is congenital. No treatment is required. Glycosuria associated with a

low renal threshold occurs experimentally when an animal is poisoned by phloridzin.

**Diagnosis.**—The presence of sugar in the urine as determined by one of the reduction tests, when there is also a present or past history of either thirst, polyuria or muscular weakness, makes the diagnosis of diabetes mellitus certain. If the urine is not tested, the presence of the disease may be overlooked, and the patient may be treated for a vague weakness and "debility"; or the possibility of diabetes underlying one of its complications, such as carbuncles, pruritus, or coma, may be forgotten. It must be borne in mind that coma may occur as a result of diabetes in persons not known to be diabetic; and that in diabetics abdominal pain, severe enough to suggest that a laparotomy is needed, may be the first symptom of the onset of coma. The chief difficulty is when a patient's urine gives a slight reduction on several occasions and there are no symptoms. The fallacies connected with the clinical tests have already been considered; but even if the presence of dextrose is proved by the fermentation and phenylhydrazine tests, it is still necessary to find out if the patient is suffering from true diabetes mellitus. If the blood sugar is above 0·16 the diagnosis will be very probable, but if lower than this it will be best to carry out a sugar tolerance test with analyses of the blood after a dose of dextrose, and this will also assist the prognosis.

**Prognosis.**—Diabetes mellitus is a very serious disease, tending to run a more rapid and unfavourable course in young than in elderly subjects. At the same time the prognosis has improved with the introduction of insulin and careful dietetic treatment. In some cases the sugar tolerance increases as the result of treatment. The writer's greatest success has been with a boy brought into hospital in coma, who after coming round required 70 units of insulin a day to keep the blood sugar normal, *i.e.* 0·103 per cent. In seven weeks' time this was reduced to 20 units, and as the blood sugar was 0·087 per cent., a further reduction to 16 units a day was made in another six weeks' time. At the time of writing he is taking 8 units at night only, and the blood sugar at 1 p.m. was 0·13. He is on a strict diet. Without treatment the prognosis is uniformly unfavourable in young subjects, as the disease tends to progress. It may progress in spite of treatment. The prognosis is the more unfavourable the later in the disease that treatment is instituted. In elderly subjects with so-called "alimentary glycosuria," a fatal result need not occur in the absence of treatment; but there is always the possibility of complications such as carbuncle, cataract and retinitis, so that adequate treatment should always be instituted to prevent these occurring.

**Prevention.**—Since early treatment is so important, the periodic examination of the urine has been recommended. This should certainly be carried out in the case of the healthy members when diabetes runs in a family. The best way of avoiding diabetes is to live a healthy life with plenty of regular exercise and to avoid obesity and focal sepsis.

**Treatment.**—The first treatment of diabetes on rational lines was carried out by Rollo, who prescribed a diet of animal food containing no starch and sugar. This plan was very generally adopted up to 1915, enormous amounts of protein and fat being allowed. It was comparatively rare for the urine of a diabetic to become sugar-free on such a diet. Experiments carried out on depancreatised dogs by F. M. Allen and clinical observations at different times by Von Noorden, Guelpa and Graham showed the importance of undernutrition in treatment. The plan usually adopted consisted in first of all making the urine sugar-free by fasting and then in giving food in gradually increasing amounts, in severely restricting the carbohydrate intake, and in permanently underfeeding the patient, so as to keep him thin. In an underfed person the basal metabolism is low, so that there is less call on the island tissue, and this is of great advantage where the islands are deficient as in diabetes, because they steadily deteriorate

if they are overworked. A high protein diet causes a high metabolism; this has been called the *specific dynamic action* of protein. The treatment of diabetes has been revolutionised by the discovery of insulin: but although fasting is now no longer necessary, some limitation of diet is still necessary, particularly in the more severe cases.

At the beginning of treatment possible focal sepsis which may be due to dental apical infection, septic tonsils, gall-bladder, or appendix should be eradicated. The aim of treatment is to administer insulin in such quantities and at such times that the blood sugar remains within normal limits during the twenty-four hours.

The principles of treatment can be deduced from Dr. W. W. Payne's determinations of the blood sugar of a patient shown in Fig. 59. Curve B is on the average higher than curve A, because carbohydrate was substituted for some of the protein. Otherwise the diet was the same in A and B. The effect of giving insulin twice a day at twelve hours' intervals before breakfast and supper, is, broadly

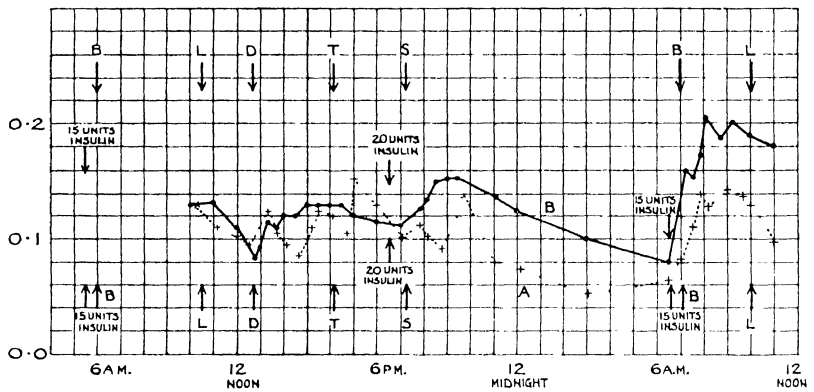


FIG. 59 — Blood sugar of a man, 40 years old, weighing 138 lb. Curve A, was obtained after he had been for 3 days on diet containing 72 grammes of protein, vegetables and fat. Curve B, after 3 days on a similar diet except that for 30 grammes of the protein, a similar amount of carbohydrate was substituted in the form of bread and potatoes at breakfast and supper. The calorie value was just over 1800. I, insulin administered; B, breakfast; L, lunch; D, dinner; T, tea; S, supper. Blood sugar, by Hagedorn and Jensen's method.

speaking, to produce a double curve, a rise immediately after the meal followed by a fall. In this case, with rather a smaller dose of insulin, the morning rise was much higher than the evening rise; in curve A the rise certainly began before breakfast, and may be explained by the increased metabolic activity which suddenly occurs on waking. In other cases, with equal doses of insulin, the evening rise may be the higher, and when three doses of insulin are given at 6 a.m., 12 noon, and 9 p.m., *i.e. after* supper, a triple curve is obtained which may be just as high at night as in the day, presumably because protein metabolism takes place more slowly than carbohydrate metabolism, and so is delayed till nighttime (**12**).

When treatment is first begun, injections of 4 or 5 units are given before breakfast and supper. The dose is increased by 4 or 5 units every two or three days. The sugar in the urine becomes less and eventually disappears, but the increase is still continued until the patient experiences mild symptoms of hypoglycæmia two to six hours after the dose, viz., shakiness of the limbs, sweating, hunger, emptiness or nausea, giddiness or dizziness, mental disturbances, headache, palpitations, exhaustion, faintness, numbness, feelings of heat or cold, and occasionally diarrhœa. These symptoms are arranged roughly in their order of

frequency (**12**). By this means the patient experiences at an early stage, and while under medical supervision, exactly what it feels like to receive an overdose of insulin. His future treatment will be carried out with greater confidence for this experience. The maximum dose tolerated by the patient will be just less than that which causes symptoms, and this is the dose to be prescribed, since it will keep the blood sugar normal and the urine permanently sugar-free. By such means blood sugar examinations can be dispensed with to a large extent. Whereas a reduction of blood sugar to about 0·07 per cent. in normal people usually produces symptoms, the latter may be experienced by certain patients, who have been for a long time hyperglycæmic, at much higher levels of blood sugar, such as 0·09 per cent., 0·012 per cent., or even 0·18 per cent., because the body has been for long adapted to the hyperglycæmia. If the symptoms are used as a guide to treatment the blood sugar may still fall to normal in time. Great care must be taken with severely emaciated or phthisical patients. Sugar must be given to the former to prevent severe hypoglycæmic symptoms early in the treatment, and, in the case of the latter, the dose of insulin must be increased slowly, so as to bring down the blood sugar gradually, as otherwise there may be a severe exacerbation of the infection.

In some severe cases of diabetes 30, or even 40, units twice a day will be insufficient to produce a normal blood sugar, in others a dose of insulin may produce hypoglycæmic symptoms in two or three hours, and the blood sugar may then rise rapidly and glycosuria occur, before the next dose in twelve hours' time. Both these occurrences are indications for the administration of insulin three times a day, viz., at 7 a.m., 1 p.m., and 10 p.m., because the principle of spreading the insulin as evenly as possible over the twenty-four hours applies also in these cases.

Sometimes patients complain of stinging immediately after injection owing to the acid in the insulin solution. This may be obviated by neutralising it by drawing up into the syringe, and mixing with the insulin, about half its volume of $\frac{1}{100}$ N. caustic soda containing 0·25 per cent. tricresol. The exact amount required varies with different brands, but enough should be used to cause a slight turbidity in the syringe from the precipitated insulin. Urticarial wheals at the site of injection, coming up twelve hours later and subsiding in two or three days, may sometimes be partly controlled by neutralising the insulin. They are chiefly due to impurities in the particular brand and another one should be tried. They are not due to hypersensitiveness on the part of the individual to the proteins of the particular animal from which the insulin has been prepared.

*Diet.*—Efficient dietetic treatment is essential for treatment with insulin. The normal weight of the patient is ascertained from Table I (p. 560) after determining the length of his body. The daily ration of protein allowed is $\frac{1}{2}$ gramme per lb. of body weight. Eggs, meat, chicken, fish, cheese and perhaps a small quantity of some diabetic food, containing the requisite amount of protein, are divided among the three principal meals of the day. The calculation can be simplified by reckoning that 1 ounce of all these protein foods or one egg, contain about 6 grammes of protein; so that if, for instance, 60 grammes of protein were to be eaten in the day, the patient would be told to take 10 ounces of these foods, counting each egg as equivalent to an ounce. The calorie value of this food is then calculated. The total calorie value allowed to the patient, so long as he is leading a sedentary life, can be calculated from the patient's age and normal body weight by means of Fig. 55 (p. 559). The difference represents the calorie value still to be added in the form of butter and cream and vegetables.

Many vegetables are specially valuable for diabetics because the carbohydrate content and calorie value are low, and the protein content may be neglected. They are satisfying, because they are bulky and fill up the stomach. The patient may be allowed to eat any quantity of these vegetables he likes, *e.g.* 24 or 32 ounces. The most useful are those which are often classed together as containing 5 per cent. carbohydrate. This is of course, only a very rough average. Raw vegetables

contain more carbohydrate than when they have been boiled in water. The following *raw* vegetables and fruit may be used: lettuce, cucumbers, endive, celery, tomatoes, watercress, mustard and cress, lemons. The following common vegetables are placed approximately in their order as regards carbohydrate content after boiling once in water (Cammidge): marrow, celery, rhubarb, spinach, cabbage, sea-kale, cauliflower (not the stalk), onions, beetroot, sprouts, turnips, swedes, carrots. In these the carbohydrate content varies between 0·3 and 4·5 per cent. After boiling in three changes of water, it is reduced in all of them to less than 1 per cent. The roots should always be sliced before boiling. Other vegetables that may be allowed are asparagus, beet greens, egg plant, radishes, leeks, French beans, scarlet runners, broccoli, and Jerusalem artichokes. Artichokes, when sliced thin and fried, provide a good substitute for potatoes. Bran and agar cakes, according to Allen's receipt, made thin and crisp, can be taken in any quantity, but they may cause indigestion (**13**). The composition of other common articles of diet is shown in Table 3. Butter-nuts (*Juglans cinerea*),

TABLE 3.

| One ounce (30 grammes) contains approximately: | Carbohydrate. Grammes. | Protein. Grammes. | Fat. Grammes. | Calories |
|---|---|---|---|---|
| Vegetables, 5 per cent. group | 1 | 0·5 | 0 | 6 |
| Brazil nuts | 2 | 5 | 20 | 210 |
| ½ Grape fruit or 1 small orange | 10 | 0·5 | 0 | 40 |
| Potatoes | 6 | 1 | 0 | 28 |
| Boiled rice | 7 | 1 | 0 | 32 |
| Cooked macaroni | 4·5 | 1 | 0·4 | 26 |
| Oatmeal | 20 | 5 | 2 | 120 |
| Bread | 15 | 3 | 0·3 | 75 |
| Flour | 21·5 | 3·5 | 0·3 | 104 |
| Meat, cooked, lean | 0 | 8 | 5 | 75 |
| Bacon | 0 | 5 | 15 | 155 |
| Chicken, cooked | 0 | 8 | 3 | 65 |
| Fish, cooked | 0 | 6 | 0 | 25 |
| Egg, one | 0 | 6 | 6 | 75 |
| Cheese | 0·1 | 8 | 10 | 130 |
| Milk protein or casein bread | 0 | 18 | 9 | 158 |
| Butter | 0 | 0 | 25 | 225 |
| Milk | 1·5 | 1 | 1 | 20 |
| Cream, 20 per cent. | 1 | 1 | 6 | 60 |
| „ 40 „ | 1 | 1 | 12 | 120 |

pignolias and Brazil nuts contain less carbohydrate than other kinds of nuts, and so are most suitable for use. Most so-called diabetic foods, made commercially, are unsuitable for diabetes except in small quantity, owing to their high protein content. Water, tea and coffee without milk, and clear broth can be taken freely. In debilitated subjects alcohol is allowed in the form of whisky, brandy and dry wines, such as sherry and sauterne. Beer must not be taken; 2 ounces of absolute alcohol can be oxidised by a diabetic per diem. Saccharine may be used as a sweetening agent.

After the correct dose of insulin has been determined, while the patient is taking such a diet as the one just described, the question will arise as to whether the carbohydrate content of the diet may be increased. Such an increase should only be allowed in slight degree at breakfast and supper in the case of young persons where hope is entertained that they may eventually regain some sugar tolerance. In older patients this consideration does not arise, and some increase may be allowed in the form of milk, fruit, such as oranges, grape fruit, etc., potatoes, porridge, and bread, preferably toasted—1 or perhaps 2 ounces—at breakfast and supper. The more carbohydrate allowed, the greater will be the amount of insulin required for its utilisation, and the more uncertain will be the exact regulation of the blood sugar to within the normal limits.

When the patient begins to get about and take more exercise the caloric value

of the diet may be increased by carbohydrate, if allowed, and fat to the extent of 20, 30, or even 50 per cent. At this stage some ketosis may be found when the urine is examined by Rothera's test. This often occurs when too little insulin is being used, so that the blood sugar remains above normal (**14**), but it is also due to there being too little carbohydrate or too much fat in the diet, and some modification should be made if the reaction in the urine is marked. A slight Rothera's reaction is of no consequence.

*Choice of Case for Insulin Treatment.*—Insulin treatment must not be used for renal glycosuria. The glycosuria of pregnancy is frequently of this nature; but where pregnancy is complicated by true diabetes the woman should be given the choice as to whether the pregnancy should be terminated or not, and if not, insulin treatment should be instituted. The success of insulin in removing symptoms in practically all patients with glycosuria and hyperglycæmia indicates that in all such cases the pancreas is primarily at fault, and it is reasonable to supply the deficiency by administering insulin, and this should certainly be done when complications are present such as gangrene, cataract, etc. In very mild *elderly* cases the dietetic measures just described may be sufficient by themselves to bring down the blood sugar to normal, though greater relaxation in diet can be allowed if insulin is taken. In *young* subjects, however mild the case, insulin treatment should be instituted at once both to prevent the disease becoming worse and by resting the islands to produce some curative effect. Dextrose and a large dose of insulin, *e.g.* 40 units, should be given before a general anæsthetic.

*After Treatment.*—Patients must be taught to administer insulin to themselves, and should know how to test the urine for sugar. In elderly patients insulin may be discontinued at any time and no harm will result. Its administration may also become unnecessary. In the young the dose of insulin should be the maximum that the patient can tolerate without producing symptoms. The urine should always be sugar free. Such patients will usually put on weight. It is safer for them to remain thin by not allowing too big an increase in the calorie value of the diet, because a large increase in weight, which means an increase of body fat, renders the patient more liable to develop a serious ketosis, if he contracts an acute infection or stops insulin treatment. Under no circumstances should his weight be more than the average for his body length (*see* Table 1, p. 560). Patients should take less insulin if they are to take unaccustomed muscular exercise, since the latter tends to lower the blood sugar. A patient treated along the lines just laid down may find after a time that the dose of insulin to which he is accustomed is becoming too large, since he is experiencing hypoglycæmic reactions. This means that he is improving, and the dose must be diminished, and later on, perhaps, the amount of carbohydrate in the diet increased. Past experience has shown that in young subjects diabetes is a progressive disease, and so improvement of this kind should not be followed by the complete omission of insulin and relaxation of diet. If a cure with insulin is possible, it will probably require some years before it is complete.

*Diabetic Coma.*—At the beginning of diabetic coma a large dose of insulin, *e.g.* 60 units, should be given in part subcutaneously and in part intravenously and the subcutaneous injection should be repeated every four hours, until the urine becomes free of sugar and ketone bodies; insulin is then stopped until sugar appears again in the urine, and it is then continued in smaller doses at longer intervals. It is important to test every specimen of urine for sugar as it is passed, and to catheterise the patient in order to get a specimen, if there has been no urine to test for three or four hours. The reason for these precautions is that with the vigorous treatment necessary for diabetic coma, it is very easy for the patient to pass into hypoglycæmic coma, without any intermediate return of consciousness. This can be prevented by stopping the insulin for the time being as soon as the urine is free from sugar. The symptoms of hypoglycæmic coma are to a certain extent the converse of diabetic coma. There is profuse sweating, the

pulse is full and bounding, the veins stand out prominently. There may be some œdema of the lungs with cyanosis and fluid expectoration. The patient is convulsed, and there are no ketones in the urine. One patient complained of not being able to breathe.

An additional very important method of treatment is to give large amounts of fluid, containing dextrose (or cane sugar) and sodium bicarbonate. This will have the effect of keeping up the flow of urine and so will help to get rid of the poisonous aceto-acetic acid, which is probably the cause of diabetic coma. A stomach tube, Einhorn's tube or a long nasal tube and funnel, are essential for administering the fluid to an unconscious patient. The patient is made to lie on the right side, so that the fluid will gravitate at once towards the duodenum. A pint of warm sterile, isotonic solution is given every hour until a free flow of urine is established. On no account should this treatment be stopped if the patient vomits, but the actual duration of the administration should be prolonged so as to prevent distension of the stomach. Care must be taken by frequent examination to see that œdema of the lungs does not develop. From twenty-four to forty-eight hours of such treatment will be required before the patient becomes fully conscious after being in deep coma. When death occurs in spite of vigorous treatment, uræmia may be the cause, since in several cases the blood urea has been found to be high (**12**).

*Hypoglycæmia.*—There is no danger of severe hypoglycæmia in the early stages of treatment, if the latter is carried out correctly. Later on, it may result from severe muscular exertion, especially if large doses of insulin are being taken in order that a high carbohydrate diet may be metabolised. Patients should carry some lumps of sugar with them to eat in case of emergency.

In a case of *hypoglycæmia coma* a subcutaneous injection of 15 to 20 minims of adrenalin (1 in 1,000), or failing this the same dose of pituitrin may be given. This treatment will be of value if the liver contains a good store of glycogen. Plenty of sugar—4 to 8 ounces of cane sugar or dextrose—is given by mouth or by stomach tube. If the patient does not recover consciousness rapidly, an intravenous injection of a 5 or 6 per cent. solution of dextrose is given.

# DISEASES OF THE THYROID GLAND

## GOITRE

(*Bronchocele*)

A goitre is an enlarged thyroid gland.

**Ætiology.**—Goitre is frequent in certain localities, and especially in limestone districts. In England it occurs in Derbyshire. On the Continent it is frequent in the mountainous regions of Savoy, Switzerland, Northern Italy, the Tyrol, and Styria. It is not, however, confined to the hills, but spreads down into the villages on the plains beneath. It has been conclusively demonstrated that endemic goitre results from lack of iodine and tends to occur among people who live far away from the sea. There are also contributory factors which predispose to goitre by making bigger demands on the thyroid than usual. One of these may be the contamination of drinking water with some organic material in the soil, possibly fæcal. Another may be infection, since acute goitre sometimes occurs in epidemics (**15**). Other factors are puberty, pregnancy, sex, and possibly a diet which contains too much fat. Goitre is commonest in females and in young people. Sporadic goitres occurring in non-goitrous districts are to be explained along these same lines. Endemic goitre is often found to be associated with *myxœdema* and *cretinism*.

**Morbid Anatomy.**—Goitre occurs in the following forms: (1) colloid goitre;

(2) encapsuled adenoma, which may be solid or cystic; (3) malignant disease; (4) exophthalmic goitre; (5) toxic adenoma.

Goitres, which are mostly bilateral, arise as the result of a diminution in the iodine content of the gland which stimulates its activity; there is increase in the height of the vesicular epithelium and increase in the vesicular cells, and in the parenchyma cells which lie in between the vesicles. New vesicles may form from the parenchyma cells, and these may at first contain little colloid and iodine. There is also increase of fibrous stroma and blood supply. At any stage in the process there may be involution with complete recovery. On the other hand, this active stage may be followed after some years by degeneration of two kinds: (*a*) atrophy of the cells and their replacement by fibroblasts, leading on to fibrosis and cyst formation, or (*b*) great enlargement with increase in the colloid of the gland—"colloid goitre," which represents a resting stage of the gland with accumulation of colloid and iodine after the period of previous activity, due to want of iodine. Adenomas of the thyroid probably arise from cell rests which grow under the same stimulus that produces colloid goitre. The differentiation of adenomas from colloid goitres is not absolutely clear cut, and intermediate types are common. Adenomas may pass through the same stages of involution, atrophy, cyst formation, or colloid formation, as affect the whole gland. As a rule adenomas are multiple and develop about the age of puberty. The size of a goitre varies from a moderate prominence of the neck on either side to a mass as large as the fist or a fœtal head, which hangs down in front of the upper part of the sternum, such as those which have been so common in Switzerland and Savoy.

**Symptoms.**—Enlargement of the neck and a feeling of fulness are often the only symptoms. If the goitre is very large, there may be dysphagia from pressure on the œsophagus, or dyspnœa from compression of the trachea, or of the recurrent laryngeal nerves. If there are any symptoms other than the local effects of the enlarged gland, they are indicative of diminution of the thyroid function, or hypothyroidism (*see* Myxœdema). The basal metabolism is normal or slightly reduced in colloid goitre.

Malignant disease occurs generally after middle age, and forms a hard, rapidly growing tumour, which infiltrates and presses upon surrounding parts. When carcinoma or sarcoma is the cause of a goitre, the functions of the gland are generally carried on; and if in such a case there are secondary growths in internal organs, the total removal of the thyroid does not cause myxœdema.

**Diagnosis.**—This is, as a rule, easy. The thyroid nature of any enlargement is proved by its movement up and down with the larynx during the act of swallowing.

**Treatment.**—Prophylaxis in areas of endemic goitre consists in administering 0·1 gramme of sodium or potassium iodide once a day for ten days in the spring and autumn. In the canton of Appenzell, in Switzerland, 0·25 to 0·5 gramme of potassium iodide has been added to each kilogram of salt, so that all the inhabitants cannot fail to get some. Congenital goitre can be prevented by treating the mother during pregnancy. In the treatment of the earliest stage of goitre, when adenomas are not present iodine may be given internally as tincture in doses of 2 or 3 minims thrice daily. The administration of iodine has caused adenomatous goitres to overact and hyperthyroidism may continue for years without further administration of iodine. Iodine does not intensify the hyperthyroidism of true exophthalmic goitre. Iodine treatment is not so successful in a developed colloid goitre, and thyroid extract may be tried. Surgical measures may be necessary in very hard or very large goitres and in malignant disease, if recognised sufficiently early; they are the enucleation of an encapsuled tumour and the removal of the greater part of the gland. The Röntgen rays may also be employed.

## EXOPHTHALMIC GOITRE AND TOXIC ADENOMA

*(Graves' Disease, Basedow's Disease)*

This disease, occurring more frequently in women than men in the proportion of 3 to 1 (**16**), was first described by the Dublin physician Graves in 1835, and by a German physician, Basedow, in 1840. The prominent symptoms are: protrusion of the eyeballs, enlargement of the thyroid gland, frequent action of the heart, and tremor.

**Ætiology.**—It is now recognised that these symptoms result from over-activity of the thyroid as the result of two distinct conditions:

1. *True Exophthalmic Goitre.*—This is of unknown origin; the onset is sudden and there are frequent remissions and exacerbations. There are corresponding alterations in the respiratory exchange or basal metabolic rate (*see* p. 558). The latter is high when the disease is severe, and *vice versâ*. It occurs usually in young persons. Sometimes there has been a neurotic tendency, as shown in hysteria or epilepsy, or mental disease in the family. In a few cases it has followed rapidly upon some emotional or mental excitement, or even direct injury to the head. Hyperthyroidism was frequently noticed among soldiers during the war, and was due to the intense mental strain. A hereditary connection has sometimes been observed: it has been observed in mother and son or daughter; more often it attacks brothers and sisters in the same family. The fact that the incidence of exophthalmic goitre was diminished in Germany during the war suggests that abundant feeding may help in its causation (**17**). The incidence of exophthalmic goitre is greater in rural districts than in towns, and in Wales and the west of England than the east.

2. *Toxic Adenoma.*—There is a long history of swelling of the thyroid. Symptoms of over-activity begin to develop about the age of thirty-five or forty years, and may result from the administration of iodine to a case of adenoma. Usually, but not always, separate adenomas can be felt in the gland. The disease pursues a steady course over years with symptoms that gradually become more severe. There are no remissions. There is a slow, steady increase in the respiratory exchange. Exophthalmos is often not very marked. There is a special liability to cardiac irregularities, such as auricular fibrillation, and advice may first be sought on account of cardiac failure.

**Morbid Anatomy.**—The vesicular and parenchymatous cells proliferate more, and there is greater activity in the gland than is seen in parenchymatous goitre. The contents of the vesicles lose their colloid nature and become mucous and granular. In later stages the gland may become fibrous or cystic. These changes may be diffused throughout the gland, or they may be limited to well-defined adenomas in the gland (toxic adenoma).

The thymus gland is often persistent and enlarged, and there is sometimes enlargement of the spleen, of the cervical or bronchial glands, or of Peyer's patches in the intestine. There are no material changes in the cervical sympathetic ganglia or in the brain or spinal cord. The blood shows a lymphocytosis.

**Pathology.**—From the normal thyroid gland Kendall has isolated a crystalline substance containing iodine, *thyroxin*, which is the active principle of the gland. That the disease is due to hypertrophy of the thyroid producing an excess of thyroxin is shown by the resemblance of the symptoms to those which follow large doses of thyroid extract, by their contrast with those of myxœdema, and by the improvement which has occurred after partial excision of the hypertrophied gland. In colloid goitre the body probably reacts to a deficiency of iodine, by storing it in excess in the gland, while in Graves' disease the secretion is not stored but liberated into the system, so that the gland contains proportionately less iodine than the normal gland. Discussion has, however, taken place for a long time as to whether the disease is merely due to an over-production

of normal thyroxin or whether the thyroxin is altered in some way, for instance, by containing less iodine in the molecule than normal. The arguments in favour of the latter view are that the symptoms cannot be exactly reproduced by giving excess of thyroid to animals, *i.e.* there is no exophthalmos. Further, there are some cases in whom some symptoms of hyperthyroidism and myxœdema appear to co-exist; but this does not necessarily mean that the two diseases co-exist, because the hyperthyroidism symptoms may be a relic of a previous over-activity, which has now died down. On the whole, there is not sufficient evidence for the second view.

There is evidence that hyperthyroidism leads to increased activity of the suprarenals, and an increase of adrenin has been found in the blood. There is some evidence that these factors act by causing a mobilisation of the glycogen of the liver, which floods the circulation in the form of dextrose, and may lead to glycosuria. The mobilisation of glycogen may also be responsible for the increased respiratory exchange (oxygen intake and $CO_2$ output) that is a characteristic feature of the disease. This can be measured by the Douglas bag method, and it may be used as a means of diagnosis (*see* p. 558). The determination must be made with the patient at rest in bed in the morning on an empty stomach. The activity of the suprarenals will also stimulate the sympathetic, which may account for the exophthalmos, von Graefe's sign, tachycardia, and sweating. In Cannon's well-known experiment the phrenic nerve of the cat was sutured to the cervical sympathetic on the same side. The excitation of the sympathetic thus produced caused exophthalmos on the same side. The exophthalmos has been regarded as due to the stimulation of Müller's muscle, which lies in the membrane lining the orbit over the spheno-maxillary fissure; but in man the muscle is rudimentary, consisting of a few scattered fibres. Another possibility is that the eyeballs are pushed forward by œdema fluid, since œdema of the recti has been found, and then there follows a secondary deposit of fat.

The emaciation is due to the high respiratory exchange, combined with alimentary disturbances which hinder absorption. As the fat depôts become exhausted protein is drawn upon, so that there is also an abnormally high protein breakdown. In spite of the increased combustion, the body temperature is not raised, except occasionally, because, owing to profuse sweating, the loss of heat from the body is increased.

**Symptoms.**—The symptoms may come on suddenly, but generally they come on rather gradually, and, as a rule, the cardiac symptoms appear first, the protrusion of the eyeballs and the thyroid swelling not till some months later. Occasionally they may appear in a different order, or one or other of the three chief symptoms may be absent; but the circulatory trouble is the most constant. In the fully developed disease, the *heart* beats quickly and forcibly; the impulse is in the natural position, but it is felt over an abnormally large area; there is often a systolic murmur in the pulmonary area, and occasionally one at the apex. The carotids and large arteries pulsate with great force, and the patient feels the violence both of the cardiac beat and the arterial throbbings. The pulse reaches 120, 130 or 140 in the minute. The electrocardiogram is normal; but auricular fibrillation and very occasionally heart block are observed, especially if digitalis has been given in large doses. The patient suffers from shortness of breath on exertion in proportion to the cardiac disturbance. In course of time the heart may become dilated and hypertrophied. Variations in pulse rate are usually followed closely by corresponding variations in basal metabolism; but sometimes the pulse may still remain high, from damage to the heart, after the activity of the disease has died down, so that the basal metabolism gives a more certain picture of the activity of the gland.

The enlargement of the *thyroid body* is symmetrical, usually moderate in dimensions, and rarely equal to that of the larger endemic goitres. If the hand be placed over it, a thrill can be felt which is due to the movement

of blood in its dilated vessels, and a systolic murmur can be heard with the stethoscope.

The *prominence of the eyeballs* (exophthalmos, proptosis) is the most striking characteristic of the disease, and gives to the patient an unpleasant, terrified appearance. It affects both eyes, and may reach such a degree that the sclerotic is seen both above and below the cornea, and the eyelids are unable to meet. Even when the eyelids can be voluntarily closed, they may lie apart during sleep; and if the exophthalmos is extreme, there may be irritation and ulceration of the cornea as a result of exposure. Sometimes the prominence is only simulated in consequence of spasm or retraction of the upper eyelid (Stellwag's sign). In association with the exophthalmos there is a want of uniformity in the movements of the eyeball and the upper lid, so that, when the patient lowers the eyeball to look down, the upper eyelid is not depressed to a corresponding extent (von Graefe's sign). This is not present in every case, although it has sometimes been noticed even before the protrusion. On the other hand, when present it is of importance, as it does not occur in other kinds of exophthalmos. Weakness of the convergent muscles may be also present (Möbius' sign), and in some cases diplopia or definite paralysis of some or all of the ocular muscles. The pupil and accommodation are unaffected, and the ophthalmoscope reveals little beyond overfull and tortuous retinal veins.

A fourth very constant symptom is a more or less continuous fine *tremor* of the limbs, and even of the whole body.

The sufferer is out of health in many ways besides. She is languid, unfit for exertion, subject to nervousness, headache, vertigo, fits of temper or crying, irritable, restless, or hysterical, but not anæmic. In some cases melancholia, hallucinations and even mania have occurred; and tetany is an occasional event. The nervousness and cardiac action are increased by attention or by any excitement. The patient is generally thin, and may waste a good deal. A moderate degree of fever is occasionally present; and some patients show various pigmentary changes of the skin, such as moderate bronzing, chloasma or leucodermia. A subjective feeling of heat, flushing of the head and neck, sweating and intermittent albuminuria have been noticed in different cases. Patients are very subject to attacks of diarrhœa, sometimes with vomiting. Glycosuria sometimes occurs, owing to hyperglycæmia; that is, the patient cannot take the normal amount of sugar without its appearing in the urine. True diabetes mellitus sometimes follows, and patients with Graves' disease have sometimes died of diabetic coma. In many cases the blood sugar is normal and the sugar tolerance curve also normal (*see* p. 565). The symptoms are liable to aggravation from time to time.

**Diagnosis.**—There is difficulty in diagnosis in the early stages, before exophthalmos or swelling of the thyroid is noticeable. The disease must be distinguished from tuberculosis. A persistent tachycardia with a normal electrocardiogram is suggestive, especially if there is sweating as well. However, auricular fibrillation is common in toxic adenoma. The respiratory exchange may be measured (*see* p. 558). The Goetsch test has been much used in America: 0·5 c.c. of 1 in 1,000 adrenin is injected subcutaneously. A positive diagnosis is made, if there is a rise of ten beats in the pulse rate and 10 mm. in the systolic pressure during the first quarter of an hour, with symptoms of heart consciousness, asthenia, a feeling of dread, tremor, and pallor or flushing. It is safer to try a smaller dose first of all, as these patients are often very sensitive to adrenin. A rise of ten is probably too low, since this rise is often obtained in non-goitrous subjects. It should be thirty or forty (**18**).

**Prognosis.**—Campbell has recently followed up the histories of 127 cases treated medically at Guy's Hospital between 1908 and 1917. The results agreed very closely with a similar investigation carried out previously by Hale White. At the end of the period 8 per cent. were absolutely well; 30 per cent. were

almost well and able to do a full day's work, but one or two signs persisted slightly; 34 per cent. were much improved, being able to do light work; 13 per cent. were not improved or worse; 15 per cent. were dead from the disease. Operations have been carried out extensively within the last few years. The mortality in certain hands has been reduced to 2½ or 5 per cent., and the percentage of cases improved after the operation is larger than with medical treatment; but it is too early at present to say whether the improvement is maintained and to compare these results with those of medical treatment, mentioned above.

**Treatment.**—A cure can only be expected from prolonged treatment, and no drug can be said to have been uniformly successful. A simple satisfying diet should be employed. Alcohol and tobacco are likely to be prejudicial. Rest in bed for some weeks certainly has a beneficial effect on patients. In a series of fifty-one patients in which this was the only treatment, three-quarters had a basal metabolism under 18 per cent. (*see* p. 558) within six months (**4**). When the patients get up very mild exercise should be allowed; but plenty of rest should be taken.

There are two chief methods of reducing the activity of the thyroid, viz., (1) X-ray treatment or radium; (2) removal of the gland by operation preceded sometimes by ligation of one or two thyroid arteries. X-rays are usually employed at the beginning of the disease and operation may be considered later if no improvement results. X-ray treatment is not considered to make subsequent operative treatment more difficult. In true exophthalmic goitre great temporary benefit may be obtained by giving Lugol's solution (5 per cent. iodine in 7·5 per cent. potassium iodide solution), 15 drops once a day. The metabolism falls to normal in a week or two. This may be of great value as preoperative treatment. The result is not permanent. A similar result may be obtained with iodine in 10 per cent. alcoholic solution. The dose is 15 minims a day, and this is reduced to 3 or 4 minims after a week or two (**19**).

Exophthalmic goitre cases are tolerant to insulin, and some benefit may be produced by giving 15 or 20 units twice a day on a normal diet, possibly combined with parathyroid $\frac{1}{10}$ grain three times a day by mouth (**20**). Many other drugs have been tried, such as arsenic, convallaria, strophanthus, digitalis, belladonna, potassium bromide, ergot, and sodium salicylate, rodagen, which is a mixture of milk sugar, and the desiccated milk of goats deprived of their thyroids, and thyroidectin, which is the dried blood of thyroidectomised animals. It is doubtful whether these drugs are of any value. The application of ice to the thyroid is soothing. The patient should take plenty of rest, but exercise in moderation should be allowed.

## MYXŒDEMA

*(Cachexia strumipriva)*

Myxœdema and cretinism are the results of disease of the thyroid gland, causing deficiency of its secretion; cretinism is congenital; myxœdema arises sometimes in childhood (juvenile myxœdema), but more commonly in later life.

The prominent features of myxœdema are swelling of the skin and subcutaneous tissue, with dryness and roughness of the surface; pronounced mental failure, consisting of dulness, apathy, hebetude, slowness of speech or action; and atrophy or other destructive change of the thyroid gland.

**Ætiology.**—It is much more frequent in women than in men; and, in the majority of cases, the symptoms begin between the ages of thirty and fifty, though they have begun as early as eight and a half and as late as sixty-seven. Some indications of heredity have been observed, and it has been more often seen amongst the poorer classes. All types of hypothyroidism are common in districts where goitre is endemic.

**Morbid Anatomy.**—The changes in the skin are some nuclear proliferation

and development of connective tissue in the neighbourhood of the sweat glands, sebaceous glands, and hair follicles. Gelatinous and œdematous skin have only a few times been recorded, and the considerable amount of mucin discovered in one of Ord's early cases (to which the name *myxœdema* was given by him) has not been always found in others. There is a fair amount of subcutaneous fat.

The thyroid body is usually reduced to one-half or one-third of its normal size; it is pale, yellowish white or buff-coloured, tough or indurated, fibrous, or structureless. The gland consists mainly of fibrous tissue, with scattered groups of cells, the remains of the vesicles; and, finally, nothing but dense fibrous tissue is left. It is sometimes enlarged by new growth. The pituitary body has been enlarged, or enlarged and degenerated in some cases; and interstitial nephritis and cardiac hypertrophy have been found. Hypothyroidism may possibly lead to arterio-sclerosis.

**Symptoms.**—These are at first insidious, so that in most cases the disease has not been noticed till it has been well developed. The appearance is then characteristic: the face is broader than it was in health, and the nose, eyelids, and lips are thicker; its colour also is markedly yellow with a rather bright red patch on each cheek, and deep red or almost livid lips. The skin of the body generally is thickened, and the legs and feet have the appearance of slight œdema, although in many cases, but not in all, pitting is entirely absent. The shape of the hand also undergoes changes: it becomes broader opposite the heads of the metacarpal bones, and the fingers become thick and uniform in shape; this change has received the not very distinctive name of "spade-like." The feet are similarly affected. Perspiration is often deficient or absent, the skin dry and scaly, and the hair falls out, leaving only a thin covering on the head, or causing actual baldness of the scalp ("frontal alopecia" and "cassowary neck"), the outer third of the eyebrows ("eyebrow sign"), and eyelids. The nails are stunted and brittle. The mucous membranes show the same change; at any rate, the uvula and soft palate are swollen, and the tongue is large and thick; moreover, the teeth become carious or loose. The nervous system of the patient is the next thing that strikes one: she appears dull, apathetic, and slow in thought and movement. She speaks languidly and deliberately, as if the thick tongue mechanically interfered with articulation, but the slow movements of the eyes and the muscles of expression accompanying speech indicate that the neuro-muscular apparatus is also faulty. Articulation is thick or blurred, and the voice is monotonous. The movements of the body and limbs are correspondingly slow. On the mental side, memory is defective; the patient is often irritable, or suspicious, or dull and sleepy; and hallucinations, delusions, and convulsions have been noticed in a certain proportion of cases. Tetany sometimes occurs, as it does after the operative removal of the thyroid. The temperature is mostly subnormal, the patient suffers from cold very readily, and the hands and feet are often cold and blue.

The pulse is weak or slow; in advanced cases there is cardio-vascular disease and arterio-sclerosis, the urine being dilute and sometimes containing a little albumin. Examination of the blood shows anæmia, with reduction of the red cells to 3,000,000 or less, and of the hæmoglobin to a corresponding degree. The bowels are constipated. The tolerance for sugar is increased, so that large quantities, even up to 10 ounces in some cases, can be taken without any glucose appearing in the urine. In females the menses are usually deficient or absent, and menorrhagia is exceptional. Epistaxis, hæmorrhage from the gums and hæmorrhoids are not uncommon. Where the thyroid gland could be felt it has nearly always been described as small; but in the majority of cases there is some difficulty in satisfying oneself of the condition of the organ, or even of its actual presence; but the same, perhaps, may be said of persons who have not got myxœdema. The progress of the case is slow; patients are known to have had the disease ten years or more without material change. Nevertheless, it

undoubtedly shortens life ; the sufferers die from myocardial and arterio-sclerotic changes or from intercurrent diseases, such as pneumonia and bronchitis, or sink from general or nervous exhaustion.

**Diagnosis.**—When once the typical features have been recognised, the diagnosis of subsequent cases is generally easy. Myxœdema differs from Bright's disease not only in the absence of albumin, but in the absence of pitting, in the peculiar yellow and red colour of the face, and in the defective mental or neuro-muscular condition. The colour of the face is sometimes even suggestive of *mitral disease :* but the swelling is more than is common in cardiac affections, there is rarely a murmur, and the nervous condition is again distinctive.

**Treatment.**—Very striking improvement in the condition of patients with myxœdema has been obtained by supplying from animal sources the deficiency of the thyroid body. This has been done in two ways — by subcutaneous injection of a glycerine extract of sheep's thyroid (Murray) and by administering the pounded or minced thyroid or its extract internally (H. Mackenzie, Fox). The latter is the most convenient method, and thyroideum siccum (B.P.) made from the dried gland in doses of 3 to 5 grains in cachet or pill may be given once or oftener daily. Thyroid tablets made from the fresh gland are sold by the various commercial firms. It must be noted that 1 grain of dry thyroid is equivalent to 5 grains of the fresh gland. The beneficial effects may be seen in the quickening of the pulse, the rise of temperature, the growth of the hair, the diminution of the swelling of the face and hands, and the greater mental and vital energy. During this treatment also the skin desquamates over the body. The dose of thyroid must be continued indefinitely once or twice a week to secure a permanent result.

If the thyroid be given in excess, the patient suffers from headaches, palpitation, quick pulse, muscular tremors, pains in the limbs, thirst and feverishness, much depression, and nausea, vomiting or diarrhœa. The occurrence of these symptoms must be the signal to diminish the dose.

## CRETINISM

Cretinism is seen as an *endemic* disease in the mountainous parts of Europe (Switzerland, North Italy, and Savoy) and in Northern India (Chitral and Gilgit), where goitre also is extremely prevalent. The two conditions are often associated in the same individual ; indeed, many of these cretins are goitrous ; of 208 cretins in India, McCarrison found eighty-eight to be goitrous.

*Sporadic cretinism* occurs in other parts, *e.g.* in England ; in the subjects of it the thyroid is deficient, or there is a slight goitre.

**Pathology.**—The association of endemic cretinism with goitre in the individual and in the community, the absence of the thyroid in some cases, and the resemblance to myxœdema, show the relations of the disease to the thyroid gland. When endemic cretinism and goitre appear in the same individual, the former precedes the latter, and so is not due to it. The cretinism is due to the goitrous condition of one or both parents, and the defective thyroid function in the mother acts prejudicially in the fœtus. The lesion involves the parathyroid as well as the thyroid bodies.

**Symptoms.**—Cretinism is characterised by stunted growth, a large, broad head, thick features, wide separation of the eyes, flat nose, large mouth, much wrinkling of the coarse and rough skin even in early life, a narrow chest, full abdomen, crooked legs, and considerable mental deficiency, amounting to idiocy.

The characteristic features are generally noticed in the latter half of the first year of life ; walking is acquired very late, and the arrest of growth may be such that an adult cretin is not taller than a child of five or six. Puberty is long delayed, or the sexual functions are entirely absent. Speech is acquired very slowly or not at all, and some are deaf mutes and idiots. Nystagmus,

strabismus and spastic rigidity of the legs occur in some cases. Above the clavicles are often found subcutaneous tumours formed of masses of fat. Remarkable defects are present in the osseous system; the basi-occipital and basi-sphenoid are prematurely ossified; the long bones are permanently shorter than normal, the legs are bowed, and fibrous tissue from the periosteum grows in between the epiphysis and the shaft of the bone. The centres of ossification of the bones of the foot and wrist appear very late. Radiographic examination of these structures affords a valuable means of diagnosis.

**Treatment.**—In cretinism, as in myxœdema, thyroid extract has been used with much success. Under its influence children have grown rapidly, have lost the œdematous infiltration of the tissues, and have become more intelligent.

It must, however, be admitted that the child improves physically more readily than he improves mentally.

# DISEASES OF THE PARATHYROID GLANDS

The parathyroid glands are small bodies, generally four in number, situated near or within the substance of the thyroid, and consisting of groups of epithelioid cells in a fibrous meshwork, but without the vesicular arrangement and colloid contents of the thyroid; each measures 6 or 7 mm. by 3 or 4 mm. by 1½ or 2 mm., and weighs about ½ grain. Changes in these bodies—fatty degeneration, fibrosis, cyst and colloid formation—may occur.

Removal of the glands in animals is generally fatal; and death is preceded by a group of symptoms, called tetania parathyreopriva, similar to those of tetany, viz. fibrillary contractions, cramps, clonic contractions, and, finally, convulsive attacks; there is increased irritability of the peripheral nerves to mechanical and electrical stimuli. These phenomena are brought about by an influence exerted upon the lower neurons, since the symptoms are not stopped by the removal of the cerebral cortex. The symptoms are relieved by injection of parathyroid extract; but when they are due to total extirpation of parathyroid tissues, they can only be cured by the successful graft of a parathyroid from an animal of the same species. Noel Paton and Findlay have suggested that, since the guanidine in the urine and blood is increased in experimental and idiopathic tetany, this substance may cause the symptoms, the metabolism of guanidine being connected with the parathyroids. They point out that the symptoms of guanidine poisoning in animals are very similar to those of tetany. On the other hand, MacCallum has found that the symptoms are relieved by calcium salts. Recently parathyroid has been used in many varieties of ill health, infections, etc., in which the amount of ionised calcium in the serum has been found low. It is claimed that clinical improvement followed when it became raised, and that the parathyroid was responsible for these changes (**23**).

## TETANY

There is evidence pointing to disease of the parathyroids as being the cause of some forms of clinical tetany, but this is certainly not so in many cases.

**Ætiology.**—It occurs at all ages, but is especially frequent in infants and young adults. In children males are more often attacked than females; in older people the reverse holds good. In children rickets and diarrhœa are the commonest predisposing causes; in adults the causes are pregnancy and lactation, recovery from febrile diseases, the existence of dilatation of the stomach and intestinal obstruction and, in the case of the writer's, gastro-colic fistula with

free HCl in the stools. It has occurred after the operative removal of the thyroid body, and is probably due to simultaneous removal of all the parathyroids. McCarrison says it is common in women in the high valleys of Gilgit (Northern India), and that all such sufferers are goitrous. In these cases it may be due to associated parathyroid disease. Similar paroxysms have been seen as the result of ergotism and in association with osteomalacia, and from poisoning with sodium bicarbonate. Tetany is sometimes present in certain nervous diseases, particularly in epilepsy. Tetany also occurs in epidemics among workmen at certain seasons of the year, in particular European cities (Vienna, Heidelberg). It may be produced artificially by continued forced breathing, with washing out of $CO_2$ from the body (*acapnia*).

**Pathology.**—The similarity of the symptoms of tetania parathyreopriva in animals to clinical tetany has already been mentioned. The disease occurs in places where goitre is endemic and after complete removal of the thyroid in man. All this is compatible with the parathyroid hypothesis. The essential characteristic of the disease is over-excitability of the peripheral motor neuron from some toxin, possibly guanidine, so that the parathyroids may be related to the metabolism of this substance. However, the variety of conditions in which human tetany occurs makes it likely that there may be other causes. Recent experiments by MacCallum and others have shown that repeatedly washing out the stomach of a dog whose pylorus has been obstructed causes convulsions, which can be stopped by administering sodium chloride intravenously. It is thus possible to group together the tetany of dilated stomach, intestinal obstruction, and gastrocolic fistula as due to a loss of sodium chloride or hydrochloric acid from the body, and in these cases the plasma chloride has been found to be low and the plasma bicarbonate raised, and there is also an increase of urea in the blood. The increased plasma bicarbonate suggests that a condition of *alkalæmia* may be present (*see* p. 547). A similar condition may arise in these diseases without vomiting, and the suggestion has been made that in this case the hydrochloric acid passes into the tissues to neutralise some poison produced in the course of tissue breakdown. Similar alterations in the blood are found in acapnia and in sodium bicarbonate poisoning (*see* pp. 547, 549). In all these cases the tetany may be due to the alkalæmia, though this has been denied (**28**); or there may be some interference with the calcium metabolism of the blood, since the serum calcium is always low in tetany (**21**).

**Symptoms.**—There may be some discomfort or malaise, or stiffness in the arms, or tingling for some hours or days before the attack. Sometimes the paroxysm comes on suddenly without warning. The hands are then bent on the wrist; the fingers are flexed at the metacarpo-phalangeal joints, extended at the phalangeal joints, and pressed closely together with the thumbs bent into the palm of the hands, so that the fingers form a cone ("*main d'accoucheur*"). The elbows are slightly flexed, and the arms are adducted to the sides. Sometimes the four fingers are flexed into the hand, the wrists extended, and the elbows fully flexed. In the lower extremities, the foot is extended on the leg, the tarsus arched, and the toes flexed and crowded together. These are the characteristic contractions, and in most cases these alone occur. In very severe cases spasm affects the muscles of the abdomen, chest, face and tongue, as well as those of the back, causing slight opisthotonus, and of the eyes, causing strabismus. There may be some cramp-like pain in the parts affected; the back of the hands may be tumid, and the veins swollen. There may be sweating, flushing, and slight rise of temperature. The spasm ceases in from five to fifteen minutes, or it lasts one, two or more hours; it gradually subsides, and recurs after an interval of some hours or days.

In the intervals the nerves and muscles show an increased susceptibility to mechanical irritation (Chvostek). Percussion of the nerves causes contractions in the corresponding muscles, and this is well shown in the face by percussing

midway between the zygoma and the angle of the mouth. Stroking the face from above downwards causes contraction of the muscles one after the other. Trousseau first showed that in the intervals a fresh paroxysm could be brought on in a few minutes by firmly grasping the arms, or by pressure on the nerves and arteries. The motor nerves also are unduly susceptible to faradism and still more to galvanism (Erb). Hyperexcitability, suggestive of tetany, exists if galvanic currents below 5 milliamperes produce a contraction on cathodal closing or anodal opening (*see* p. 732).

But there is not always an interval between the paroxysms. In infants a continuous spasm is more common, and in adults the spasm may not entirely relax, so that this form is called *remittent*, while the form with complete intervals is called *intermittent*. In *latent* tetany, which is fairly common, there are no spontaneous attacks of spasm.

In rachitic children the undue excitability of the nervous system is represented not only by tetany, but also by laryngismus stridulus, and by convulsions.

The disease lasts from a few days to a few weeks, and recovery is the rule. Occasionally some weakness of the legs remains for a short time after recovery, and muscular atrophy and fibrillary tremors have been seen. But death may occur from exhaustion when the paroxysms are severe, or from pneumonia as a result of interference with respiration, or in infants from the diarrhœa which caused the disease.

**Diagnosis.**—The distribution of the spasms—namely, their occurrence chiefly in the hands and arms—distinguishes it from *tetanus*. *Hysterical* contractions may assume the form of tetany; they are generally unilateral, and are associated with other hysterical conditions. Latent tetany can be diagnosed by Chvostek's and Trousseau's signs, and by the presence of certain symptoms commonly associated with it, viz., defects in the enamel of the teeth, perinuclear cataracts, dystrophies of the hair and nails and recurring conjunctivitis.

**Treatment.**—Calcium chloride (15–20 grains, in the case of infants) is given by mouth every four hours (**22**). The chlorine passes into the circulation to supply the deficiency of chloride, while the calcium is excreted by the bowels. Ammonium chloride in large doses may also be tried. The ammonia is converted into urea, so that here also the hydrochloric acid may be utilised. These drugs may also be given per rectum. The predisposing condition of the patient should be as far as possible removed. Thus gastric dilatation should be treated by surgical operation (gastro-jejunostomy); in children, diarrhœa should be treated, and rickets met by cod-liver oil, iron, suitable diet, etc.; women should give up nursing their children, and should take iron and other tonics. Chloral, Indian hemp, Calabar bean and morphia have also been given with more or less success. Chloroform inhalation stops the spasms for a while. Galvanism with the anode on the nerves may also be tried. Thyroid extract, parathyroid extract and bromides have been given. A case of tetany caused by the removal of part of the thyroid recovered after the use of thyroid extract, and of calcium lactate.

# DISEASES OF THE THYMUS GLAND

The thymus gland weighs from 7 to 10 grammes at birth, increases proportionately to the growth of the child until two years of age, and diminishes very little until the age of eight, ten or twelve, but about the age of twenty has almost disappeared. Even in adult life, however, its remnant can be found if carefully sought, consisting of fibrous and fatty tissue, islets of lymphocytes, and a few Hassall's corpuscles (Dudgeon).

Its morbid changes are atrophy in children and hypertrophy or enlargement, or persistence with enlargement, from various causes in adult life.

In atrophy, which is associated with marasmus or tuberculous and other chronic wasting diseases in children, the organ is reduced in size, and may weigh only 3 or 2 grammes, or less than 1 gramme. It is often white, soft, and œdematous; but the change is chiefly fibrous, there is an absence of the creamy, pus-like, but really lymphocytic, fluid found in the interior in health, the lymphoid tissue is scanty, and Hassall's corpuscles are degenerated and calcareous.

In acute disease in children hæmorrhages, some fibrosis, and an increase of eosinophil corpuscles are often found.

Persistence, with enlargement, of the thymus occurs in a number of diseases, of which the most important are leukæmia, especially the lymphatic form, Hodgkin's disease, exophthalmic goitre and myxœdema, acromegaly, Addison's disease, myasthenia gravis, epilepsy, anæmia, rickets, scurvy, and the condition known as status thymico-lymphaticus. In the last condition it may attain a large size, weighing often from ½ ounce to 1½ ounces (15 to 45 grammes); but it has been known to weigh 6 ounces, and in a case of lymphatic leukæmia described by Sir Frederick Taylor more than a pound (586 grammes). The enlarged thymus in lymphatism has often a normal structure, except for the presence of numerous eosinophil corpuscles, of which the great majority have a large, round nucleus, while eosinophil cells having a polymorphic type of nucleus are small in number (Dudgeon).

The gland may be converted into a fibro-caseous mass as a result of tubercle, and it may be the subject of new growth.

## STATUS THYMICO-LYMPHATICUS

(*Status Lymphaticus, Thymic Asthma, Mors Thymica*)

Clinical syndromes associated with enlarged thymus have long been generally recognised, and though in certain cases mechanical pressure by the enlarged gland on the trachea may account for some of the symptoms, it seems probable that many of them are due to the production of a hormone by the thymus in excessive amounts (*hyperthymismus*).

**Morbid Anatomy.**—In children there is usually found a general hyperplasia of the lymphatic structures throughout the body, enlargement of the tonsils and adenoids and the follicles at the back of the tongue, the lymph glands in the neck, axillæ and groin, and the solitary follicles of the intestine; and it may be the thyroid gland, spleen, and liver. Hyperplasia of the red bone marrow, and hypoplasia of the heart, and narrowing of the aorta have been found in some cases.

**Symptoms.**—In addition to the general increase of lymphoid tissue just described, the blood shows a pronounced lymphocytosis; the skin has a pasty look; the middle incisor teeth may be large and the lateral ones small. Enlargement of the thymus will be suggested by dulness over the manubrium sterni contrasted with resonance over the corpus. The dulness may extend on each side, particularly to the left. X-rays may show a widening of the median shadow, and in the oblique position the space between the trachea, aortic arch and manubrium will be filled by a shadow, which does not pulsate and does not move on swallowing, and so can be differentiated from aneurysm and thyroid. The trachea may be seen to be displaced. A shadow between the trachea and vertebræ will be due to enlarged glands.

Such children suffer from seizures of asphyxiation, though the parents may think they are holding their breath. There is cyanosis, convulsive movements and unconsciousness. Though usually the child recovers, occasionally death ensues. Such attacks are most common between the sixth and twelfth month of life. They must be distinguished from attacks of laryngismus stridulus (*see* p. 211).

In young adults the hair on the body is scanty; it tends to be distributed as in the opposite sex. There is hypoplasia of the external genital organs.

*Mors thymica.*—Children and young adults with status thymico-lymphaticus are subject to sudden death without obvious adequate cause, during sleep, while taking a bath, or being punished, after an injection of antitoxin or during anæsthesia, etc. Sudden death of this nature together with the attacks of asphyxiation in children have been attributed to an unstable condition, or "endocrine misbalance" from the excessive thymic secretion. These conditions are certainly rather similar to certain seizures which occur in the allergic state (*see* p. 224) and which have been loosely attributed to anaphylaxis.

Sudden death, however, occurs in children, the subject of such pathological changes, independent of anæsthesia; and its occurrence in either case is difficult to explain. It is attributed by some to pressure of the thymus on the trachea, by others to toxæmia, to a defective condition of the blood, to intravascular clotting, to diminished powers of resistance, or to reflex nervous influence.

H. C. Cameron proposes as an explanation of this condition in children that the lymphatic overgrowth is due to the prolonged irritation of catarrh of the different mucous membranes (nasal, buccal, bronchial and gastro-intestinal) and of the skin, which these children so often present. He also calls attention to the watery condition of their tissues, which gives these children a fictitious appearance of plumpness and health, but which rapidly disappears in febrile conditions and leaves them thin and wasted. He looks upon this as the result of excessive carbohydrate diet in bread and sugar, and advises an alteration of the regimen as a means of removing successively the watery state of the tissues, the related catarrh, and finally the lymphatic enlargements.

The **Treatment** of status thymico-lymphaticus has been attempted with the Röntgen rays applied to the thymus, apparently with some success, and by operations upon the thymus, raising it by sutures or removing it altogether. Cameron advises as diet skim milk, meat, fish, eggs, green vegetables and fruit, while the carbohydrates are reduced to a minimum. Cod-liver oil and syrup of iodide of iron are often beneficial.

# DISEASES OF THE SUPRARENAL CAPSULES

The suprarenal capsules consist of two parts:—

1. The *cortex* (*interrenal system*) is derived from mesodermal cells closely related to the cells of the genital organs. It contains cholesterin esters and lecithin in large quantity, and its yellow colour is due to these. It is about 90 per cent. of the whole gland in weight, and becomes rather larger during pregnancy. The lipoids of the cortex disappear within a few days as the result of an acute febrile illness of an infective character. On the other hand, they are not discharged in cases of inanition, *e.g.* in malignant disease. In these respects they offer a marked contrast to the ordinary body fat. Very little is known about their function, but it is probably quite distinct from the function of the medulla; although in man both parts of the gland have the same blood supply, in many animals the two parts are quite separate. The cortex may have some relation to the growth of the brain, but there is a very definite relationship with sex. In rare cases, where the cortex is hypertrophied, there is precocious development of the sexual organs, with an increase of male characteristics. More commonly these symptoms are caused by carcinoma of the cortex (*q.v.*). Conversely, hypoplasia of the adrenals has sometimes been seen in cases of disappearance or original absence of hair from the genitals and hypoplasia of the genital organs.

2. The *medulla* is derived from the same cells as the ganglion cells of the sympathetic nervous system. It has a characteristic staining reaction with bichromate, and so is called a *chromaffine body*. It elaborates a substance, *adrenin* or *epinephrin*, which has been prepared synthetically, and is chemically ortho-dioxyphenyl-ethanol-methylamine. This substance is poured out into the circulation on stimulation of the splanchnic nerves. It has a powerful action on all sympathetic nerve endings. Under normal circumstances this liberation into the circulation occurs when the powerful emotions of excitement, pain, fear and rage are produced; the processes associated with digestion and reproduction are inhibited; the animal becomes prepared for battle or for flight. The pupils are dilated; the skin becomes pale; the hairs stand on end; the heart is accelerated. The sugar in the blood is increased by action on the liver; the skeletal muscles show increased power and are less readily fatigued. The coagulability of the blood is increased, which is of advantage in case the animal is wounded. There can be little doubt that the proverbial strength of the madman is due to the outpouring of adrenin, while his keepers, who experience his power, are not actuated by the same strong emotions.

But the liberation of adrenin into the circulation may have something to do with the normal tone of the vasomotor system. This is shown by extirpating the glands in animals. Such an operation is fatal after about a week; but before death takes place the blood pressure becomes very low—about 20 or 30 mm. of mercury—while the injection of adrenin may restore it to some extent. This experiment probably has its pathological counterpart in Addison's disease. In dementia præcox it has been found that the medulla is deficient and shows histological changes (**24**).

## ACUTE INADEQUACY

In acute infectious diseases, such as diphtheria, there may be cloudy swelling, necrosis, and infiltration with leucocytes. In the same disease hæmorrhages and acute hæmorrhagic necrosis may take place in the medullary portion of the gland, and the same occurs in enteric fever, pneumonia, erysipelas and purpura.

In cholera and diphtheria it has been found that the amount of adrenalin yielded by the medulla is much diminished; and it has been shown by T. R. Elliott that in persons dying slowly with general distress the adrenalin content is from 0·2 milligramme to 2·3 milligrammes, whereas normally and in cases of sudden death the amount is about 4·5 milligrammes. Batelli estimates the normal adrenalin content as 1 part in 1,000 of the whole gland.

Some of the more acute infections and hæmorrhagic necrosis are responsible for fatal symptoms, of short duration, which may be reasonably attributed to acute interference with the secretory functions of this gland. Cases of this kind have been classified as follows: sudden onset of epigastric pain and tenderness, followed by abdominal distension and death within a few days; profound asthenia, ending with death in a few days; cases with convulsions, coma and delirium, or a typhoid stage; cases of sudden death, in which hæmorrhage is generally found; cases with a purpuric eruption, or with hæmorrhages into the abdominal viscera.

## ADDISON'S DISEASE

This disease was first described by Dr. Thomas Addison in 1855.

**Ætiology.**—Most cases occur in early adult life, or middle age, though young children and elderly persons are not exempt; and it affects males more frequently than females. It appears to be a good deal more common amongst the poor and labouring classes than amongst the well-to-do As to its immediate causation, it must be noted that in a certain number of cases it occurs in connection with phthisis or other tuberculous disease; in other cases it seems to

follow upon inflammatory lesions in parts adjacent to the capsules—for instance, caries of the dorsal or lumbar vertebræ, psoas abscess or other suppuration in this neighbourhood. But in many cases it is a primary tuberculous infection of the gland. In a small number of instances, in which the symptoms of Addison's disease have been undoubtedly present, the capsules have been atrophied to an extreme degree without evidence of previous tubercle. In a few cases the glands have been attacked by growth, or there has been thrombosis of vessels with extravasation of blood. In yet other cases the semilunar ganglia have been destroyed by growths, etc., the suprarenal capsule remaining intact.

**Morbid Anatomy.**— The change of colour in the skin is due to pigment which is deposited, for the most part, in the deepest layers of the epidermis, a condition similar to that which obtains naturally in the skin of the negro. A few pigment granules are also scattered in the upper parts of the papillary layer of the true skin.

The suprarenal capsules usually show obvious tuberculous infiltration, presenting on section a combination of translucent greyish or greenish-grey tissue and opaque yellow cheesy substance. Sometimes the caseous matter has softened down into a cavity containing pus. But another important feature must be noticed—namely, that the chronic inflammation of the suprarenal body leads to thickening of the connective tissue around it and adhesion to neighbouring structures, and that the solar plexus, the semilunar ganglion, and the terminations of the phrenic and pneumogastric nerves are often involved in the disease either by compression or acute inflammation. Enlarged lymphatic glands are also found in the surrounding connective tissue as well as in the mesentery, and behind the peritoneum. The solitary follicles of the small and large intestines are often swollen, and the mucous membrane of the stomach may be "mammillated" from an overgrowth of lymphoid tissue between the gastric tubules. The serous membranes are sometimes pigmented.

In a case recently examined histologically the remaining endocrine glands showed striking changes. The pituitary showed signs of activity; the thyroid showed inactivity, resembling the structure of colloid goitre, which is of interest considering the supposed action of the thyroid on the suprarenal (*see* p. 579). There was atrophy of the seminiferous and interstitial tissue of the testis (**25**).

**Pathology.**—Addison's disease is probably to be regarded as due to destruction of the medulla of the suprarenal. In a case recently examined at Guy's Hospital, in which both glands were atrophied, histological examination showed the absence of chromaffine tissue, while cortical tissue was present.

The absence of adrenin accounts for the low pressure and general asthenia of Addison's disease, and possibly the gastro-intestinal disturbances. The pigmentation is less easily explained. The basal metabolism has been found below normal (**4**).

**Symptoms.**—The cardinal symptoms are debility, low blood pressure, vomiting and pigmentation. The onset is generally insidious, and the patient gradually suffers from weakness, depression, languor and indisposition for exertion. There may be pains in the loins, hypochondrium or epigastrium, and tenderness on pressure in one or other hypochondriac region. The heart's action is very weak, and there are faintness and giddiness on rising in bed, or breathlessness and palpitation on exertion. The pulse beats from eighty to ninety in the minute, and is small and feeble; the blood pressure is very low, measuring as little as 80 or 60 mm. of mercury. Appetite is generally deficient, and nausea, retching and vomiting are important features of the disease. A peculiar *discoloration of the skin* is the symptom which has attracted most attention, but which it is important to remember may be entirely absent. This symptom may be noticed coincidently with the above general symptoms; it may develop before them, or it may occur several months after they are pronounced. It is possible, in this last class of cases, that, if the general symptoms

are very severe, they may be fatal before the skin is affected; and thus the occasional absence of pigmentation in Addison's disease of the suprarenal capsules is explained. The pigmentation or *bronzing* is, in its lighter shades, dusky or yellowish brown, sometimes of olive or greenish-brown hue. In its more pronounced form the skin has a rich brown colour, like that of a mulatto. The pigment is uniformly distributed over different parts of the body—that is to say, large areas are discoloured, the darker tints gradually shading off into the lighter or into the natural colour of the skin. The change usually invades, first, the parts of the skin which are naturally exposed, such as the face, neck and the backs of the hands and fingers, but not the scalp or the lip under the moustache; secondly, parts which are naturally more deeply pigmented than others, such as the axillæ, penis, scrotum and areolæ of the nipples; and thirdly, seats of pressure or slight injury, such as the marks of garters and waistbands in women, and places where blisters and plasters have been applied. But the scars of wounds destroying the skin remain white, and are bordered by a deep layer of pigment. Sometimes there is darkening of the depressed lines in the palms. On the darkened parts of the skin may be seen small black specks, like moles or freckles. In advanced conditions the whole body may be covered by the pigmentation; but, as a rule, one must be prepared to recognise the disease, and, indeed, many patients die, before this stage is reached. The pigmentation is not limited to the skin. A bluish-black line may often be seen on the inner side of each lip running along the mucous membrane, parallel to the line of junction with the skin; and other more irregular patches may occur on the mucous membrane of the cheek and on the side of the tongue. Some of these seem to be determined by the presence and consequent irritation of carious teeth. The temperature is, as a rule, normal; the urine is of medium colour and specific gravity, and free from albumin. Although weak, the patient is not necessarily emaciated, nor anæmic; even a considerable layer of subcutaneous fat may persist to the end.

The course of the disease is very variable. It is often marked by exacerbations and remissions, periods of severe illness, which confine the patient to bed, alternating with times of comparative health; but after each fresh aggravation of the disease the patient is left decidedly worse than he was before it. The duration varies from a few months to six or seven years. Death takes place mostly by asthenia, the patient getting gradually weaker and lapsing into a drowsy or semi-comatose condition, with increasing feebleness of pulse. Delirium and convulsions occasionally close the scene. In some cases the general symptoms and a very slight pigmentation have been noticed only for some months, when extreme prostration has ensued and carried off the patient in a few weeks.

**Diagnosis.**—The mistakes most likely to be made are: (1) to take some other discoloration for that of Addison's disease; (2) to fail in recognising the symptoms when the pigmentation is slight or absent. The discolorations likely to be mistaken for it are slight *jaundice*, which is distinguished by the yellow tinge of the conjunctiva; *phthiriasis*, to be recognised by the scars, blood crusts, scratches, pediculi, the limitation of the colour to parts which can be reached by the finger nails, and the entire freedom of the face; the sallow or earthy tints of *malaria* and of *phthisis*; *chloasma uterinum* in women; and *tinea versicolor*. In early stages, without much darkening, the apparently causeless weakness, with small, feeble pulse, and sickness, are the diagnostic features; in some such cases the application of linseed-meal or mustard poultices has been followed by an unusual amount of pigmentation locally. It has been stated that suprarenal extract given by the mouth raises the blood pressure of the sufferer from Addison's disease, but does not affect that of persons in health. The extract should be given in from 3 to 10-grain doses three times daily for three days, and the blood pressure should be accurately estimated before and afterwards (O. Leyton). The tests for tubercle may also be applied.

**Treatment.**—This must be, on the whole, tonic. Sickness must be met by effervescing salines, bismuth, iodine, etc. Iron, arsenic, and strychnine are the most suitable tonics. The internal administration of a suprarenal extract or adrenin (30 minims of the 1 in 1,000 solution) has given some good results, at least temporarily restoring the strength, but without removing the pigmentation. One or two fresh glands may also be given daily. In a case where some healthy human gland was transplanted into a patient the treatment was successful for some months, but the piece then became absorbed.

### TUMOURS OF THE SUPRARENAL CAPSULES

The tumours affecting these glands are adenoma, sarcoma, carcinoma and neuroblastoma. Sarcoma is very rare, and occurs only in adults. Neuroblastoma occurs in children, and arises from the medulla of the gland. In the past it has been called sarcoma. It is a malignant growth, closely resembling a small-celled sarcoma, but it has rosettes which are characteristic of neoplasms of the central nervous system. It readily gives rise to secondary deposits in the bones. It may form a large mass, which may be mistaken for a renal tumour; it is likely to be fatal, unless it can be removed early. Carcinoma is rarely primary, but generally part of widespread secondary lesions. When primary, it is a small-celled carcinoma, which very readily gives rise to hæmorrhage and necrosis. The cells have a tubular or alveolar arrangement, or are grouped radially round blood vessels; and there is a general resemblance to the suprarenal cortex. Metastasis may occur in different parts of the body. When present, the growth gives rise to characteristic symptoms due to the activity of the tumour cells (hyper-interrenopathy). The same symptoms are observed with simple hypertrophy of the cortex. If the disorder begins in intrauterine life *female pseudohermaphroditism* is seen; *i e.* the individual is in reality female, as ovaries are present, but has external male characters. This condition is congenital, and is due usually to bilateral cortical hyperplasia. When the disease begins soon after birth, it produces the condition known as *premature puberty*. The children become obese. In male children there is precocious and excessive sexual development. Great muscular strength is developed, producing what is known as the *infantile Hercules* type; there are hair on the face and increased sexual functions. Similar changes are noted in girls, and there may be early menstruation. In later life hyper-interrenopathy leads to *adult hirsutism* or *virilism*. In women certain male characteristics are seen. Hair grows on the face, and the hair elsewhere on the body is increased; there is absence of menstruation and of mammary development; there is hypertrophy of the clitoris and deepening of the voice. There is increased bodily strength and mental symptoms suggestive of masculinity, such as aggressiveness and egotism, appear.

Certain other changes in the suprarenal capsules may be mentioned. Inflammation and *abscess* from proximity to suppurating foci, hæmorrhage from injury, *lardaceous* change in common with other organs, miliary tubercles in general tuberculosis, and rarely syphilitic gumma, are among the other pathological conditions which may be found.

## DISEASES OF THE PITUITARY GLAND

This gland, often called the hypophysis, consists of three parts:—(1) the *pars anterior* or *glandulosa*, derived from the buccal ectoderm and containing colloid cysts; (2) the *pars posterior* or *nervosa*, consisting of neuroglia fibres and cells;

(3) the *pars intermedia*, glandular in structure, lying between the first two. The colloid from the pars anterior (anterior lobe) is discharged through the pars posterior (posterior lobe) into the third ventricle, and is found in the cerebrospinal fluid.

The pars anterior exercises a profound influence on growth, excessive activity (*hyperpituitarism*) leading to *gigantism* in early life from overgrowth of the long bones, and in adult life to *acromegaly*, when the epiphyses of the long bones have finally become ossified. Deficiency in the secretion of the pars anterior also leads to characteristic symptoms (*hypopituitarism*). In many cases a pituitary gland which has shown signs of over-activity in the past, with the production of acromegaly or gigantism, becomes inactive owing to degeneration of its substance, so that symptoms of hypopituitarism are grafted on to the permanent bony changes due to hyperpituitarism. Such a condition is known as *dyspituitarism* and is comparable to the rare cases of myxœdema that follow exophthalmic goître.

The pars posterior, or more probably the pars intermedia, gives rise to a hormone which is sometimes known as *pituitrin:* it is possible that this substance is normally discharged with the colloid into the cerebrospinal fluid. Pituitrin in the anæsthetised animal has a direct action on muscle, particularly of the uterus, bladder and intestine; it causes a rise of blood pressure with diuresis, and an increased flow of cerebrospinal fluid and milk. There are important differences in the action of pituitrin in an unanæsthetised man. The volume of urine is diminished, and the blood pressure is not raised.

Over-activity of the pituitary (possibly the pars intermedia or posterior lobe) may cause glycosuria, which is usually quite mild. However, true diabetes may occur with ketosis, and in one such case the diabetes was greatly improved after removing the tumour (**26**). This combination of pituitary over-activity and diabetes is of great interest, since it has been found that pituitrin neutralises the action of insulin. Under-activity of the pituitary may cause adiposity and other metabolic changes, to be described later.

**Morbid Anatomy.**—According to Cushing, disease of the pituitary is of two kinds. (1) There are primary lesions in the gland itself, *i.e.* simple hypertrophy, or adenomatous enlargement, which may become cystic or degenerate. These changes were present in twenty-three of his cases, and gave rise to symptoms of hyper-, hypo- or dyspituitarism; carcinoma, fibroma, sarcoma and glioma also occur. (2) There are extrapituitary tumours, which are developmental relics from pharyngeal or infundibular diverticula, teratomas, infundibular cysts, and meningeal tumours. These press on the gland, producing hypopituitarism. They were present in six of Cushing's cases. But the gland may be affected similarly by lesions at a distance, *e.g.* subtentorial tumours. Internal hydrocephalus sometimes causes hypopituitarism, as shown by the onset of adiposity, and it is supposed in these cases that the colloid material cannot get from the posterior lobe into the cerebrospinal fluid.

**Symptoms.**—These are extremely varied in different cases, because, in addition to those due to over- or under-activity of different parts of the gland, there are the so-called "neighbourhood symptoms," caused by pressure of the tumour on surrounding structures. Sometimes these symptoms are the only ones present; in other cases they are associated with glandular symptoms, and in other cases glandular symptoms are present alone. Again, hypopituitarism may be associated with symptoms of a distant cerebral lesion, or a pituitary tumour may very occasionally cause the symptoms of general intracranial pressure, like any other cerebral tumour.

**Neighbourhood Symptoms.**—The most important symptoms produced locally by tumours in this region are those due to pressure upon the fibres of the optic nerves at the point of their decussation. As the tumour encroaches upon the posterior border of the optic chiasma, the fibres first affected are those coming

from the central part of the two nasal halves of the retinæ (*see* Fig. 69, p. 712), so that the earliest sign of visual disturbance is the appearance of a paracentral scotoma on the temporal side of the central point of each visual field. These later develop into a complete bitemporal hemianopia if the tumour continues to increase in size. These signs are accompanied by optic atrophy, so that in the later stages ophthalmoscopic examination shows the discs to be of a clear pallor, with clean-cut edges, standing out against the normal pink retina. The changes in the visual fields here described are those found in the case of a tumour occupying the mid-line; if the growth extends to the right or left, the corresponding optic tract is involved, producing a left or right homonymous hemianopia (*see* Fig. 69, p. 712). In the terminal stages complete blindness results from pressure atrophy of all the nerve fibres. The headache in these tumours is somewhat characteristic, being referred to the temporal region on both sides, and being of a peculiar boring nature.

Other valuable evidence may be obtained from an X-ray photograph of the sella turcica. In the case of a tumour originating in the pituitary gland (usually an adenoma of the pars anterior) the floor and walls of the sella become eroded, and the clinoid processes become spread apart and eventually destroyed, changes which are easily seen on the X-ray plate when compared with a photograph of the normal skull. In other cases enlargement of the sella is associated with bony overgrowth round it. It should be remembered that the outside limits of the normal sella are 1·5 cm. long and 0·9 cm. deep.

If the tumour extends beyond the bounds of the pituitary fossa it produces other signs of localising value as it encroaches upon the walls of the interpeduncular space. Of these the most important is paralysis of one or other of the third nerves, with resultant ptosis, inactive dilated pupil and palsy of all ocular movements except outwards, and downwards and outwards. Pressure on the crura cerebri will give rise to signs of injury to pyramidal fibres, *i.e.* loss of abdominal reflexes and an extensor plantar response on the opposite side to the crus involved. There is, moreover, frequently a slight degree of exophthalmos present, due probably to pressure upon the cavernous sinus.

The fact that there is no constant parallelism between the degree of optic atrophy and the distortion of the sella turcica depends upon anatomical variations in the direction of the growth. Tumours arising, not from the pituitary gland, but from the brain or meninges in the suprasellar region, may produce a symptom complex almost indistinguishable from that caused by a pituitary tumour proper, but are more likely to involve the third nerves and pyramidal fibres early, and give rise to distortion of the sella turcica only at a later stage.

Other neighbourhood symptoms are so-called "uncinate seizures," which are epileptic fits preceded by a gustatory or olfactory aura. There may be photophobia and pain in the eyes and tenderness on pressure. Mental symptoms may supervene. There may be an excessive discharge of mucus from the nasopharynx if the tumour spreads downwards, and there may be attacks of *rhinorrhœa*, which is a discharge of clear fluid and blood, if a passage is made between the third ventricle and the nasopharynx.

Separation of the malar prominences, so-called *maxillary prognathism*, may occur, and is supposed to be due to separation of the two halves of the sphenoid owing to the tumour.

**Glandular Symptoms.**—*Acromegaly.*—This is due to hyperpituitarism. The disease was described by Marie in 1880. It usually occurs in adolescence or early adult life. There is enlargement of the bones of the extremities (hands and feet) and of the face (ἄκρον, an extremity; μέγας, large). The phalanges are thickened, and there are bony exostoses. The jaw is enlarged and is undershot (*mandibular prognathism*). The teeth become separated. The soft parts are also thickened; the papillæ of the skin are hypertrophied; the nails are broad, coarse and ribbed; the skin is thick and greasy; and the lips and nose and ears

and tongue are coarse and large. The thickening of the fingers gives a characteristic appearance to the hand—*type en large* (Marie). There may also be kyphosis. Acromegaly with active hyperpituitarism is associated with a lowered sugar tolerance, so that there is hyperglycæmia and glycosuria if a sugar tolerance test is carried out. If dyspituitarism supervenes, the sugar tolerance is increased. There is often low intellectual development.

If hyperpituitarism occurs during childhood, the bones become longer than normal (gigantism). The fingers also become longer than normally, and the type of hand that results has been called the *type en long* by Marie.

*Hypopituitarism.*—Anterior lobe deficiency produces smooth, dry, transparent skin and sparse hair. The shape of the body approximates to the feminine type. The nails are small, without crescents. The fingers are tapering (*acromicria*, the converse of acromegaly), and there may be pigmentation. In adults loss of sexual desire in the male and amenorrhœa in the female may be early and important signs. Posterior lobe deficiency, or more probably deficiency of the pars intermedia, causes asthenia, drowsiness, increased sugar tolerance and subnormal temperature from lowered metabolism, and especially adiposity, and in some cases polyuria. The combination of adiposity and infantilism met with in children—*Fröhlich's syndrome*, or *hypophyseal dystrophia adiposo-genitalis*—is due to hypopituitarism. The fat is most noticeable on the abdomen, buttocks, and proximal portions of the extremities. Dwarfism may result. The genital organs remain in an infantile state, and the skin is pale, thin, soft, and smooth. In adults cases of extreme obesity may be due to pituitary disease. *Adiposis dolorosa*, or *Dercum's disease*, where the deposits of fat are painful, is probably due to the same cause (*see* p. 599). Epilepsy is also associated with hypopituitarism.

**Diagnosis.**—This depends upon the recognition of neighbourhood and glandular symptoms. Of the former the earliest signs are, as a rule, headache and visual disturbance, though it is important to realise that a patient may present considerable defects in his visual fields on perimetry without himself being aware of their existence. The ophthalmoscopic and X-ray examinations afford the most direct evidence of pituitary disease.

Of the glandular symptoms the hypopituitary group is that most frequently met with. Arrest of development (infantilism) in children, amenorrhœa and adiposity in the female, and lack of vigour in the male, are perhaps the commonest symptoms. The signs of hyperpituitarism, while less usual, are more obvious.

**Prognosis.**—This is bad, since the tumour usually progresses, although slowly. The operative mortality of sellar decompression is about 12 per cent. (Cushing).

**Treatment.**—In cases of hypopituitarism benefit has been obtained by administering the anterior lobe in tablets by the mouth. Thyroid has also been given. Pituitrin must be given subcutaneously, since it is destroyed by the alimentary canal. The progress of treatment can be estimated from the sugar tolerance. Where a pituitary tumour is growing X-rays may be applied through the anterior nares and at the side over the temple. For the relief of pressure sellar decompression by operation has been successful, with or without removal of the tumour.

## DIABETES INSIPIDUS

The term diabetes (*διαβαίνω*—I go through) is the equivalent of polyuria, or excessive secretion of urine. Polyuria may be caused by disorders of the kidney, by increased blood pressure, by the presence of sugar in the urine in ordinary diabetes, or diabetes mellitus, and temporarily in some disorders of the brain, especially hysteria and migraine. Diabetes insipidus is a persistent polyuria not traceable to any of the above conditions.

**Ætiology.**—Diabetes insipidus is a comparatively rare complaint. It occurs mostly in early adult and middle age, but sometimes in quite young children, and it is more frequent in males than in females. It cannot always be traced to a definite cause, but it has occasionally followed upon blows or injuries to the head, emotional disturbance or convalescences from acute diseases. Family predisposition has also been recorded, the disease being handed down from parents to children.

**Pathology.**—Recent investigation has shown that diabetes insipidus is due to injury or disease of the pituitary gland, probably the posterior lobe. There is no recorded case in which disease of the gland has been definitely excluded, and in many cases implication of the gland has been demonstrated. Further, subcutaneous or intravenous injections of pituitrin (extract of the posterior lobe) are specific for the disease, the urine being at once reduced to the normal volume, and the patients get complete relief from their symptoms. In the anæsthetised animal injections of pituitrin cause an increased flow of urine following a rise of blood pressure. However, Motzfeld has shown that in the unanæsthetised animal the volume of urine is diminished, and this diminution is more striking if an artificial polyuria has been produced by previously filling the stomach with water. The action of the drug is stopped if the splanchnic or renal nerves are divided.

These experiments indicate the cause of the disease. Normally the secretion of urine is controlled by the vasomotor nerves to the kidney, which are activated by secretion from the posterior lobe of the pituitary gland. In diabetes insipidus this secretion is absent or diminished in quantity. There is local vasodilatation in the kidney with increased secretion of urine. The polyuria is at once arrested if pituitrin is administered. The administration causes a slight fall of blood pressure.

**Morbid Anatomy.**—Radiograms of the sella turcica usually show no enlargement. Various lesions of the pituitary gland or its neighbourhood have been described: fractures of the base of the skull, cerebral tumour, syphilitic or tuberculous basal meningitis, caseation of the infundibulum. In one remarkable case a bullet was found pressing against the posterior lobe. Parkes Weber has described what he regards as a tuberculous infiltration of the posterior lobe of the gland.

Dilatation and hypertrophy of the bladder, dilatation of the ureters, and enlargement of the kidneys may be seen, and are attributable to the prolonged pressure of large quantities of urine.

**Symptoms.**—These begin either insidiously or suddenly. The prominent symptoms are the enormous quantity of water passed and the great thirst by which the patient is led to replace the loss. The urine may reach 15, 20 or even 40 pints in the twenty-four hours. It is very pale, almost like water, of specific gravity 1,002 to 1,005, and faintly acid in reaction. The percentage of solid constituents is, of course, small. Sometimes *inosite*, or muscle sugar, has been found; but it is also present in some cases of diabetes mellitus and Bright's disease, and even in health after large quantities of water have been drunk. Quite exceptionally a trace of albumin is present; glycosuria may occur occasionally. The thirst is excessive and uncontrollable, the patient being obliged to drink large quantities of water. Occasionally there is an increased secretion of the salivary glands.

Other symptoms are the following: The mouth and tongue are usually dry, the skin is dry, and the temperature is normal. The appetite is often poor, sometimes unaffected; exceptionally, however, it is enormous, as it is so often in diabetes mellitus. The bowels may be constipated. Beyond this the patient may be in the enjoyment of very good health, and he finds the diabetes an annoyance rather than an illness. But often, especially in the severe cases, there are emaciation, weakness and languor; the sleep is much disturbed, and there

is mental depression or irritability of temper; occasionally the sexual powers are abolished.

The course of the disease is variable. If it arises from injuries to the head, it may be of short duration; when it is due to a definite cerebral lesion, its course will be determined by this. Spontaneous and idiopathic cases may last for years if untreated. They are rarely fatal, except from the intervention of other illnesses, especially phthisis and pneumonia; occasionally glycosuria has supervened, and the case has become one of diabetes mellitus.

**Diagnosis.**—The enormous quantity of pale urine, of low specific gravity, without abnormal ingredient, and the accompanying thirst, are distinctive. But care must be taken to exclude other forms of polyuria, such as those from *Bright's disease* and *hysteria*. In the former there is generally at some time or other a distinct trace of albumin, the quantity of urine is not so considerable, and other indications are present, such as high arterial tension and cardiac hypertrophy. In hysteria the condition is but temporary. The urine of *diabetes mellitus* is at once distinguished by its higher specific gravity and by the presence of glucose.

**Treatment.**—This consists in the administration of pituitary extract subcutaneously. One cubic centimetre (15 minims) is injected twice a day to begin with. The attempt is then made to control the volume of urine with as few injections as possible. In a case of Dr. Otto May's the symptoms were relieved with five injections a week, and when reported this treatment had been continued for some years, and no septic complications had arisen. Unfortunately treatment by the mouth is useless, as the active principle is destroyed in the alimentary canal.

In syphilitic cases antisyphilitic treatment (*q.v.*) is indicated. In cases of gross cerebral disease (*e.g.* tumour, etc.) the question of operation will naturally arise. A few cases have been relieved by lumbar puncture, which suggests that a serous meningitis at the base of the brain was responsible for the disease (**27**).

# DISEASES OF THE PINEAL GLAND

The pineal body (epiphysis cerebri or conarium) is a glandular organ, weighing about 3½ grains, and consisting of epithelioid cells in loosely disposed trabeculæ, with blood sinuses between them. Like the thymus, its chief use appears to be in early life, and it undergoes a certain amount of involution later.

The lesions which have been recorded are hypertrophy and atrophy, tumours, cysts and abscess, hæmorrhage and syphilis.

In the cases of tumour which have shown changes in metabolism, indicating a disturbance of the internal secretion, the subjects have been boys up to eleven years, and the changes consisted, in different degrees in different cases, of mental precocity, unusually rapid growth of the body, enlargement of the penis and testes, precocious growth of pubic hair, and sometimes adiposity. These were sometimes associated with evidences of intracranial tumour (*see* p. 852), and the tumours present were in different cases sarcoma, cystic psammo-sarcoma, glioma or teratoma.

# DISEASES OF THE GONADS

A systematic description of the pathological changes found in the testicle and ovary will be found in text-books of surgery and gynæcology. The gonads will only be considered here from the point of view of their endocrine function, which is brought about by a hormone secreted by the *interstitial* cells of these glands. These hormones are responsible for the development and maintenance of the

secondary sexual characters, they influence skeletal growth and psychic functions. Over-activity, under-activity, or absence of the gonads are respectively known as hyper-, hypo- and a-genitalism.

**Eunuchism.**—The symptoms depend on whether castration has been performed before or after puberty. In the former case the penis, prostate, vesiculæ seminales, remain small and the subjects do not develop libido or potentia. If the operation is performed after puberty, the penis need not atrophy much, but the prostate becomes smaller. Libido may persist, and even potentia, at least for a time. Prostatic secretion is ejaculated. Psychically, the normal aggressiveness of the male disappears, but eunuchs may possess great intellectual powers and artistic ability. Physically, they are either (1) tall and thin, with long arms and legs, or (2) short and fat, with a distribution of fat similar to hypophyseal dystrophia genitalis. In both types the pelvis remains broad, the skin pale; there is drooping of the lateral half of the eyelids with a sleepy appearance, and there is generally a deficient growth of hair. In the female, double oöphorectomy brings on a premature menopause with tendency to obesity, hot flushes, respiratory, cardiac, and digestive disturbances. Neurasthenia, psychasthenic or psychotic symptoms may appear.

**Eunuchoidism.**—This term is applied to conditions resulting from loss of the gonads by disease. They are not uncommon in patients with undescended testicles (*cryptorchidism*). Like eunuchism the condition may be noticeable at puberty (early eunuchoidism) or later during the normal period of active sexual life (late eunuchoidism). As in eunuchism the patients may be tall or short and fat. Mentally, they are quiet and silent and there are disturbances in libido and potentia. The skin is pale and sometimes has large numbers of small wrinkles. Linear wrinkles radiating from the mouth make these patients look prematurely old. Female eunuchoids are tall and fat is deposited on the lower abdomen, mons veneris, buttocks and outer sides of the thighs and the mammary glands. The uterus and vagina become hypoplastic.

**Hyper and Hypogenitalism.**—While the terms eunuchism and eunuchoidism are reserved solely for results following primary disease or absence of the gonads, over- or under-activity of the latter may also result secondarily from disease of other ductless glands. Thus hypergenitalism which causes precocity or premature puberty may result from tumours of the testicle or ovary, from hyperfunction of the latter, or from tumours of the suprarenal cortex or pineal body already described. Delayed puberty, so commonly met with in both sexes, may similarly be due to hypofunction of the gonads primarily or may result from other organs acting on the gonads.

# VARIED ENOCRINOPATHIC SYNDROMES

## OBESITY

Obesity, corpulence, or excessive fatness is a condition which may amount to a disease, and sometimes calls for treatment. But it is often difficult to say where normal deposition of fat ends, and where obesity begins; and the two conditions must be dealt with together.

**Ætiology.**—There appear to be some differences in the tendency to obesity among the races of mankind. Heredity certainly has a share in its occurrence. There are some periods of life when fat is more likely to accumulate; these are infancy, puberty, during pregnancy and at the climacteric in the female, and during middle age in the male. Females, on the whole, are more liable to be fat than males.

**Pathology.**—In a normal individual whose body weight remains constant the energy supplied by the utilisation of the food must balance the energy given out in the form of heat and external muscular work. If he suddenly changes his habits by leading a more sedentary life, the energy output will be diminished, and less food will be oxidised, the excess being deposited in the body as fat. Both fats and carbohydrates are readily stored in this way; but the constituents of protein are more easily oxidised, increasing the heat output. This is sometimes called the *specific dynamic action* of protein. Hence the important factors in many cases of obesity are bodily inactivity and over-eating, particularly of farinaceous and fatty foods.

There are, however, cases of great obesity which cannot be entirely explained in this way. Amongst healthy persons there are many very fat who eat comparatively little; and, on the other hand, many big eaters are persistently thin.

These idiosyncrasies are explained by the rate of oxidation peculiar to the individual, and can be compared by measuring the *basal metabolism* (*see* p. 558). Some of the ductless glands exercise a profound influence on the basal metabolism. In hypothyroidism or myxœdema the basal metabolism is diminished, and these subjects readily become fat. On the other hand, in Graves' disease, where the activity of the thyroid is increased, emaciation occurs. Adiposity has been observed in cases of pituitary disease exhibiting hypopituitarism, in hypernephroma of the suprarenal capsule, in disease of the pineal gland, and in some cases of brain tumour and mental disorder. An inverse relation also exists between the activity of the sexual organs and the growth of fat, as shown by changes at the climacteric period; obese persons are often deficient in sexual vigour, and diminution or loss of virile power is often accompanied by adiposity. The latter is a well-known sequela of castration. In many of these cases the basal metabolism has been found to fall within normal limits; but it is probable that the increase in the metabolism (respiratory exchange) which follows the taking of food is below normal, and this would explain the obesity (**4**).

It must be remembered that adiposity affects not only the subcutaneous tissue, but also the connective tissues of the internal structures; and ultimately not only the connective tissue, but the essential parts, such as gland cells and muscular fibres, are likely to undergo fatty degeneration. Some of these conditions have already been described in various sections of this book. The fatty change which, up to a certain point, is evidence of good nutrition, becomes ultimately a serious form of degeneration.

**Associated Conditions.**—Very fat persons suffer several inconveniences, though the extent may depend a good deal upon the period of life at which the obesity is acquired. If early, the muscular system may be developed to correspond with the great additional weight; and the athletes of old were often fat, as are some wrestlers in our own times. But, as a rule, very fat persons are incapable of much exertion, are short of breath, and liable to palpitation. They are frequently drowsy or somnolent, and deficient in mental as well as in physical vigour. Yet to this there are striking exceptions. Amongst associated conditions gout may especially be mentioned, since dietetic excesses predispose to it. Obesity also predisposes to diabetes mellitus.

**Adiposis dolorosa** (Dercum's disease).—In this disease pathological changes in the thyroid and pituitary may be found, and there is neuritis in the affected parts. It occurs in two forms: (*a*) *diffuse lipomatosis*, which either involves the whole body except the hands and feet or is more or less localised to a particular part, though in this case it is symmetrical about the body; (*b*) in the form of *fatty nodules*, also symmetrically arranged. The fatty deposits are painful, particularly on pressure, and the patients are often asthenic and sometimes feeble-minded.

**Treatment.**—This is admittedly difficult, and the question whether in any given case it should be attempted often requires careful consideration. It

is probably most likely to be successful in adiposity traceable to diet and inactivity. In any case where treatment is undertaken, the patient should reduce considerably or abstain altogether from fat and foods containing it, from sugar, farinaceous foods, potatoes, peas, beans, and vegetables containing much starch; on the other hand, lean meat, game, poultry, fish, green vegetables, tomatoes and fresh fruit may be taken. A protein diet of this character is the basis of Banting's treatment. In very obese patients more drastic measures will be required. Periodic fast days should be prescribed, and the patient should eat almost exclusively 5 per cent. vegetables (*see* p. 574) and fresh fruit with just enough fish (2 or 3 ounces) to cover the daily protein loss. Jellies made with agar-agar, which is not absorbed by the alimentary canal, are useful. Such a diet possesses the advantage of having a low calorie value, and at the same time it is fairly bulky, so that the feeling of emptiness is obviated. Alcoholic drinks are best avoided; if any are demanded, a dry light wine or a small quantity of whisky well diluted is the best. Beer containing much maltose is to be avoided.

Exercise is of great value in reducing the weight due to fat. The selection must depend on the age and muscular vigour of the patient. Riding and golf are useful at all ages; but for younger patients fencing, rackets and lawn tennis are more efficient. It is often difficult to persuade very fat people to take exercise, especially where pain is present, as in adiposis dolorosa. The Bergonié treatment may then be of use. This consists in exciting painless contractions in the muscles of the trunk and limbs by a rhythmically interrupted faradic current. This is not so efficacious as voluntary muscular work, but it is better than nothing. Subthermal baths are valuable, since they increase metabolism. The duration may be up to an hour and the temperature should be as low as the patient can tolerate, perhaps 80° F.

Thyroid is the most suitable drug to employ, as it increases the metabolism. One grain of the dry gland should be given twice a day and the dose gradually increased, the rate of the pulse being watched. The drug should be stopped at once if any signs of hyperthyroidism appear. Thyroid treatment should not be looked upon as an alternative to increasing the amount of muscular exercise. The latter is usually the more important line of treatment.

## INFANTILISM

By infantilism is meant the retention of the characters of childhood for a longer time than usual, or an abnormally slow development. Infantilism is not necessarily associated with dwarfism, but the childish form may be retained, and there may be delayed ossification and absence of sexual development. There need not necessarily be any delay in the child's mental progress.

Gilford divides infantilism into two classes, symptomatic and essential. Under the symptomatic cases he includes (1) those in which the arrest of development is due to toxic causes, such as syphilis, tubercle, malaria, pellagra, scarlet fever, chronic diarrhœa, pancreatic deficiency and some inorganic poisons, such as lead and mercury; chronic interstitial nephritis in children may be associated with infantilism. He includes (2) a class in which the infantilism is associated with disease of the endocrine organs, such as cretinism, Fröhlich's syndrome, and diabetes insipidus. Other instances of this class are the delays in development which have been seen in association with achondroplasia, muscular atrophy, sclerodermia, cardiac and vascular lesions, microcephaly, hydrocephalus, amentia, hypertrophic cirrhosis of the liver, splenomegalic cirrhosis, and some other disorders.

As distinguished from these forms of infantilism, essential infantilism is seen in two forms, called *ateleiosis* and *progeria*.

Progeria is a condition in which infantilism is associated with senile characters,

arterio-sclerosis, atrophy of the tissues, baldness, and general weakness. Only a few instances have been recorded.

Ateleiosis is infantilism in which there is no obvious underlying cause; it occurs in two forms:—in one, the asexual form, there is delay in the development of the whole body, of some organs more than of others, and of the sexual organs most of all. In the other, the sexual form, there is a similar delay until puberty is reached · this indeed, may be delayed also, but ultimately the sexual organs mature, the epiphyses unite, and the individual remains in some respects a child, in others a man or woman in miniature. Ateleiosis is sporadic, occurs without apparent cause, is unaffected by environment, and may appear in two or more members of the same family. It may commence in fœtal life, in infancy, or between childhood and puberty.

## PLURI-GLANDULAR SYNDROMES

**Pluri-glandular Insufficiency of Claude and Gougerot.**—This syndrome develops between the ages of twenty-five and thirty and progresses over a number of years, death being brought about frequently by intercurrent disease. The symptomatology is varied, depending on the glands affected. The secondary sex characteristics and libido may disappear from atrophy of the gonads, and possibly the suprarenal cortex. Loss of hair, with psychic disturbances and myxœdematous symptoms may be traced to thyroid deficiency, while polyuria may be due to pituitary involvement, and pigmentation, hypotonus and asthenia to suprarenal involvement.

**Thymus-Suprarenal-Pituitary Compensatory Syndrome (Timme)** begins some years before puberty. *Stage* 1. There is disproportion of different parts of the skeleton; teeth appear late and are irregular in size; the epiphyses are late in joining the shafts of the bones. The hair is sparse. Tonsils are large and adenoids present. *Stage* 2. At puberty there is rapid skeletal growth. The menses are late and the uterus small. There is lack of hair. Mentally the patients show loss of concentration. *Stage* 3. Between twenty and thirty gigantism manifests itself with pituitary headaches. There is mental confusion with epileptiform attacks, uncinate in type. *Stage* 4. Compensation gradually takes place in later life and the headaches and fatiguability disappear (**30**).

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# DISEASES OF THE URINARY ORGANS

## EXAMINATION OF THE KIDNEY AND BLADDER

THE kidney normally extends from the lower border of the eleventh dorsal vertebra to the lower border of the second lumbar vertebra, its inner margin being, on an average, 3 inches from the middle line; and it is fixed in this position by its surrounding capsule of adipose tissue, by the layer of peritoneum in front of this, and by the renal vessels. The position of the kidneys corresponds on the anterior surface of the body to a rectangular figure, 8 inches by 4 inches, having its lower long side level with the umbilicus, and its short sides each 4 inches from the middle line.

The kidney cannot be felt, except in very emaciated persons, or when it is enlarged, or when it is more mobile than normal (*movable kidney*), or when it is pushed down by disease above. Each flank should be examined by the bimanual method, and the patient should be directed to take a deep breath; or in thin persons the flank may be grasped by one hand, the fingers behind and the thumb in front.

A renal tumour may be mistaken for the liver on the right side or the spleen on the left. If the resonance of the colon is detected in front of the mass, it is not the spleen or the liver.

With the aid of the cystoscope valuable information may sometimes be obtained as to the condition of the kidneys. If one or other kidney is the subject of pyelitis, pyelo-nephritis, tubercular disease, or similar lesion, and the ureter is involved, the orifice of the ureter in the bladder may be patent, with swollen lips and vascular adjacent mucous membrane. The ureters may be catheterised and the urine from them collected separately in order to find out if disease is localised in one of the kidneys. The relative volume of the urine from each kidney is noted, and examination for blood, pus and albumin is carried out.

In the diagnosis of certain congenital malformations of the kidney, hydronephrosis, kinking of the ureter, renal tumours projecting into the pelvis and renal calculus, *pyelography* may be of great assistance. After catheterising the ureter and allowing urine to escape, a 20 per cent. solution of sodium bromide, which is opaque to the X-rays, is cautiously run in and a radiogram is then taken. The normal pelvis will allow 6 c.c. of the solution to enter. No anæsthetic is given and the injection is stopped if pain in the loin is experienced. Only one ureter is catheterised at a sitting. To show up the bladder 10 per cent. sodium bromide may be used, or 10 per cent. colloid silver (**1**).

## EXAMINATION OF THE URINE

**Quantity.**—The water content of the body is a balance between that which is taken in by the mouth and that which is excreted from the kidneys as urine, from the skin as sweat, from the lungs, in the fæces, etc. The secretion of urine depends on the amount of blood flowing through the kidneys. Excessive secretion of urine is called *polyuria*, its suppression *anuria*. The volume is increased by the ingestion of fluids and exposure of the body to cold, because in the latter

case the secretion of sweat is diminished, while it is diminished by abstinence from drink, by free sweating, and the loss of fluid from the circulatory system. The average daily volume of urine is 1,500 c.c.

In disease, variations are seen in the following circumstances: The flow of urine is increased in the earlier stages of chronic granular kidney, in lardaceous disease of the kidney, in rare cases of cerebral disease, in diabetes insipidus, and in diabetes mellitus. A temporary increase is seen in hysterical attacks, as a result of mere nervous excitement (not infrequent during medical examination for life insurance), and from the administration of substances having a diuretic action, such as the acetates, citrates, and tartrates, and, perhaps most commonly, alcoholic drinks. The urine is scanty or suppressed in acute nephritis, in the last stages of chronic nephritis, in obstruction of the ureter unless the other kidney is equal to the task of secreting twice its normal amount, in all febrile processes, in cardiac failure, and after repeated vomiting, or profuse diarrhœa. The diminution in fevers is explained by increased transpiration of aqueous vapour through the lungs and skin, and by diminished arterial tension; in cardiac failure the diminished arterial tension is the efficient cause.

**Specific Gravity.**—This varies directly as the amount of solids in the urine, inversely as the quantity of water—that is, of the urine itself. The excretion of both water and solids varies with the period of the day, and it is therefore desirable to mix the whole of the urine secreted during twenty-four hours, and to take the specific gravity, or density, of the combined liquids, which varies between 1,000 and 1,050, the specific gravity of water being taken as 1,000. A hydrometer, called a *urinometer*, is used for this purpose. If the quantity of urine is too small to float the urinometer, it may be diluted with one, two, or three volumes of water, as may be required, and the specific gravity will be obtained by multiplying the last two figures by the number which represents the dilution. The density varies with the temperature. For accurate record all observations should be made at 60° F., or corrections should be made by adding one degree of density for every 8° F. above 60° F.

A single observation, such as is made commonly in examinations for life insurance, is of little value, since the specific gravity may be altered several degrees in a few hours, or even minutes, by free drinking of liquids, by exposure to cold, by profuse sweating, or by nervous excitement.

The urine in health has a specific gravity of 1,015 to 1,025. It is diminished by most of the causes of polyuria, so as to be 1,010, 1,006, 1,004, or less, as in chronic granular kidney, lardaceous disease, diabetes insipidus, hysteria, nervous excitement, and under the influence of alcohol or other diuretic. It is increased by all the causes of scanty urine, such as heart disease, acute nephritis, and profuse sweating, and by conditions which increase the solids actually or relatively to the fluid secretion. In diabetes mellitus the specific gravity may be increased to 1,030, 1,040, or 1,050, although the quantity of urine is many times more than the normal; the unnatural secretion of large quantities of sugar accounts for this exceptional condition. But if the urine reduces copper *slightly*, this is more likely to be due to sugar, if this specific gravity is low. Albumin occurs in urine of both low and high specific gravity; it must help in increasing the density, but it is, as a rule, in very small quantity when the conditions of the kidney allow a free secretion of urine.

**Urea.**—This constitutes about one-half the total solids of the urine, and is the chief form in which the nitrogen of the body is excreted.

*Quantitative Estimation.*—A convenient method of estimating the urea is to decompose it by the addition of *sodium hypobromite* (or *hypochlorite*) into carbonic acid, water, and nitrogen—

$$CON_2H_4 + 3NaBrO = 3NaBr + CO_2 + 2H_2O + 2N$$

A measured quantity of urine (5 c.c.) is mixed with a quantity of the hypo-

bromite solution in a bottle, and the nitrogen is collected in a graduated tube and measured. The bottle and tube are surrounded with water so as to keep the temperature constant. The hypobromite solution rather readily undergoes decomposition on keeping.

The nitrogen is measured, and from that the amount of urea can be calculated, the proportion of urea to its contained nitrogen being as 60 to 28, or 15 to 7. Really the hypobromite method shows only about 92 per cent. of the nitrogen contained in the urea, but it obtains another 2 per cent. from creatinin and other bodies.

The determination of the output of urea in the twenty-four hours is a rough measure of the amount of protein metabolism. A more accurate way of measuring the latter is to estimate the total nitrogen of the urine by Kjeldahl's method. Allowance must also be made for the loss of nitrogen in the fæces, sweat, etc. Under normal conditions the nitrogenous content of the body remains constant, so that the excretion of nitrogen exactly balances the amount contained in the protein of the food, and varies greatly from day to day as the amount of protein in the food varies. In disease the nitrogenous content of the body may vary, and this will produce its effect on the nitrogen excreted in the urine. For instance, in fever there is a destruction of body proteins, whereas in Bright's disease there may be a retention of nitrogenous products. This will show itself by an increase or decrease in the urinary nitrogen relatively to the protein consumed in the food. Such changes can only be measured if the patient eats an absolutely constant diet over a period of days. The average daily excretion of urea is about 32 grammes; it forms normally about 2 per cent. of the urine.

**Chlorides.**—Their presence is shown by a white precipitate on the addition of nitrate of silver to the urine, acidulated with nitric acid. Normally the precipitate is thick and curdy: with diminished chlorides it may be a mere turbidity. The daily excretion of chlorine is about 7 grammes, equal to about 15 grammes of sodium chloride. The chlorides are lessened in all acute febrile processes, and especially in pneumonia, where they may be entirely absent.

**Sulphates.**—The daily excretion of sulphates is from 1·5 to 3 grammes. Of these the greater part are alkaline sulphates, precipitable by barium chloride; but from 5 to 10 per cent. are aromatic or ethereal sulphates, not precipitable by barium chloride, but convertible into the alkaline sulphates by boiling with hydrochloric acid. The ethereal sulphates are partly the result of putrefaction in the intestines, and are largely increased in disorders of the intestines (obstruction, peritonitis, constipation) leading to such changes.

**Phosphates.**—The daily excretion of phosphoric acid in the urine is from 2 to 3·5 grammes, combined with the alkaline bases, sodium and potassium, and with the earthy bases, calcium and magnesium. The amount is chiefly determined by the food, phosphoric acid being provided by tissues rich in nuclein.

The chief function of the phosphates in the urine is to play the part of what are known as "buffers." This means that a large amount of acid or alkali may be passed from the blood into the urine without altering very greatly the acidity (hydrogen on concentration) of the latter. In this connection the two most important compounds are the monosodium dihydrogen phosphate ($NaH_2PO_4$) and the disodium monohydrogen phosphate ($Na_2HPO_4$). The result of excreting acid is to form more of the former compound, and of excreting alkali more of the latter. The urine in health may be made more alkaline from the ingestion of much vegetable matter or other food containing citrates, tartrates, or malates of potassium and sodium. These are converted into alkaline carbonates in the intestines, and absorbed as such into the blood, and hence diminish the acidity of the urine. Under these circumstances the less soluble phosphates preponderate, and those of the earthy salts, calcium and magnesium, may be precipitated even in the bladder.

Thus occasionally the urine, when passed, is quite turbid from a white deposit,

consisting mainly of amorphous phosphate of calcium ($Ca_3(PO_4)_2$). This settles as a bright white deposit, which is immediately soluble in dilute acetic or nitric acid. Rarely there is mixed with this a crystalline phosphate ($CaHPO_4 + 2H_2O$), showing under the microscope rods and needles, lying loose or grouped into rosettes, stars, fans, and sheaf-like bundles, or club-shaped or bottle-shaped masses. This is called sometimes stellar phosphate. Magnesium phosphate ($Mg_3(PO_4)_2$), in elongated plates with oblique ends, has also been identified.

A much more common occurrence is this, that on heating a feebly acid or neutral urine a turbidity or thick white deposit of earthy phosphates occurs, which closely resembles albumin, but is at once distinguished from it by being dissolved by a drop of acetic or nitric acid. The heat acts by decomposing the monohydrogen phosphate ($CaHPO_4$), which is moderately soluble, into the more soluble dihydrogen phosphate ($Ca(H_2PO_4)_2$) and the calcium phosphate ($Ca_3(PO_4)_2$), which is less soluble, and is precipitated.

The urine may become alkaline from the presence of ammonia, which is formed as the result of bacterial decomposition of urea, either within the bladder (cystitis) or after standing in a vessel exposed to the air. In this case ammonium magnesium phosphate is precipitated ($MgNH_4PO_4 + 6H_2O$). It forms triangular prisms, with bevelled ends, often very perfect indeed, but sometimes modified by the edges or angles being, as it were, planed off, or the ends or surfaces hollowed out. They often reach a large size, and are strongly refracting. This deposit may be mixed with that of the phosphate of calcium.

**Calcium Oxalate.**—The quantity of calcium oxalate secreted daily is very small, but it is sometimes seen as a deposit in the urine, or it forms calculi in the kidney. In urine containing an oxalate deposit there is mostly a pale grey mucus-like sediment, and above this a white dense layer, with a wavy, sharply defined surface. Under the microscope will be found the minute octahedra of calcium oxalate, looking often like square envelopes, and measuring from $\frac{1}{5000}$ to $\frac{1}{500}$ inch in the side. In different positions they may seem to have a rhombic or hexagonal outline, and if the edges are not developed they may form square prisms, with pyramidal ends. A not uncommon variety is that of the "dumb-bell," which is really a disc, with a central depression on either face, lying on its side, and seen edgeways. Such formations no doubt result from slow precipitation in the presence of colloid matter.

Calcium oxalate crystals are with difficulty soluble in hydrochloric acid, and insoluble in acetic acid. Their deposition is sometimes due to excessive ingestion of vegetables (cabbage, rhubarb, spinach, strawberries) containing oxalates; or to changes in the urine after secretion; or to disturbances of digestion. Their connection with symptoms such as languor, depression, and hypochondriacal feelings (the so-called *oxalic acid diathesis*) is very doubtful, unless they and the symptoms together are the results of indigestion.

**Uric Acid and Urates.**—The daily excretion of uric acid, which is in combination as urates, is only from ·5 to 1 gramme, so that the proportion in the urine is very small; nevertheless, deposits of the free acid and of its salts are not infrequent. As in the case of the other constituents, this precipitation does not prove that the quantity actually formed is in excess.

*Uric acid* is generally precipitated in very acid urine. Its deposits are distinguished by their yellow, orange, or red colour, and consist of minute shining grains, which with a lens or even the naked eye can be seen to have a crystalline, acicular, or prismatic shape. Under the microscope they show themselves as fusiform or lozenge-shaped crystals, with sharp ends and rounded sides; or as shorter and thicker crystals, with blunt extremities and more barrel-shaped; but generally in some modification of the diamond shape. They are frequently grouped together in radiating clumps or star-like masses. Thick crystals lying on their edges may seem to be rectangular, and the lozenge shape may not be detected till they roll over. Sometimes the sides of such masses are marked by

a division into several plates (*striated*). These differences of shape are due to the conditions of the medium in which precipitation takes place. Their colour is that of the urinary pigment, for which they have great affinity. The crystals are insoluble in acids, but can be dissolved in alkalies.

*Urates* are, as a rule, precipitated, in an amorphous form, as a thick pink or red sediment (*brick-dust* or *lateritious*). The causes of their deposition are (1) the cooling of the urine, because they are abundantly soluble at the body temperature, and very much less so at 50° or 60° F.; and (2) the concentration of the urine or the relative deficiency of the water, so that when cooling takes place it is insufficient to keep in solution the perfectly normal amount of urates present. The usual causes of concentration are, in health, deficient intake of fluids and free perspiration; in disease, vomiting, cardiac disease which acts by lowering arterial pressure in the renal circulation, and febrile reaction causing diminished arterial tension, and increased cutaneous and pulmonary transpiration of aqueous vapour. A gentle heat will at once dissolve the deposit—for instance, the addition of hot water to the vessel containing it; and if the urine is being tested for albumin, the urates dissolve, and leave the fluid clear before the albumin begins to appear. (3) Acidity of the urine also favours the deposition of the urates; and in some urines the addition of acetic acid prior to testing for albumin will cause a precipitate of urates, which will redissolve on heating.

The uratic deposit consists chemically of the urates of sodium, potassium, ammonium, and calcium, which are acid salts, but whether *quadrurates* (formula $MHU,M_2U$) or *biurates* (MHU) is open to question. *Ammonium biurate* occurs in ammoniacal urines in the form of dumb-bells or spheres, and the deposit is generally pale; *sodium biurate* constitutes the well-known acicular crystals found in gouty joints. *Neutral urates* ($M_2U$) do not occur in the animal body. The colour is due to a urinary pigment (*uroerythrin*), but it may be absent or amount to nothing more than a yellow tinge.

*The Murexide Test.*—If a particle of uric acid or a urate is heated on porcelain or a glass slide with a drop of nitric acid, and a drop of solution of ammonia is added to the dry residue, a purple-red colour is developed. Liquor potassæ changes this to purple-blue.

Uric acid ($C_5N_4H_4O_3$) is one of the purin bodies, that is, substances related to purin ($C_5N_4H_4$); and these are derived partly from the food ingested and partly from the metabolism of tissues in the body, such as muscle and the nuclei of breaking-down cells. It has an important relation to gout; and it is excreted in very large quantities in some diseases, *e.g.* leukæmia. Other purin bodies are xanthin, hypoxanthin, adenin, and guanin.

**Urinary Pigments.**—The urine varies in colour both in health and disease. As a rule, it is sufficient to distinguish between pale urines, normal-coloured urines, and high-coloured urines in health; while in disease we may observe, in addition, different shades of red, reddish-brown, and brownish-black from the admixture of blood, urobilin, or bile-pigment, and an opaque white colour in chyluria.

The difference of tint in pale and high-coloured urines is mostly dependent upon the amount of water contained in them. *Pale* urines are of low density, and contain a large quantity of water and a small percentage of solids. They result from all causes which increase the flow of urine, such as free ingestion of fluids; a check to the cutaneous transpiration, as from cold; increased arterial pressure, as in early chronic Bright's disease; and nervous conditions, as hysteria, nervous excitement, and diabetes insipidus. The urine of diabetes mellitus forms an exception in being pale and abundant, while it has a very high density from the quantity of sugar contained in it. *High-coloured* urines are commonly of great density, scanty in quantity, and contain a large percentage of solids. They occur after profuse sweating, or diarrhœa; in fever, and in disorders of the circulation, by which the flow through the glomeruli is diminished.

There are several pigments in the urine. Probably its colour is chiefly due, as shown by A. E. Garrod, to an iron-free pigment named *urochrome*. This pigment obscures the violet end of the spectrum, but gives no absorption bands. *Urobilin* exists in the normal urine in only small quantities, 1 to 2 grains in twenty-four hours; it gives a definite spectrum, with an absorption band at Frauenhofer's line F—that is, between the green and the blue. The chemical test of its presence is to render the urine strongly alkaline with ammonia solution, filter, and add a few drops of a 10 per cent. aqueous or alcoholic solution of zinc chloride: a green fluorescence occurs if urobilin is present. Urobilin is probably absorbed from the intestine, where it is originally derived from bilirubin. Urobilin is in excess in fevers, in some diseases of the liver, in cardiac failure, in excessive hæmolysis (*e.g.* in pernicious anæmia and in acholuric jaundice), and during absorption of extravasated blood. It is diminished in chlorosis, and when the formation of bile is checked (phosphorus poisoning, acute yellow atrophy) or the bile duct is occluded. *Hæmatoporphyrin* is derived from hæmoglobin; it occurs in minute quantities both in health and in disease, and more abundantly in the urine of rheumatic fever and some other disorders. It may not cause any appreciable difference in tint, and though it exists in quantity in some dark red urines passed after the administration of sulphonal, the dark colour is mainly due to other pigments. There is a rare in-born error of metabolism, in which hæmatoporphyrin is passed in the urine giving it a ruby-red colour. Porphyrins are present in the fæces and the skin is very sensitive to light, which causes a bullous eruption of hydroa æstivale (**2**). *Uroerythrin* is another pigment, which gives the colour to pink urates. Uric acid deposits are coloured by urochrome, and also sometimes by uroerythrin (Garrod).

The urine may also contain *chromogens*—that is, bodies which do not at the time colour the urine, but develop a colour either on standing, or on the addition of oxidising agents. The following are known: (1) the chromogen of urobilin, urobilinogen, shown to exist in febrile urine by the addition of nitric acid; (2) a chromogen found sometimes in the urine of anæmia, which, though quite pale when passed, may yield a deep red colour on the addition of nitric acid; (3) the chromogen of *melanin*, a black pigment which is developed on exposure, or on the addition of nitric acid, in the urine of patients suffering from melanotic sarcoma, although it is clear when passed; (4) indican, or potassium indoxylsulphate, the chromogen of indigo-blue. This is the result of the absorption from the intestinal canal of indol, which results from the bacterial decomposition of proteids. It exists in normal urine to a very small extent, but is greatly increased in all conditions leading to retention of intestinal contents, such as constipation, intestinal obstruction, and peritonitis. Its presence is detected by the addition of an equal quantity of hydrochloric acid, and then a few drops of a saturated solution of calcium oxychloride. Indigo is thus formed, and colours the mixture blue or violet. It can be separated by shaking with chloroform, which then forms a blue layer at the bottom of the test-tube. Sometimes the addition of nitric acid alone develops a blue, violet, or blackish colour, due to the separation of indigo.

In *alkaptonuria* the urine is of a natural colour when passed, but darkens on exposure, is darkened rapidly by alkalies when warmed, is turned deep blue by a dilute solution of ferric chloride, and is found to contain homogentisic acid, and sometimes uroleucic acid. The condition is rare, but in many of the cases it is congenital, and occurs in two or more members of the same family, and the children of parents who are blood-relatives. It causes no symptoms (*see* Ochronosis).

Several *medicinal and other chemical substances* colour the urine, or give colour-reactions with tests employed for other purposes. Rhubarb, which contains chrysophanic acid, makes the urine a deeper yellow; and santonin the same The addition of an alkali will turn these urines red. Logwood gives a reddish

tinge to the urine. Carbolic acid, taken internally, or absorbed from carbolic dressings, often causes the urine to become dark brown or greenish-black on exposure, though clear when passed, from the presence of pyrocatechin and hydrochinone. Creosote may have the same effect. Methylene blue, taken internally, renders the urine blue, or, if in small quantity, green. Eosin, used to colour toys or sweets, has caused a bright red urine in children. If potassium iodide or potassium bromide is being taken, nitric acid may darken the urine from the liberation of free iodine or bromine. Free iodine or bromine can be separated by shaking with chloroform.

**Reaction of the Urine.**—The reaction of the urine (hydrogen ion concentration) is usually on the acid side of neutrality. (Hasselbalch has shown that the $p_H$ may vary from – 5·0 on a meat diet or during starvation to – 8·0 when a diet containing vegetables and free alkali is eaten.) It has already been pointed out that the reaction depends on the relative amounts of $Na_2HPO_4$ and $NaH_2PO_4$ present in the urine. It so happens that at the particular reaction corresponding to the point when the phosphate is present solely as $Na_2HPO_4$, the indicator *phenophthalein* turns pink. On the other hand, when all the phosphate is present as $NaH_2PO_4$ *methyl orange* turns pink. The relative amounts of these two phosphates in a specimen of urine may be determined by a double titration, decinormal acid being used with methyl orange as the indicator in one case and decinormal alkali with phenolphthalein in the other. The ratio of the amounts used in the two titrations gives the ratio in which the two phosphates are present, and from this, by reference to a curve, the hydrogen ion concentration can be obtained. The reaction of the urine representing this ratio may also be expressed as so much per cent. alkali or so much per cent. acid by calculating what percentage of the total phosphate is present as the alkaline or acid salt (Leathes).

The kidney may also be regarded as an organ which secretes a large amount of acid in the twenty-four hours, forming from the neutral blood a liquid, viz. urine, with an acid reaction, and this is another way of looking at the acidity of the urine. The amount of the acid excreted is determined by titrating the urine with decinormal soda back to the neutral point, *i.e.* the reaction of the blood, neutral red being used as an indicator. In health there are wide variations; but the average value for twenty-four hours on a rather high protein diet is about 600 c.c. decinormal acid (Folin).

The term *amphoteric reaction* is sometimes used when the urine turns blue litmus red, and red litmus blue.

After it has passed from the body the reaction of the urine generally undergoes a change, becoming first more acid, then again less acid, and finally alkaline. These changes are due to fermentation from the presence of micro-organisms, for if urine is properly protected from contact with such bodies, it may be preserved for years. The increased acidity is due to more acid phosphates, as well as lactic and acetic acids. The alkalinity results from the decomposition of urea and the formation of ammonium carbonate. In the former case uric acid is often deposited; in the latter ammonium-magnesium phosphate, ammonium urate, and calcium phosphate and carbonate are thrown down.

When it is desired to render a patient's urine alkaline, the best means of doing so is to administer repeated drachm doses of the potassium or sodium salt of citric, tartaric, or malic acids, which act as above explained (*see* p. 605); but sodium bicarbonate may also be given. The acidity of the urine is increased in fevers and diabetes. It may be artificially increased by administering acid sodium phosphate (30 grains) by the mouth three or four times a day.

## THE ESTIMATION OF THE RENAL FUNCTIONS

In the course of various forms of renal disease it may be desirable to estimate the power of the kidneys as excreting organs, and in diseases which affect the

organs unequally to know which organ is the better or the healthy one. The amount of albumin passed *per diem* is no real measure of the damage suffered by the kidneys; and the diminution of the solids can be but a rough estimate of the excretion of nitrogen, since nearly one-half of them are salines, and the relative proportion of the urea and salines varies from day to day considerably. The following are some of the methods by which an estimate is attempted.

**Estimation of Urea in Blood.**—The simplest and shortest method is Yvon's. Ten cubic centimetres of blood from a vein are mixed with 5 c.c. trichloracetic acid to precipitate the proteins and made up to 25 c.c. in a small measuring flask. The mixture is filtered into a measuring glass and the volume of the filtrate noted. This is made alkaline with caustic soda, phenolphthalein being used as an indicator. The measuring apparatus consists of a small funnel connected below by a stopcock with a short burette. On the lower end of the burette a rubber bag is fixed containing some glass beads. The bag is squeezed, and the apparatus is filled with the alkaline filtrate, which is carefully washed in, all the air being expelled from the bag and burette, and the stopcock is then closed. Some sodium hypobromite is poured into the funnel, and is allowed to enter the burette. The contents of the latter are mixed by inverting it several times. The nitrogen liberated is collected at the top of the burette, and is measured by transferring the burette to a cylinder of water and removing the bag. The calculation follows from the fact that 1 gramme urea corresponds to 370 c.c. of gas measured over water at 15° C. and 760 mm.

Normally the blood contains 30 milligrammes per cent. Figures of ten times this amount are found in uræmic coma. Cerebro-spinal fluid may also be used. In this case it is unnecessary to remove the proteins with trichloracetic acid

**Urea Concentration Test.**—This is de Wesselow and Maclean's modification of a urea test introduced by Albarran and McCaskey. The patient empties his bladder, and immediately drinks 100 c.c. of a solution containing 15 grammes urea flavoured with tincture of orange. One hour later he passes urine, and again after another hour. The percentage of urea is determined in the two specimens. If the percentage is above 2, the excretory power is satisfactory. With nitric acid; severe cases the concentration does not rise above 1·5 per cent. Cases of exposure, centration of under 1 per cent. are rare. If there is a marked diuretic melanotic figures may be obtained. The volume of urine in the second sample indoxyl- be greater than 150 c.c. It is claimed that this test is more than the absorption blood urea estimation. Since it is possible that urea in high decomposition poisonous, it would appear rather unsafe to use the test in cases it is greatly blood urea is high; but Maclean has denied that there is any facts, such as uræmic cases. detected by

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**Test for Œdema Formation.**—The patient is given a fixed diet and weighed every day for a week. He is then given daily 10 grammes of salt spread over two or three meals for ten days. An increase in weight of 2 or 4 lbs. means

that fluid is being retained, as may happen in hydræmic nephritis, and the test must be stopped at once.

# DISEASES OF THE KIDNEYS

## NEPHRITIS AND BRIGHT'S DISEASE

### GENERAL CONSIDERATIONS

To Dr. Richard Bright belongs the credit of having first recognised the association of general dropsy and albuminous urine with a morbid condition of the kidneys. In his first paper, in 1827, he showed that dropsy was often associated with large, pale and smooth kidneys, and that the urine contained albumin. In 1837 he published another paper to show that in many cases of albuminuria, where there had been no dropsy, the kidneys were small, shrunken and hard. These changes are largely of an inflammatory nature, and thus *Bright's disease* came to be almost synonymous with non-suppurative inflammation of the kidney. But a closer study of kidney diseases shows that inflammation of the kidney may arise in many conditions which do not fall within the range of Bright's diseases. Thus nephritis is caused by: (1) the toxins of various diseases, such as scarlatina, measles, and diphtheria; (2) micro-organisms carried by the blood-vessels, as seen in the metastatic suppurative nephritis of pyæmia; probably also in pneumococcal nephritis, typhoid nephritis, the speckled kidney of malignant endocarditis, and the more local effects of embolism in the same disorder; (3) micro-organisms spreading up the urinary passages, as in so-called consecutive suppurative nephritis (ascending nephritis), in tubercular nephritis, and in nephritis from urine with bacillus coli communis; (4) the presence of calculus in the pelvis the kidney; (5) the toxins of syphilis and of the pyogenetic micro-organisms with dec the kidney the lardaceous degeneration; this is frequently accomneutral reflammatory changes both in the epithelial and connective tissues, but the averains concerned are probably the cause of the inflammation as well 600 c.c. decinoration: (6) the primary contracted kidney is generally regarded

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But these distinctions are not absolute: in parenchymatous nephritis the connective tissue does not escape, and conversely in chronic interstitial nephritis the tubules and glomeruli are involved; moreover, the lardaceous disease is accompanied by both parenchymatous and interstitial changes.

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From the histological standpoint one must recognise the following structures in the kidney: (1) the tubules, with their epithelium, forming the parenchyma of the kidney; (2) the interstitial tissue, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes; (3) the blood-vessels, and the glomeruli, consisting of the vascular tuft, the capsule, and the epithelial cells covering the former and lining the latter. These tissues are somewhat differently involved in various diseases of the kidney. Thus the nephritis of scarlet fever is called an acute glomerular nephritis, or a glomerulo-tubular nephritis. Then there is an acute interstitial nephritis and a chronic interstitial nephritis, and a chronic parenchymatous or tubal nephritis which chiefly involves the tubules. The vessels as well as the glomeruli are early invaded by the lardaceous degeneration: *waxy, amyloid,* or *lardaceous* kidney.

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mation, affecting the several tissues at the same time, but in different circumstances involving the tubal epithelium, or the glomeruli, or the interstitial tissue most, so that a distinction between tubal, glomerular, and interstitial kinds may still be maintained. In addition, we must recognise both *acute* and *chronic* lesions.

Among the above forms of nephritis, the term *Bright's disease* is generally applied to :

1. *Acute nephritis* in part—*i.e.* when arising in the course of a fever, especially scarlet fever, or without obvious cause, and especially when accompanied by dropsy. The inflammation may be glomerular or glomerulo-tubular.
2. *Chronic parenchymatous* or *tubal nephritis*, producing a large white kidney.
3. *Chronic interstitial nephritis*, resulting in a *secondary contracted kidney.*
4. The *primary contracted kidney* with predominant arterial changes is not primarily inflammatory, and therefore not to be included under Bright's disease. But there can be little doubt that Bright included these cases in his observations, and the cases during life are commonly still so classed.

The lardaceous kidney is at the present time by common consent excluded from the category of Bright's diseases, unless the degeneration should be grafted upon a preceding tubal inflammation.

**A Provisional Theory of the Pathology of Bright's Disease.**—The function of the kidney epithelium is to be looked at from two points of view : (1) It has to keep back from the urine substances that properly belong to the blood, *e.g.* albumin and blood cells ; (2) it has to excrete waste products which are removed by the urinary flow ; these functions may be affected independently.

1. Some poison may injure the epithelium, so as in effect to produce a number of holes in it sufficiently large to allow the smaller protein molecules to escape into the urine, or, possibly, as recent observations suggest the poison is a wax that lowers the surface tension of the plasma, so that protein escapes through the normal spaces in the epithelium (**4**). A deficient blood supply such as may occur in heart disease, or may be produced experimentally by clamping the renal artery for a time, damages the renal epithelium, and so is another possible factor. The loss of these smaller protein molecules means that the osmotic pressure in the plasma due to the proteins is greatly diminished and the equilibrium between the blood and tissue fluids is upset, and so fluid and salts escape into the tissues, which become œdematous. The œdema may be removed (1) by increasing the protein content of the plasma, as Epstein endeavours to do by high protein feeding, so that the osmotic pressure of the plasma is raised ; (2) by diminishing the intake of salt, because the kidney must excrete salt from the blood, and if there is a deficiency, salt, and with it fluid, will be drawn back from the tissues into the circulation and excreted ; (3) by giving large doses of calcium chloride by mouth (**6**). This tends to cause acidæmia, because the calcium is removed as carbonate in the fæces while hydrochloric acid enters the circulation. A profuse diuresis results, possibly because the body proteins, which are amphoteric electrolytes and in the body play the part of acids, tend to give up adsorbed water as they approach the neutral point and their basic nature begins to come into play. It is probably inadvisable to use this method, as the patient is usually already acidæmic. The œdema in certain cases of nephritis is due to the loss of power of the kidney to excrete salt, in particular the sodium ion. Water is retained in the tissues simultaneously, producing an isotonic solution. These are explanations of so-called *hydræmic* nephritis, and it is not in itself a fatal condition.

2. The true excretory function of the epithelium becomes lost in proportion as the epithelium becomes destroyed, and this can occur without the loss of enough protein to produce hydræmic nephritis. The result is called *azotæmic nephritis ;* but this name gives a one-sided view of the true state of affairs. There certainly is a retention of urea ; but the phosphate and sulphate acid radicles are also retained, so that there is acidæmia with a lowered bicarbonate

content of the blood (*see* p. 547). This is a serious state of affairs and may ultimately lead to *uræmia* where the patient is poisoned by his own retained waste products. Mixed types presenting features both of hydræmic and azotæmic nephritis are common. It is at present not possible to correlate these functional alterations with the appearances of morbid anatomy; but it is possible that in a pure hydræmic nephritis permanent glomerular changes are not prominent, though the tubules may show cloudy swelling and fatty changes. In this connection it is to be noted that tubal epithelium has great powers of regeneration. When glomerular changes begin, as in contracting white kidney (*see* p. 624), some degree of azotæmic nephritis is probably always present, which reaches an extreme degree in the contracting white kidney or the secondary contracted kidney, in both of which the majority of the glomeruli have been converted into mere hyaline nodules.

The changes in the urine which occur more or less in all forms of Bright's disease and nephritis will now be described. They are—(1) Albuminuria. (2) Hæmaturia. (3) The presence of casts in the urine.

## ALBUMINURIA

The term *albuminuria* is used to denote the presence both of albumin and globulin in the urine. It is the most constant sign of Bright's disease, and may be detected in various ways.

**Heat.**—If clear acid urine containing albumin be heated in a test-tube, it will become opaque from the precipitation of this substance. According to its quantity the precipitate will be a mere opalescence, a decided turbidity, or a thick creamy deposit; on the cooling of the urine it will separate into small particles or flakes, and gradually subside to the bottom of the test-tube. The best way of applying the test is to fill a test-tube to one-half or two-thirds of its length, and, holding it by its lower end, to warm the upper part of the urine. The heat is thus confined to that portion of the urine, and whatever slight change takes place in the clearness can be recognised by comparison with the cool urine below; whereas, if heat were applied to the bottom of the test-tube, it would reach, by convection, the whole of the urine at once, and a slight opalescence might, for want of comparison, be overlooked. The value of this method is shown in cases where an albuminous urine is turbid from a deposit of urates. Heat will at first dissolve the urates, and then precipitate the albumin. A long column of such urine may be heated in its upper two-thirds to clear it from the urates, and then further heated in its upper one-third to throw down the albumin, when the three strata of urates, clear urine, and albumin may be compared with one another.

Some precautions are necessary. First, heat may precipitate substances which are not albumin. In certain states of the urine, a precipitate comes down which is to the eye indistinguishable from albumin; it is due to the earthy phosphates (*see* p. 605), and is at once dissolved by a drop of nitric or acetic acid, whereas a precipitate of albumin is unaffected or becomes denser. If the two occur together, there will only be a partial clearance on the addition of nitric acid. Heat also throws down serum-globulin, or paraglobulin, which probably always accompanies the serum-albumin of Bright's disease.

Secondly, albumin, though present, may fail to be coagulated by heat. This occurs when the urine is not sufficiently acid, and the serum-albumin has been converted into alkali-albumin, which is not precipitated by heat; the fallacy can be guarded against by the addition of acetic acid to the urine after boiling, so as to render it acid. In any case the experiment must be performed upon a clear urine: if turbid from urates, a gentle heat clears it; if from phosphates, a drop or two of acetic acid should be added; if from any other deposit, the urine should be filtered.

With these precautions the heat-test is a very delicate test for the presence of albumin in the urine. A few drops of dilute acetic acid should be added to the column of urine in a test-tube: the column should be heated in its upper half: and the upper and lower halves should be critically compared in different lights to see if there is any opalescence in the upper. If the acetic acid itself causes opalescence, it is due to nucleo-protein (*see* p. 616) or euglobulin.

**Nitric Acid.**—This precipitates serum-albumin, as well as alkali-albumin, and acid-albumin. If the albumin is in large quantity, a drop or two of strong nitric acid added to the urine will carry down a thick curdy white precipitate; but for smaller quantities the test is best applied by placing a little nitric acid in the bottom of a test-tube and very gently pouring the suspected urine down the side of the tube, so that it flows on to the surface of the acid without mixing with it. At the line of junction a layer of albumin forms, of white colour if abundant, or a thin grey disc when the quantity is very small. In the former case also it forms at once; in the latter it may take several seconds, or two or three minutes, or even half an hour.

There are but few fallacies connected with this test. First, in urine containing an excess of urates, these are sometimes precipitated as a cloud or haze some distance above the nitric acid, instead of lying immediately upon it; the application of a gentle heat will at once dissolve them. Secondly, nucleo-protein (mucin) is precipitated as a haze in the same position. Thirdly, the urine of persons who are taking copaiba internally contains a resinous acid, copaivic acid, in combination with bases. If nitric acid be added to this the resinous body is thrown down, generally as a cloud, evenly diffused through the urine. This precipitate is also dissolved by heat. Fourthly, in many specimens of urine the addition of nitric acid gives rise after some time, when the mixture has become cold, to a crystalline deposit of nitrate of urea; but this has no resemblance to albumin, consisting, as it does, of lamellar crystals, radiating in various directions. Nitric acid also precipitates the albumoses, which are redissolved by heat (*see* p. 616).

**Picric Acid.**— A test-tube should be more than half filled with urine, and a saturated solution of picric acid, which has a low specific gravity, should be poured on to it so that the liquids may mix as little as possible; at the line of junction a delicate white line, or a thicker white cloud, at once forms, which, if albumin, is not dissolved by heat. Besides albumin, picric acid also precipitates urates, alkaloids, and albumoses. These are said to disappear at once on warming the urine. Quinine is the only alkaloid that is likely to be taken in sufficient quantity to be precipitated by picric acid. Nucleo-protein is also precipitated by picric acid and is not dissolved by heat. If picric acid gives no precipitate albumin is certainly absent.

**Salicyl-sulphonic Acid.**—A saturated solution of this acid is a delicate test for albumin: added to clear urine in a test-tube, it throws down an opalescent cloud of albumin. It is useful for confirming the results obtained by the heat test. Albumoses are also precipitated.

The following are *quantitative tests* for albumin:

**Weight of Precipitate.**—The most accurate method is to precipitate by heat with the addition of so much acetic acid that a protein-free filtrate is obtained as tested by the addition of salicyl-sulphonic acid. The correct proportion of acetic acid must be determined for each urine by preliminary experiments. The precipitate is filtered, washed with boiling water till free from chloride, then boiling alcohol, dried with ether and weighed.

**Esbach's Test.**—This is the best clinical method. A test solution is prepared, consisting of one part of picric acid and two parts of air-dried citric acid in 100 parts of water. A graduated tube from 6 to 8 inches long and ½ inch in diameter is filled up to a certain level (2¼ inches) with urine, and then for a certain distance (1½ inches) with the picric solution. The precipitated

albumin is allowed to settle for twenty-four hours, and the marks on the tube show the amount of albumin contained in 1,000 parts of the urine, *i.e.* grammes per litre. From this, of course, if the daily amount of urine is known, the absolute quantity of albumin passed can be calculated. In some urines the deposit is increased by the presence of uric acid, and thus the albumin may be over-estimated. This method is useful for comparing the variations in the amount of albumin from day to day in a case of nephritis, or for estimating and recording the amount when it is not less than $\frac{1}{4}$ gramme per litre. But it is probably not very exact, and the above apparatus is useless for the estimation of so-called "traces" or "minute traces" or a "mere opalescence," which measure only $\frac{1}{10}$ gramme per litre, or less.

**The Causes of Albuminuria.**—In considering the reason why albumin appears in the urine in Bright's disease, we must remember that the occurrence of albuminuria is not limited to cases of nephritis, but accompanies a variety of other disorders. The different conditions in which albuminuria has been observed may be enumerated as follows:

I. Arising in the kidney—
1. Acute and chronic nephritis, and primary contracted kidney, forming Bright's disease; consecutive nephritis and cystic kidney.
2. Suppurative nephritis.
3. Degenerative changes, such as lardaceous disease and tuberculous kidney.
4. Acute febrile and infective processes and various poisons, probably causing temporary degeneration of the tubal cells.
5. Venous obstruction in diseases of the heart and lungs, and local disturbances of the circulation.
6. Malignant endocarditis, with its associated acute or embolic nephritis.
7. New growths and parasites.
8. Temporary obstruction of the ureters.
9. Nervous disorders such as apoplexy, convulsions, and concussion.
10. Chronic general disorders, like leukæmia, diabetes, and anæmia.
11. Disturbances of digestion, and disorders of a temporary nature, such as that caused by violent exertion.
12. Albuminuria of adolescence (*see* Functional Albuminuria).
13. The influence of certain poisons, and the presence in the blood of forms of albumin other than serum-albumin.
14. Pregnancy (*see* p. 661).

II. Arising in the urinary passages below the kidney—
1. Disease of the pelvis of the kidney, calculous pyelitis, and tuberculous disease.
2. Tuberculous disease of the ureter.
3. Cystitis.

In this last group, in which the albuminuria is always slight, there is no difficulty in explaining its occurrence; it is not infrequently accompanied by blood, and may be the result of rupture of vessels, as in the case of calculus, or it is due to inflammatory and ulcerative processes by which an albuminous secretion is provided in the urinary passages, just as it would be on any other mucous surface or on the skin.

**The Presence of other Proteins in the Urine.**—Besides serum-albumin, other proteins may be found in the urine.

*Serum-globulin* commonly accompanies albumin and is precipitated by heat, by cold nitric acid, and by picric acid. It is often in excess in the urine of lardaceous disease, and may be detected by pouring the urine into a large bulk of distilled water, when the globulin is precipitated, or by saturating the neutralised urine with magnesium sulphate. On the other hand, in chronic parenchymatous

nephritis the globulin in the urine is much diminished relatively to the albumin, the ratio being 1 to 6. In cases of functional albuminuria this ratio is usually 1 to 2.

*Nucleo-protein, Euglobulin.*—If a few drops of acetic acid be added to the cold urine, especially if diluted, a precipitate may form, which was once thought to be mucin, and more recently a nucleo-protein derived from the renal cells. Such precipitates have been considered by Mörner to be compounds of serum-albumin with chondroitin-sulphuric acid, with nucleic acid, and in cases of jaundice with taurocholic acid. On the other hand, they have been thought to be a mixture of euglobulin and fibrinogen (Oswald). The practical importance of this substance, euglobulin, or whatever else it turns out to be, is that it may be the substance that causes functional albuminuria in young persons (*see* p. 660). It is generally agreed that its presence in quantity definitely excludes the case from the category of true nephritis, so that the prognosis is good.

*Albumoses* also occur in the urine, generally in association with albumin, but sometimes without it. They are due mostly to absorption from purulent effusions such as empyema, and large abscesses; to diseases of the liver, especially acute yellow atrophy; to ulceration of the bowel in typhoid fever or otherwise; to acute infectious diseases and acute inflammation, especially pneumonia; to scurvy; and to toxic and puerperal conditions. They may occur in large amount without albumin in some cases of chronic Bright's disease (granular kidney). Albumoses are precipitated by salicyl-sulphonic acid, redissolved on boiling, and again thrown down on cooling. Nitric acid, added gradually, gives a precipitate, soluble on heating, and reappearing in the cold.

Bence Jones *protein*, which occurs in multiple myeloma of the bone-marrow (*myelopathic albumosuria*), and occasionally in myelogenous leukæmia, is coagulated by a lower temperature than albumin, that is 58° C. (137° F.), as compared with 75° C. (167° F.), and the coagulum is re-dissolved as the temperature is raised to boiling. It is precipitated by a saturated solution of ammonium sulphate. Strong hydrochloric and nitric acid throws down an abundant precipitate, which is only dissolved in very great excess of acid, but is soluble on boiling. In smaller quantity, an amount of the protein equal to 0·5 per 1,000 will give a white ring at the junction of the fluids, when the urine is floated on the acid (Bradshaw's test).

*Peptones* rarely, if ever, occur in the urine. They give the biuret reaction—a pink or rose colour when the urine is floated in a test-tube over a small quantity of Fehling's solution—but they are not precipitated by nitric acid, nor by saturation of the fluid with ammonium sulphate, which precipitates the other proteins. This and other salts may be used for the separation of the proteins, since ammonium sulphate precipitates all proteins, except peptones, sodium magnesium sulphate precipitates serum-albumin, and magnesium sulphate throws down serum-globulin.

### HÆMATURIA

In acute nephritis the urine often contains blood or some of its constituents other than albumin. It sometimes gives the urine a bright red colour, and more often a dirty-brown colour and turbid appearance from the presence of methæmoglobin or hæmatin. Generally with this there is a granular reddish-brown sediment. The presence of blood is determined with certainty in several ways.

**The Microscope.**—By this we can recognise red blood cells in cases where there is no suspicion, from the colour of the urine, that blood is present; if the urine is distinctly red, or brown and turbid, the discs will be there in abundance. From suspension in the urine they may have lost their bi-concave form, and are often shrivelled, and have crenated edges, or may present protrusions of their substance. They remain visible for the longest time in acid and dense urines, but may be quickly dissolved in ammoniacal urine, or in urine of low specific gravity.

**The Spectroscope.**—The spectrum of urine containing oxyhæmoglobin shows two absorption bands in the yellow and green portions between Frauenhofer's lines D and E, the narrower, darker, better-defined band being nearer to D. Methæmoglobin gives three absorption bands, two in very nearly the same position as those of oxyhæmoglobin, and a third about half-way between C and D. Acid hæmatin shows a fourth band between E and F.

**The Guaiacum Test.**—To the urine, in a test-tube, are added a few drops of tincture of guaiacum, and then about ½ drachm of ozonic ether or ozonic alcohol, which contain hydrogen peroxide. Quickly or slowly, according to the quantity of blood, a blue colour forms at the junction of the fluids, and diffuses itself through the ether which floats on the surface; its appearance may be hastened by gently shaking the mixture. The test is not absolutely trustworthy. The urine of patients taking potassium iodide will give a blue colour, but it appears only slowly unless the iodide is in very large amount.

It is often desired to know in what part of the urinary passages the hæmorrhage has arisen. In renal hæmorrhage the blood is often intimately mixed with the urine; in bleeding from the bladder it is more separate; in urethral hæmorrhage the blood comes apart from the urine. The diagnosis is also helped by any coagula that may form: in renal hæmorrhage blood-casts of the renal tubules are found, in hæmorrhage from the ureter there may be long stringy coagula, and in vesical hæmorrhage large flat oval clots 1½ inches in length and 1 inch broad with fringed edges.

### CASTS

These are solid bodies, which are detected by the microscope in the urine, and if sufficiently numerous form a sediment visible to the naked eye. Where they are too few to be easily detected, they may be found after centrifugalising the urine. They are cylindrical in shape and from 0·01 to 0·05 mm. in breadth; but they vary still more in their length, which may reach 1 or even 2 mm., so as to stretch right across the field of the microscope, but is more often from five to ten times the breadth. Their connection with the kidney is proved by finding them after death, *in situ*, usually in the junctional tubules. They are probably formed in the loop of Henle, since the lumen of the tube is much narrower here than elsewhere, so that the material becomes squeezed as it passes through. The following varieties are distinguished

**Hyaline Casts.**—These are transparent colourless cylinders, with refractive properties so like those of the fluid in which they lie, that they are discovered with great difficulty, unless they are stained by the addition of carmine or iodine, or one of the aniline dyes, such as gentian-violet. They are homogeneous, soft, and flexible, straight or curved, and varying in length. Occasionally they have other deposits adherent to, or embedded in, them, such as red blood corpuscles, leucocytes, epithelial cells, granular masses, fatty granular globules, crystals of urates or oxalates, or particles of hæmatoidin.

Some of the varieties of casts described below probably have the same hyaline material as a basis, which is then completely covered by, or mixed up with, the other elements.

Hyaline casts are often spoken of as "fibrinous," but it appears that they are not pure fibrin. Most probably they are formed from an albuminous coagulate, which has transuded through damaged epithelium from the blood. More rarely they are formed from tubular epithelium, which has undergone hyaline degeneration.

They occur in the different forms of nephritis, and in the congestion produced by heart disease; they are nearly always associated with the albuminuria of renal origin, but may precede for a few hours or days the appearance of the albumin, and may continue for a time after its disappearance.

**Epithelial Casts.**—These consist of cells of the renal epithelium held

together by, or embedded in, the coagulable material which makes up the hyaline casts. The epithelial cells may be more or less distinct; they may come from the convoluted tubule or the loop of Henle.

**Granular Casts.**—These are not so transparent as hyaline casts, being sometimes like ground glass, sometimes darker and much more opaque. They are formed by the degeneration of epithelial cells, and in many cases it is possible to see the outlines of the epithelial cells in the cast.

**Fatty Casts.** -These are either hyaline casts in which globules and granules of fat are embedded, probably from the disintegration of epithelium in a state of fatty degeneration; or they actually consist of such fatty epithelial cells themselves.

**Blood Casts.**—These result from the coagulation of blood which has been effused into the renal tubules; they are easily recognised by their colour, and by the size and close aggregation of the blood corpuscles composing them. Their presence in a case of hæmaturia proves that the blood comes from the kidney itself.

**Waxy Casts.**—These are large and highly refractive casts, which are sometimes found in cases of lardaceous disease, but also in other chronic forms of Bright's disease. They are brittle, and break irregularly, which gives them rather the appearance of wax. They often do not give the reactions of lardaceous material, and their nature is doubtful, but it is possible that they are a later stage of the granular cast.

Though the character of the casts found in the urine must be determined to some extent by the condition of the kidney, conclusions on this last point must not be too hastily drawn from them. Frequently two or more forms are found in the same urine, and we must be guided by the predominance of one or the other. Hyaline and granular casts are found in all forms of nephritis, acute or chronic. Blood casts and epithelial casts are most common in acute cases; fatty casts are most frequent in cases of chronic nephritis, in which fatty degeneration has supervened.

**Leucocyte Casts.**— This is the name given to casts in which pus cells are embedded.

**Cylindroids.**—These are large hyaline bodies with tapering or branching ends, a wavy outline, and longitudinal striation: they are probably threads of mucus, the undissolved residue of nucleo-proteid, and not really casts. They have no significance.

## ACUTE NEPHRITIS

**Ætiology.**— The cause of the most important group of cases to which the term *acute nephritis* is applied is at present unknown, although exposure to cold and damp, particularly if the patient is under the influence of alcohol, is sometimes regarded as a predisposing cause. There is, however, a general consensus of opinion that the disease is due to infection, though whether the kidneys are themselves the place where the organisms are growing or are solely affected by toxins which arise from micro-organisms growing in quite a different part of the body is an open question. In many of these cases the tonsils are thought to be the seat of the infection. A very good example of the type of nephritis arising apparently spontaneously is the so-called "trench nephritis," which occurred among soldiers in the recent war; it was not confined to soldiers in the trenches. Similar outbreaks have occurred in other campaigns, notably the American civil war. There is no essential difference between trench nephritis and the acute nephritis of civilian practice. In other cases the infective origin of acute nephritis is more obvious. Scarlet fever is a common cause of the condition, and in this case children are more often the subjects than adults. As a rule, the symptoms are first observed during convalescence, and here again they have been attributed to cold; but nephritis also arises in cases which have

been most carefully nursed, and have not been exposed. Sometimes it commences before convalescence is established.

Several other febrile diseases are from time to time, but much less frequently than scarlatina, the causes of acute nephritis. Of these cases also a very small proportion develop into a typical Bright's disease, with dropsy, secondary inflammations, uræmia, etc., but the majority present only temporary changes in the urine, with but slight constitutional disturbance. They are measles, variola, cholera, varicella, typhus, enteric fever, pneumonia, relapsing fever, erysipelas, Henoch's purpura, tubercle, syphilis, and septic conditions. Diphtheria is frequently accompanied by albuminuria, sometimes clearly indicative of nephritis. The micro-organisms of some of these infections have been found in the urine, *e.g.* pneumococci and typhoid bacilli. Pregnancy is not infrequently the cause of a nephritis, which may be of the most severe type, with uræmia and retinal changes; it probably acts by means of toxins.

A number of substances taken internally set up irritation of the kidney, which, according to the dose, may be severe congestion, or may amount to a definite nephritis. They are cantharides, turpentine, potassium nitrate, salicylic and carbolic acids, mercury salts. Indulgence in alcohol, though probably more often contributing to the chronic form of nephritis, occasionally seems to produce an acute inflammation. Uranium nitrate has been used experimentally to produce nephritis in animals.

**Morbid Anatomy.**—According to Gaskell's classification of the various types of acute and chronic nephritis, there are four distinct types of acute inflammation of the kidney. *Glomerulo-tubular nephritis* is the name given to what is commonly called acute Bright's disease. This will be described at length. *Acute interstitial nephritis* is found specially in the kidneys of patients who have died of scarlet fever early in the disease, but have not developed typical scarlatinal nephritis. The interstitial tissues are infiltrated with round cells, chiefly plasma cells. In *toxic nephritis* which occurs in pregnancy, and is also due to poisons, the tubular epithelium is especially attacked, while the glomeruli show little change. *Acute embolic local nephritis*, which is seen in infective endocarditis, has already been described (*see* p. 331). However, it must be understood that these terms only indicate the structures in the kidney which are chiefly affected. Slight changes may be seen in other parts as well. Thus in acute interstitial nephritis the parenchyma is also affected to a small extent, and similarly in toxic nephritis the glomeruli and interstitial tissue show some changes.

*Glomerulo-tubular Nephritis.*—In some cases the kidney may be of normal size and of normal appearance to the naked eye—a remarkable fact considering that the patient has died very often from virtual suppression of urine. This has been noted in cases of trench nephritis and scarlatinal nephritis when death has occurred rapidly. In other cases the kidney is more or less swollen, it may be twice its normal size, it has a rounded form, is tense and elastic, the capsule strips readily, and the surface is paler than normal. On section, the cortex is considerably swollen, and of greyish-red colour, while the pyramids are dark red from congestion. Here and there are bright red spots: some of them are congested glomeruli, others are small hæmorrhages. In yet other cases the kidneys are intensely congested, dark red or chocolate in colour, dripping with blood, and showing on section a still more extreme congestion of the pyramids.

In the most acute and fatal cases of trench nephritis it was remarkable how little was seen under the microscope. The changes were confined to the glomeruli (*glomerulo-nephritis*). There was proliferation of the endothelial cells of the tuft, with increase in its size so that it completely filled the capsule and was herniated into the passage leading to the tubules. No blood was seen, except very occasionally when there was an infarction of the whole glomerulus, the capillaries being dilated and engorged with corpuscles. Similar appearances have been noted in scarlatinal nephritis.

When the tubules are also affected, in cases which are not quite so acute, the epithelial cells become swollen and granular (*cloudy swelling*), and in more advanced conditions are filled with fat granules, or become necrosed, and are separated from the wall of the tubule (desquamation). The tubule is then filled or even distended by accumulations of such altered cells, mixed with albuminous fluid, leucocytes, and granular *débris*, and here and there blood escapes into them from distended intertubular vessels. Many tubes, both in the cortex and in the pyramids (*e.g.* Henle's loops), are filled with hyaline or other casts. The distension of the tubes gives a pale colour to the portion of the kidney affected, partly from the opacity of the fat granules, partly from compression of the intertubal vessels; on the other hand, such compression increases the congestion of the glomerular tufts.

The size of the largest kidneys is perhaps due to interstitial exudation. This may be either a general infiltration with inflammatory fluid, or the extravasation, more or less irregularly, of leucocytes from the blood vessels.

**Pathology.**—There is interference with most of the renal functions during an attack of acute nephritis, but there is much variation from case to case. Owing to failure to excrete non-protein nitrogenous substances, there is a corresponding retention of them in the blood: this is shown by the rise in the urea content of the blood, which is considerable in some cases (180 milligrammes per cent.), though it is slight in others. The œdema is probably due to the lowered osmotic pressure of the plasma, which is itself secondary to loss of protein from the kidneys (*see* p. 612). There may or may not be some deficiency in the excretion of diastase. A rise of blood pressure, both systolic and diastolic, accompanies the œdema in acute nephritis. The systolic pressure may reach on occasions a value of 180 mm. The rise is greater at night than in the morning. Its probable effect is to increase the circulation of blood through the kidneys, and so help to keep up the flow of urine. It rapidly falls to normal when the œdema subsides and the volume of urine is increased.

**Symptoms.**—The onset of symptoms is often insidious. Very often œdema is the first symptom noticed by the patient or his friends. The face looks puffy, the fluid collecting beneath the eyes in particular, when the patient rises in the morning. It may subside in the course of the day, but if the feet be examined at night there is œdema just below the ankles. There is, indeed, a small quantity of effusion into the subcutaneous tissue, which always seeks, by force of gravity, the most dependent part. During the daytime it reaches the feet; in the recumbent position of sleep it diffuses itself generally, but is most pronounced in the loose tissues of the eyelids. If the patient takes to his bed, it will leave both the face and the feet, and accumulate in the tissue over the sacrum, which has now become the most dependent part. The puffiness of the face is in marked contrast to the onset of œdema in cardiac failure. In the latter case the fluid, which is poured out in excess in the neighbourhood of the heart, gravitates downwards, becoming first of all visible in the legs. The duration of the œdema of acute nephritis varies greatly. In mild cases it may disappear in a day or two. On the other hand, it may continue, and the case eventually becomes one of parenchymatous nephritis. Associated with the œdema there is diminution in the volume of urine and albuminuria. In the worst cases only a few ounces of urine of high specific gravity may be passed during the day, and it may become solid on boiling owing to the high concentration of albumin. In milder cases the volume of urine is greater, and it may be turbid and have a colour which is due to the presence of fresh or altered blood, and is dusky brown, deep brown, "porter-coloured," pink, or distinctly red, according to the quantity and condition of the blood. In some cases hæmaturia may be present with no more albuminuria than would be accounted for by the blood. The volume of urine is often not diminished in such cases. The presence of plenty of blood is often a favourable element in the prognosis, because it means that there is a free flow of blood through the kidneys. The urine deposits a sediment of fresh

or altered blood corpuscles, or fragments of them, renal epithelial cells, hyaline, granular, epithelial, or blood casts, granular *débris*, and, it may be, after some time uric acid crystals. Fatal cases are usually associated with considerable diminution in the volume of urine, and not much blood. It has already been pointed out that microscopic sections may show the almost complete absence of blood from the glomeruli in acute cases.

Quite early the pulse becomes hard, and the blood pressure is raised, sometimes up to 180 mm. The heart sounds are modified. The first sound (systolic) increases in intensity, and the second sound (over the base of the heart) is also accentuated or " ringing " in character.

The patient may feel unwell; there may be some rise of temperature; there is anorexia and usually vomiting. Headache is complained of. There is also pain in the loins, and very often over the epigastrium. Anæmia may be present. Dyspnœa was a symptom that was very frequently noticed in trench nephritis. Œdema of the lungs may have accounted for these symptoms in some cases. In severe cases the heart dilates, and the impulse may be displaced outwards.

*Uræmia in Acute Nephritis.*—In those cases where the suppression of urine is considerable *uræmia* may supervene. (1) This may be similar in its biochemical changes to the uræmia associated with chronic interstitial nephritis, which has been called the chronic or asthenic type of uræmia (*see* p. 629). (2) But there is another special type of uræmia called the *acute* or *sthenic* type, or sometimes *eclamptic pseudo-uræmia*, which is very characteristic of acute nephritis, and which is not associated with a high urea or phosphate retention in the blood. In this type headache and vomiting become more severe, the pulse is slow, and there are *convulsions* or *eclampsia*. Experience of trench nephritis during the war showed that these symptoms were due to cerebral œdema, since they could always be stopped by lumbar puncture, and the cerebro-spinal fluid was under considerable pressure. The attacks have a very close resemblance to the ordinary attacks of epilepsy. There is often a short tonic stage, and then general clonic convulsions of all the muscles of the limbs, face, eyes, and trunk. Consciousness is lost, the face becomes livid, there is frothing at the mouth, the saliva may be tinged with blood, and the pupils are dilated. After some minutes the convulsions subside, and the patient may at once regain consciousness, and then improvement in the general condition may begin, for convulsions are by no means always an unfavourable sign. In other cases the patient lapses into a state of *coma*, from which he may again pass into convulsions; and these are repeated again and again with intervals of complete coma in which he may die. During the convulsion the respiration is hurried, and the pulse is small and quick; the temperature is variable, and it may reach 104°, or higher. This sequence of events may be repeated several times. A temporary *paralysis* is sometimes observed. *Delirium* or mania is another form of disturbance which results from uræmia, but it is not so common as the convulsive and comatose symptoms; it may follow the fits. It is also after convulsions that blindness (*uræmic amaurosis*) generally occurs; it rarely precedes the fits, or happens without them. It may last from one to three days, and frequently passes off entirely. As the pupil reactions are unaffected, it is probably due to the influence of toxins upon the higher visual centres. Deafness may be also noticed.

*Ocular Changes.*—In the majority of cases of acute nephritis no changes in the retinæ are observed, with the exception of small more or less circular hæmorrhages, which, however, are apt to occur in people failing in health. Out of 119 soldiers suffering from trench nephritis, Foster Moore observed definite eye changes in only five, coming on about two months after the beginning of the illness. The blood pressure was higher (average 180 mm.) in these cases than in the remainder (average 143 mm.). The changes consisted of exudations at the back of the retina, leading often to its detachment. They were seen as pale, soft

edged, oat-shaped swellings, arranged radially in some cases so as to form a star. Later on these swellings coalesced as the exudations became more extensive. Hæmorrhages were also present, and the edges of the optic disc were blurred by exudations (papillitis). In severe cases the swelling of the disc is so marked as to conceal completely the retinal vessels, and resemble the papilloedema of cerebral tumour. More often there is slight prominence, but diffused opacity spreads far on to the surrounding retina. These various changes are seen more often in chronic parenchymatous nephritis. They may be regarded as being due to toxic action. Atrophy of the retina and papilla may follow on the preceding inflammation. See Plate VIII., p. 368.

**Course.**—In the majority of cases there are no uræmic convulsions at all. After a few days or a week or two improvement sets in, often quite suddenly. The volume of urine is greatly increased as the dropsy disappears; the blood pressure falls to normal. The albumin and blood disappear more gradually, small amounts being passed for weeks afterwards.

But the course of acute nephritis may be less favourable in several ways. In a small proportion of cases death follows rapidly from uræmia.

In other cases the symptoms may continue, so that eventually one of the two chronic types of nephritis is established. It has already been mentioned that there is derangement of all the renal functions during the acute attack, but certain of these recover first. In some cases there may be early diuresis or disappearance of œdema, but the patient still remains ill. The percentage of urea in the blood remains high, or if this is not greatly raised, the power of the kidneys to concentrate urea is deficient. The diastase in the urine is low. The volume of urine is normal or increased. It contains albumin, but not in very large amounts. Such is the picture of the *azotæmic* type of nephritis, and it eventually leads to secondary contracting or possibly secondary contracted kidney.

On the other hand, the œdema may continue. The percentage of urea in the blood soon becomes normal. The volume of urine is decreased, but the percentage of albumin is large. Such cases conform to the *hydræmic* type of nephritis, but in using this term it must be remembered that the blood is not always watery. Chronic parenchymatous nephritis becomes established in such cases. Effusion takes place into the peritoneum, pleural cavities, and pericardium. The lungs at the same time are partly œdematous, partly compressed by the pleural fluid, and death may take place from interference with their functions. Sometimes the excessive distension of the skin, especially in the lower extremities, causes gangrene and sloughing, or erysipelas occurs around the punctures which have been made to relieve tension; and death in either case may follow. In other instances, again, the acute serous inflammations, pleurisy, pericarditis, and peritonitis, are fatal; or acute œdema of the glottis brings about asphyxia, unless promptly relieved by tracheotomy.

In other cases there is a mixture of these two types.

**Diagnosis.**—This rarely presents any difficulty. The sudden appearance of a general dropsy, with albuminous urine, in one previously quite healthy, or recovering from scarlatina or other fever, can scarcely be mistaken for anything else. If there is a small quantity of albumin only in the course of an acute disease, this may be due to a change in the epithelium, not usually regarded as constituting nephritis, though it is not essentially different from what actually takes place in the typical conditions. Lesions of the *lower urinary passages* may cause bloody urine and albuminuria. Dropsy and casts are absent, but there is pus in the urine. *Hæmoglobinuria* causes a red urine, in which, however, there are no blood corpuscles. Rarely the dropsy of nephritis occurs without albuminuria, especially in children; but there is usually hæmaturia. Such cases must be distinguished from some insidious diseases (tuberculosis, leukæmia) which have other characteristic lesions.

**Prognosis.**—It is, on the whole, favourable, as there are a great number of

mild cases which recover in a few weeks, and some even after months. Some cases are fatal in the acute stages, and others run on into chronic nephritis. The unfavourable indications are excessive dropsy, very scanty or suppressed urine, high urea in the blood, very high tension of the pulse, asthenic uræmia, or the later condition of feeble pulse with obviously failing heart, hydrothorax, and serous inflammations. But there are scarcely any of these serious dangers from which recovery may not take place, and sometimes, after living for months on the verge of death, the patient may ultimately get quite well.

**Treatment.**—The direct treatment of the inflamed kidneys, either locally or by drugs, is, as a rule, not attempted. Only exceptionally, where there is much lumbar pain or free hæmorrhage from the kidneys, may dry-cupping of the loins be employed, or for the former symptoms a hot poultice or fomentations.

The usual treatment of nephritis consists in keeping the patient at rest, removing all sources of irritation, limiting the supply of food likely to put a strain upon the excreting functions of the kidney, and supplementing these functions as far as possible by the use of other secreting organs—the skin and the bowels. In addition, we have to deal with special symptoms and complications as they arise. Rest in bed in a warm room diminishes the strain upon the heart, while the secretion from the skin is likely to be promoted.

The diet is best confined to milk and farinaceous foods in the early stage of the disease. If this continues more than a week or two a more liberal diet can be allowed. This depends on the type of disease. In the azotæmic variety the intake of protein food should be diminished, but meat, fish and eggs can be allowed in moderation. In the hydræmic form there is no necessity to restrict the protein in the food, and an ample dietary should be allowed to cover the loss of protein in the urine.

In mild cases the medicinal treatment should be a saline diaphoretic, such as liquor ammonii acetatis or liquor ammonii citratis, in doses of 3 or 4 drachms every four hours; and the bowels may be kept active by occasional purges of pulvis jalapæ co., 20 to 40 grains, or senna and sulphate of magnesia. Lemonade may be given as a beverage, or the Imperial drink, which may act mildly on both the bowels and the skin. This consists of a drachm or a drachm and a half of cream of tartar dissolved in a pint of boiling water flavoured with lemon peel and sugar, and drunk, *ad libitum*, when cold.

If uræmic convulsions set in, the patient's tongue should be protected as in epilepsy, by placing a piece of stick between the teeth. Probably the most valuable of all methods of arresting the convulsions is by lumbar puncture. This was definitely established in trench nephritis. A few whiffs of chloroform will also check them quickly, and though they may return on the removal of the anæsthetic, the effect may be kept up by its occasional use in small quantities, and thus a great deal of the violence of the fit is prevented. Bleeding from the arm will also sometimes stop them, and though it is, as a rule, undesirable that patients with pronounced renal disease should lose much blood, this remedy may be properly used if the convulsions are violent and persistent. The blindness which may occur after convulsions generally recovers of itself. Coma should be treated by purgatives and by subcutaneous pilocarpine injections. Vomiting will require effervescing mixtures, dilute hydrocyanic acid, or tincture of iodine in 2 or 3-minim doses every hour.

During convalescence iron should be given to restore the condition of the blood, but it is not well to use it in the early stages. The perchloride and the ammonio-citrate are suitable preparations. Attempts to lessen the amount of albumin by drugs, such as lithium citrate, sodium benzoate and tannate, strontium lactate and others, have not been very successful. The patient throughout should be carefully kept from cold, and should, on getting up, wear warm clothing with flannel next the skin. The treatment of dropsy is considered in the next section.

## CHRONIC PARENCHYMATOUS NEPHRITIS

*(Chronic Tubal Nephritis, Large White Kidney)*

**Ætiology.**—This condition may appear as a sequel to acute nephritis, but it also arises insidiously, there being no history of an acute attack. A predisposition to its occurrence possibly arises from cold and damp and habits of intemperance. Toxic and infectious agencies are very probable causes, though it is not generally evident what these are in particular cases; malaria, syphilis, and tuberculosis may be mentioned in this connection. Parenchymatous nephritis occurs from childhood to middle age. It is not so common in elderly people, and males are more liable than females.

**Morbid Anatomy.**—The *large white kidney*, or inflammatory fatty kidney of chronic tubal nephritis, is larger than normal—the two together may weigh as much as 800 grammes; often they weigh 500 grammes. It is smooth on the surface, and the capsule strips easily; it is of yellowish or greyish-white colour, and is covered with venules radiating from a central point (stellate veins). On section, the cortex is broader than normal, of the same colour as the surface with an appearance of coarse striation, while the pyramids are more or less dark red. Here and there are red spots, due to hæmorrhage into the tubes, and these are in some cases so abundant as to justify the term *hæmorrhagic nephritis*. Microscopically marked changes are seen in the convoluted tubules. The cells are either very much distended with fluid (hydropic degeneration), or else they contain fat droplets (fatty degeneration). The cells also become detached from the basement membrane. Epithelial and granular casts occupy the straight tubes This distension of the tubes with opaque material, and the compression they exert on the vessels, account for the white or grey colour of the cortex. The glomeruli may look comparatively normal: On the other hand, in other cases, they may show evidence of progressive inflammation. There is proliferation of the endothelial cells covering the glomerular tuft and also of those lining the capsule. In cases of some standing these layers coalesce, obliterating altogether the lumen of the glomeruli; the tuft may eventually become hyaline. The intertubal tissue is irregularly infiltrated with leucocytes; these interstitial changes, leading to fibrosis, become more pronounced as the case progresses. The kidneys of such a case are sometimes called *contracting white kidneys*. The "myelin kidney" is a particular form of fatty degeneration of the kidney, which may be due to syphilis. A doubly refracting lipoid fat, consisting of cholesterol esters, is deposited in the connective tissue spaces (**3**). Doubly refracting fatty casts may be found in the urine.

**Symptoms.**—If the disease follows acute Bright's disease, there is a continuance of the symptoms already described under acute nephritis: general dropsy, effusion into the serous cavities, scanty albuminous urine. Or there may be an apparent recovery from the acute attack, and after a short interval the symptoms recur.

In primarily chronic cases the beginning is more or less insidious. Pallor, loss of appetite, nausea, headache, and frequent micturition are the first signs, and then œdema may be observed in the lower extremities at night, and around the eyelids in the morning. The dropsy gradually increases.

In advanced cases the dropsy becomes general, and the skin of the whole body is œdematous. The face is full and rounded; the eyelids are distended, and almost close the eyes; the limbs become enlarged, shapeless, and remind one of bolsters; the trunk is enlarged; the loose skin of the penis and scrotum is so stretched that the prepuce looks like a bladder, and the scrotum may attain the size of a fœtal head. Wherever slight pressure is applied, as by the finger of the doctor, or by the bands, strings, or folds of clothing, it produces by the displacement of the fluid a deep impression, which is only slowly effaced by the

return of the fluid. This is called *pitting on pressure*. Even then the influence of gravitation on the distribution of the dropsy may be seen, for if the patient lies for any length of time on one side, the arm of that side will become more swollen than the arm which is uppermost; and the same will happen to the side of the face. When this general dropsy, or *anasarca*, is present, there is, as a rule, some effusion into the peritoneal cavity (*ascites*), into one side, or more frequently both sides, of the chest (*hydrothorax*, dropsy of the pleura), and it may be into the pericardial sac. Of these the first is perhaps most often recognised, while the pericardial and pleural effusions may be comparatively slight.

If incisions be made into the skin, or if one or more Southey's drainage tubes be inserted, a quantity of fluid will drain away, which may amount to 8 or 10 pints in a few hours, and the dropsical limbs will rapidly get smaller. The fluid is colourless, of low specific gravity (1,007 to 1,012), and contains a small quantity of albumin, inorganic salts, and urea. A feature which is constantly present in renal dropsy is a high degree of pallor—the lips are almost colourless, the cheeks are pallid, and the whole body has a waxy whiteness. This is partly due to the dilution of the blood and its poverty in red corpuscles, partly to the distension of the skin and subcutaneous tissues.

Epstein's theory of renal œdema has already been alluded to (*see* p. 612). Such a quantity of albumin is being lost in the urine that the blood becomes impoverished of serum albumin, the serum globulin remaining the same as before. There is normally a balance between the blood in the capillaries and the tissue fluids, which prevents any excessive exudation of fluid from the capillaries. The osmotic pressure of the blood proteins plays a part in this balance. In parenchymatous nephritis the osmotic pressure is lowered owing to loss of serum albumin, and hence water and dissolved salts pass into the tissues, the protein content of the dropsical fluid being very low. Epstein also finds an excess of lipoids in the blood, and it is well known that some cases show lipæmia, the blood being quite pale from fat droplets in the plasma. This lipæmia is due to mobilisation of fat from partial starvation of the tissues. Where the lipoids disappear from the cells fluid is imbibed to take their place, and this helps to increase the œdema This theory has an important application in the treatment of the disease.

The urine in parenchymatous nephritis is scanty, highly albuminous, sometimes bloody, is deficient in urea, and contains casts. Its quantity varies from 10 to 25 ounces, though in later stages for a time it may be more abundant. The albumin forms a precipitate which occupies one-third to one-half of the boiled urine, really amounting to 2 or 3 per cent. by weight, or a daily discharge of from 100 to 400 grains. The specific gravity is at first rather high (1,015 to 1,025), but later, with the progress of contraction, it becomes much lower. The casts are granular, hyaline, and epithelial; and fatty casts are numerous in the cases of large white kidney with much fatty degeneration of the renal epithelium. They are accompanied by leucocytes, loose epithelial cells, and granular *débris*. In some cases the urine contains much blood.

The percentage of urea in the blood may be normal, which is a great contrast to chronic interstitial nephritis. But in long-standing cases, by the time the glomeruli begin to show the structural changes already described, some of the features characteristic of secondary contracted kidney may be seen, such as defect in urea excretion with values in the blood of, say, from 0·04 to 0·08 per cent., cardio-vascular changes and diminished urinary diastase. The retinal changes described under Acute Nephritis are present in some cases. Extensive œdema of the retina may lead to its detachment.

In chronic parenchymatous nephritis, as also in acute nephritis, there is a tendency to inflammation of the serous membranes. Pleurisy is the most common; pericarditis is often the precursor of a fatal termination; acute peritonitis is perhaps more rare, unless it follows tapping the abdomen. Chronic inflammation of the peritoneum covering the liver (perihepatitis) has already been

referred to in connection with Bright's disease. Bronchitis is a common, and endocarditis an occasional, complication; pneumonia not infrequently occurs towards the end. Ulcerative colitis is rather a characteristic complication. Several lesions of the skin may complicate Bright's disease—namely, (1) pruritus and urticaria, the former especially in early stages; (2) eczema; (3) an acute general dermatitis, with free desquamation, not unlike exfoliative dermatitis; (4) patches of erythema affecting the dropsical limbs; (5) erysipelas; (6) purpura or hæmorrhage, followed by necrosis and ulceration, conditions which have also been observed in the alimentary mucous membranes; and (7) very rarely the skin has the appearance of having been dusted with flour or pounded sugar. This is probably due to evaporation of the sweat, leaving crystals of salt on the skin.

**Course.**—In the majority of instances, the disease is fatal; but uræmia is not commonly the cause of death. This is in most cases due to complications, such as pleurisy, pericarditis, pneumonia, œdema of the lungs, œdema of the glottis, increasing dropsy, or inflammation or sloughing of the skin; and this commonly occurs in from six to eighteen months, though some cases go on for two or three years. In a few instances the disease may disappear completely. In long-standing cases the various changes characteristic of secondary contracted kidney may occur.

**Diagnosis.**—When acute nephritis changes into chronic parenchymatous nephritis, the characteristic changes in kidney function are established quite early in the disease, so that a distinction between the two is rather artificial. From four to six months is usually regarded as the duration of the acute disease, so that chronic parenchymatous nephritis would be diagnosed if the disease had lasted longer than this. There will be no difficulty in differentiating it from secondary contracted kidney. It might be confused with lardaceous disease or primary heart disease with albuminuria. From lardaceous disease it is distinguished by the absence of such causes as prolonged suppuration, phthisis or syphilis, and by early scantiness of urine and abundant deposit. The similarity between this and heart disease may be close, especially when there is much œdema in the latter case. The presence of abundant casts in the urine is an indication of nephritis. The history, the general character of the dropsy, and the large quantity of albumin will help to distinguish primary renal disease from secondary albuminuria.

**Treatment.**—This is not essentially different from that of acute nephritis. Rest in bed, flannel clothing to promote warmth and a free action of the skin, are actually essential in the early stages. The bowels must be kept active and diaphoresis excited. Mild diuretics are sometimes given, potassium citrate, and even caffeine citrate and thiocin sodium acetate in small doses.

Diminution of the dropsy is sometimes obtained by the use of a diet as free as possible from sodium chloride, for one cause of dropsy is the retention of water in the tissues by the sodium chloride which the diseased kidneys fail to eliminate; and, indeed, healthy persons have sometimes had œdema from eating excessive quantities of salt. But abstention, if pushed too far, may cause much prostration. If dropsy is considerable, treatment by purgation and diaphoresis must be more decided. Diaphoresis should be promoted by hot-water bottles or by the hot-air bath. The *hot-air bath* is administered by raising the bedclothes from the patient by means of a low cradle and fitting them close about his neck and round the sides and end of the bed. The heat may be supplied by a number of electric light bulbs placed inside the bedclothes or by burning a spirit lamp under a funnel connected with a tube which leads under the clothes. The exposure should be from fifteen to twenty minutes; but it is desirable to take the temperature of the patient, as it may be inconveniently raised, if free sweating does not occur. The most efficient diaphoretic drug is the nitrate of pilocarpine, of which $\frac{1}{6}$, $\frac{1}{4}$, or $\frac{1}{3}$ grain may be injected subcutaneously once a day.

In extreme dropsy, especially where the skin is tense and threatens to become inflamed or to slough, the dropsical fluid may be removed under antiseptic conditions, either by small incisions with a lancet or punctures with a needle, or by the use of Southey's tubes. Two or more of these may be placed in each leg, and by this means several pints of serum may be withdrawn in a few hours. Only occasionally, in extreme cases, is it desirable to tap the abdomen or to aspirate the pleural cavity.

The treatment of parenchymatous nephritis along the lines of Epstein's theory may produce very remarkable results in lessening the œdema and promoting diuresis. The diet given is very rich in protein, with an almost entire absence of fat. A little carbohydrate is allowed. It consists of lean meat, lean ham, whites of eggs, oysters, jelly, lentils, split peas, green peas, rice, oatmeal, skimmed milk, coffee and tea, salt in moderation and fluid as reasonably desired. The administration of urea in 15-grain doses by the mouth has also been recommended for its diuretic action. Anæmia in prolonged cases should be met by the use of iron preparations, such as the iodide, tartrate, or ammonio-citrate. In the more chronic forms, with albuminuria but little dropsy, benefit may be derived from residence in warmer climates—Bournemouth or Tenby in the British Isles, the south of France, Italy, or Egypt.

Cases of chronic Bright's disease have been treated by Edebohls in America, by exposure of the kidney, decapsulation, and fixation of the organs; and the results of this surgical treatment have sometimes appeared to be good. Cases of parenchymatous nephritis are most suited to this procedure.

## CHRONIC INTERSTITIAL NEPHRITIS

*(Granular Kidney)*

Three separate types of kidney disease are considered under this heading, using Gaskell's classification. They all have this point in common, that the substance of the kidney shows more or less fibrosis. One of these types is due to inflammation of the kidney; the other two are due to vascular changes.

### SECONDARY CONTRACTED KIDNEY

This type is called "secondary," since it arises as the result of inflammation of the kidney. It is often spoken of as contracted white kidney or small white kidney to distinguish it from the red granular kidney described next. There is, however, a consensus of opinion that the mere colour of a kidney is no criterion for its structure or origin, and so it is better omitted from the name. Secondary contracted kidney may arise directly from an acute attack of nephritis; and it has been pointed out that while the disease is still in the acute stage certain functional alterations in the kidney, such as urea retention, may be observed which are characteristic of the type at present considered. In other instances the patient may have had an acute attack of nephritis, which has apparently completely cleared up, and many years later symptoms pointing to the last stages of granular kidney are observed. In yet other instances—and this is probably the commonest occurrence of all—there is no history of any acute disease at all; the kidney changes are believed to have arisen insidiously from chronic inflammation lasting for years. Finally, it must be remembered that some fibrosis is common enough, in chronic parenchymatous nephritis which has lasted a long time. With the kidney changes there are associated well-marked cardio-vascular changes, the heart being hypertrophied and the arteries thickened. Secondary contracted kidney often occurs before middle age. The patients often die of uræmia. In rare instances it has been described in children associated with delayed rickets and infantilism.

**Morbid Anatomy.**—The size of the kidney varies: it may be normal or not much smaller, or it may be considerably reduced. Its surface is rough or granular, the capsule adherent, the colour yellowish-white or more or less mottled with red in the areas of depression between the granules, or it may be dark and reddish. The cortex, on section, is narrower than normal, and may be pale or reddish. Microscopically, the intertubal tissue is partly infiltrated with leucocytes, partly changed into connective tissue, which is more or less uniformly distributed; and it is by the contraction of this tissue that the granular condition is brought about, and the organ becomes smaller. A large number of glomerulo-tubular systems are destroyed; these glomeruli are hyaline and are blended with the surrounding tissue, which is fibrous and contains the atrophic remains of the corresponding tubules. Other glomeruli have still functioned to some extent, but they show evidence of previous inflammation with adhesions between the tufts and the capsule, so that the capsular space is broken up into pockets. The tubules corresponding to these glomeruli are greatly distended and lined with flattened epithelium. Some of them have been converted into cysts. In other tubes the epithelium has desquamated and forms casts. The small arteries are thickened and show the changes which have been described under Diffuse Hyperplastic Sclerosis (*see* p. 366). This is part of the general arterio-sclerosis that is secondary to the chronic inflammation of the kidney. Lardaceous changes of slight extent are sometimes present. It is possible that chronic inflammation accounts not only for the fibrotic changes in the renal parenchyma, but also for the intimal proliferation and fatty degeneration in the arterioles, characteristic of diffuse hyperplastic sclerosis (Evans). The hypertrophy of the media in this condition may be secondary to the raised blood pressure, as described in the next section.

**Pathology.**—The test for œdema formation (*see* p. 610) shows that there is usually in these cases no tendency to œdema; but there is a difficulty in excreting urea, and particularly in producing a high concentration of urea in the urine. This is shown by the urea concentration test; the diastase in the urine is also low. It is probable that this loss of function is due to obliteration of the smaller arteries. Injections of kidneys have been made with bismuth salts, and then photographs have been taken by X-rays. Extensive obliteration of the vessels in the cortex has been shown by this means.

There are two ways in which the body compensates for this loss of function. In the first place, the quantity of blood circulating through the kidneys is increased. This is brought about by a rise of arterial blood pressure, by means of a hypertrophied heart and constricted arteries. The weight of the heart varies from 450 to 800 grammes, and the hypertrophy in the more marked cases affects the right as well as the left ventricle. The smaller arteries show hypertrophy of the middle coat. By this means a free flow of urine is produced in spite of the reduction in the arterial bed. In the second place, the concentration of urea in the blood is increased, and this facilitates its excretion by the kidney, so that a balance takes place, the output of nitrogenous substances corresponding accurately with the amount in the food. The concentration of urea is normally 20 or 30 milligrammes per cent. Values of 100 milligrammes and over are frequently found in this disease when the patients are up and doing their ordinary work. Any great increase in the protein of the food throws more work on the kidney, and this can only be performed by some additional retention of urea, so that the percentage of urea in the blood is still further increased. The balance between intake and output becomes re-established, the kidney responding to a higher threshold value of urea in the blood. The importance of limiting the protein of the food will be at once evident.

If the lesion progresses the urea in the blood becomes higher and higher. The same thing will be caused by an acute attack of nephritis supervening on the chronic condition. Symptoms of intoxication appear when the concentration

of urea is too high. These may begin when the urea is about 200 milligrammes per cent. In the fully developed uræmic condition values for the urea of 300, 400, or 500 milligrammes per cent. have been observed. The symptoms are characteristic, and have been called *asthenic uræmia* or *chronic uræmia*. They are quite distinct from the uræmic attacks of acute nephritis. There is some evidence that chronic uræmia is, at any rate partly, due to poisoning by urea. Hewlett, Gilbert and Wackett have taken 100 to 125 grammes of urea by mouth within a few hours. A notable rise of the urea in the blood took place, values up to 200 milligrammes per cent. being observed. Definite symptoms—headache, dizziness, drowsiness, indifference, fatigue and prostration—were noticed when the concentration of urea in the blood was highest, strongly reminiscent of the symptoms of chronic uræmia. Since only 10 grammes of urea would be required to raise the concentration in the blood up to 200 milligrammes, most of the urea ingested must have been in the tissues. But other metabolic alterations are observed in chronic uræmia, and these play a part in the symptomatology. Acid in the form of phosphate and sulphate accumulates in the blood, so that the alkali reserve is lowered (*see* p. 547) (**8**). The alveolar $CO_2$ is also diminished, but not in proportion to the accumulation of other acid in the blood, so that the acidity (hydrogen ion concentration) of the blood is increased (*acidæmia*). Other unknown acids and metabolites may also be present in the blood. There is evidence that the acidæmic state is not by itself reponsible for uræmic coma, because acid values as high have been observed in other patients non-comatose. Air hunger or extreme hyperpnœa is the consequence of the acidæmia, the $CO_2$ in the alveolar air and blood being lowered by this means.

Chronic uræmia is a characteristic mode of termination in secondary contracted kidney, but it is not peculiar to it. It may occur in other forms of granular kidney and in consecutive nephritis, particularly where there is suppuration in both kidneys, and occasionally in acute nephritis.

**Symptoms.**—Symptoms often arise insidiously. There is no œdema. The volume of urine is increased, and the patient has to get out of bed once or twice at night to pass water. Albuminuria is present, but the percentage of albumin is never as great as in parenchymatous nephritis. Casts and blood cells are seen on examination of the deposit. The blood pressure is raised, but it is not usually so high as in cases of primary contracted kidney. The radial artery is hard and cord-like. The impulse of the heart is displaced outwards, the aortic second sound is accentuated, and the first sound may have a dull quality owing to increase in the muscular element.

"Albuminuric retinitis" is very characteristic of secondary contracted kidney. Owing to the vascular changes always present in this condition, the retinal changes often resemble rather closely those which have already been described under arterio-sclerosis (*see* p. 367). On the other hand, the larger, pale, soft-edged areas, or cotton-wool patches (*see* p. 621), are sometimes seen (*see* Plate VIII., p. 368). Bright patches of exudation, showing fatty changes microscopically, are sometimes arranged as a star-shaped figure in the macular region.

Hæmorrhages are frequent owing to the high blood pressure and to the changes in the smaller arteries. In addition to retinal hæmorrhages, epistaxis is common, and purpura and bleeding from the stomach and bowel may occur. The most important is cerebral hæmorrhage, which is a frequent cause of death in chronic renal disease. The patients complain of headaches and sickness, and anæmia is a marked symptom in advanced cases.

**Course.**—The patient may continue for years with varying health. In some cases there is nothing at all to suggest the presence of disease until uræmia suddenly comes on. Death may be due to one of several causes. Probably uræmia is the commonest. This is of the chronic or asthenic type.

*Asthenic Uræmia.*—The patient complains of feeling weak and sleepy, of nausea, which may be accompanied by vomiting, and of headache.

There may be cramps and tingling sensations. Periods of restlessness alternate with stupor. Twitching of the muscles, particularly those of the face, are seen; but there are no convulsive attacks as in sthenic uræmia. As coma develops breathlessness becomes obvious, giving rise to deep hissing respirations; the air hunger may be just as marked as in diabetic coma. The breathlessness increases in proportion as the acid accumulates in the blood. Sudden attacks of breathlessness, sometimes called "renal asthma," have been described, particularly at night. Cheyne-Stokes breathing has also been described. If a raised urea content in the blood is regarded as the criterion for true asthenic uræmia, it is rather doubtful if these two latter types of respirations are really uræmic. They are commonly seen in cases of so-called cardio-renal disease or myocardial degeneration, described by Lewis and others (*see* p. 325), in which the blood urea was always less than 100 milligrammes per cent., far below the true uræmic value. Further observations have shown that there is no increase of fixed acids in the blood in these cases (**6, 7**). It is probable that in the past the term *uræmia* was held to cover a number of different states, and it is only with the advent of methods of biochemical investigation that these states are in process of being differentiated from one another. When the symptoms of uræmia are less pronounced the condition is sometimes called *latent uræmia*, a name which has also been given to the results of suppression of urine from obstruction of the urinary passages (*see* p. 658).

Intracranial hæmorrhage is also a cause of death. It may take place in the neighbourhood of the basal ganglia, in the pons or medulla, or into the meninges.

Another cause of death is dilatation of the hypertrophied heart. This is usually due to myocardial degeneration. Various grave cardiac irregularities may occur,—auricular fibrillation, pulsus alternans, heart block. The mitral valve becomes incompetent, and eventually the complete picture of a primary valvular disease may be developed, with a mitral systolic murmur, engorgement of the lungs and liver, ascites, dropsy of the lower half of the body. The urine is altered in the same manner as it is by a primary valvular disease—that is to say, it becomes scanty, high-coloured, and deposits urates, thus entirely losing the characteristics of the urine of granular kidneys. The pulse may for some time retain its hardness, but eventually in some cases may become indistinguishable from the pulse of mitral disease. Finally, death may take place from cardiac failure.

Death may also be due to secondary infections, *e.g.* pleurisy, pneumonia, pericarditis and gastro-enteritis leading to severe vomiting and diarrhœa. Secondary infection is the usual mode of termination in parenchymatous nephritis. It is by no means such a danger in secondary contracted kidney.

**Diagnosis.**—Much of what is said under Primary Contracted Kidney applies here. The various tests of kidney function are of great value in differential diagnosis.

**Prognosis.**—This is bad in the developed disease. The tests most valuable in estimating the gravity of the case are the blood pressure and state of the radial artery, the examination of the retinæ, the response of the circulation to graduated exercises and the estimation of kidney function. If there is well-marked retinitis, the patient will probably not live longer than two years. If the blood urea is permanently above 100 milligrammes per cent., uræmia may supervene at any time. Patients may live many years if the blood urea is below this figure, especially if the urea concentration test is fairly satisfactory.

**Treatment.**—This is discussed in the next section.

### PRIMARY CONTRACTED KIDNEY

Other names for this type are gouty kidney and red granular kidney.

**Ætiology.**—It is a disease of middle and advanced life, occurring with

extreme rarity under twenty years of age, and most frequently between forty and fifty-five; but it has been seen in infants, and even in the new-born. It affects males more than females. From its very slow development, it might be expected that the causes would be such as were in continuous action. The most important are gout, whether latent or developed, and chronic lead-poisoning. Alcohol, also, has considerable influence; sometimes, perhaps, by inducing the gouty habit. Some cases may be due to heredity, to chronic dyspepsia, or to climatic influences. It has been stated that renal diseases are more common in temperate climates, with their greater atmospheric variations, though these seem more likely to produce acute nephritis. Persistently high blood pressure or *hyperpiesia* may certainly exist independent of renal disease; but it is the forerunner of renal disease, since increased blood pressure undoubtedly leads to arterio-sclerosis, and general arterio-sclerosis to vascular changes in the kidney.

**Morbid Anatomy.**—The kidneys are reduced in size, sometimes to a remarkable extent, and often one more so than the other; the two organs together may only weigh 3 or 4 ounces, and even less than 1½ ounces. The shape of the kidney is not materially altered, except from some irregularity in the rate of contraction in different parts. The capsule is adherent, and, if stripped off, carries with it small portions of renal tissue; the whole surface of the kidney is then seen to be covered with minute elevations (granulations) of $\frac{1}{16}$ to $\frac{1}{8}$ inch in size, with intervening depressions, and here and there may be cysts varying from $\frac{1}{8}$ to $\frac{1}{3}$ inch in diameter, and containing a clear fluid or colloid material. The colour is mostly brownish-red, or dark red or pink, varying somewhat with the vascularity and the amount of interstitial tissue. The whole organ is tough; on section the cortex is found to be very narrow, sometimes reduced to $\frac{1}{4}$ or $\frac{1}{8}$ inch; the pyramids are also smaller than normal, and generally somewhat darker in colour than the cortex. Cysts may be present in the substance of the organ, and the divided vessels often stand out prominently on account of their abnormal thickness.

Sometimes the kidneys do not show any changes macroscopically, but under the microscope the characteristic change is seen to be a great thickening of all the arterioles affecting the intima particularly, and this often shows fatty changes. Where the arteries are so thick that the lumen becomes obliterated, the glomeruli and corresponding tubules become destroyed, so that the picture may resemble somewhat the secondary contracted kidney.

**Pathology.**—This condition is of vascular origin. The arterioles of the brain and kidney are particularly affected, their walls being hypertrophied. These changes have been described under the heading Diffuse Hyperplastic Sclerosis, when the subject of arterio-sclerosis was dealt with. There is a great rise of blood pressure and a markedly hypertrophied heart. The patients often die from rupture of a cerebral vessel leading to hæmorrhage in the brain, pons or medulla. However, cardiac failure and less often uræmia and secondary infection are also causes of death.

The condition is primarily vascular, and its effect on the function of the kidney, *e.g.* the excretory power for urea, though qualitatively the same as in secondary contracted kidney, is not usually so marked. The symptoms resemble one another to some extent.

**Symptoms.**—The onset of primary contracted kidney is generally quite slow, and marked by a few distinctive features. Often, indeed, the kidneys are found to be granular in patients who die of other diseases, or a patient is struck down by cerebral hæmorrhage without any symptom having attracted attention to the condition of these organs. Amongst early symptoms, which, occurring in a middle-aged person, should make one think of granular kidney, if not accounted for in other ways, are recurring or persistent headache, nausea and vomiting, shortness of breath, anæmia, loss of appetite, and general weakness. Often quite early it may be elicited that the patient is passing a large quantity of water,

especially that he has to get up frequently at night to empty his bladder, and that the urine is unusually pale. Occasionally no symptom may be sufficiently prominent until the sight is affected by arterio-sclerotic retinitis, and the patient's eyes are examined with the ophthalmoscope.

The urine is quite normal at first; but in course of time it becomes abundant, pale in colour, almost watery, and of low specific gravity, 1,005 to 1,012. The quantity of albumin is small, often a mere trace, generally not more than 0·5 per cent. It may be for days entirely absent, or it is more abundant at one time in the day than another. The urine is quite clear, or throws down a very scanty deposit, in which a few hyaline or granular casts may be found. There is not at first any dropsy. Even after albuminuria has been recognised some time, there may be no more than a slight œdema of the ankles, or a little puffiness of the eyelids, or a watery conjunctiva. If the dropsy becomes considerable, either it has the characters of the cardiac form, and is the result of the secondary implication of the heart; or it is a renal dropsy, and is due to the supervention of acute nephritis upon the chronic disease. The heart and pulse reveal the changes in the circulation that have been described. The blood pressure is much above the normal and higher than in the secondarily contracted kidney; values of 300 mm. are sometimes found for the systolic pressure. The diastolic pressure is also raised, but not in proportion.

Cerebral hæmorrhage and thrombosis when they occur are not necessarily fatal, but may lead to paralysis of varying degree according to their extent and situation.

The symptoms produced by failure of the circulation have been described in the previous section.

**Diagnosis.**—Granular kidney must not too hastily be assumed from the detection of a small quantity of albumin in the urine of a middle-aged person. The early symptoms must be carefully inquired into, the heart and pulse investigated, and the urine examined on several occasions.

The conditions generally considered to justify a diagnosis of primary contracted kidney are a small quantity of albumin in the urine, associated with symptoms of ill-health, and evidence of cardio-vascular disturbance, such as high blood pressure, thickened arteries, cardiac hypertrophy, retinal hæmorrhages, or retinitis, and changes in the retinal arteries, and œdema of the feet.

In a more advanced stage of the patient's illness, it is often very difficult from the clinical features alone to determine whether a case is one of *primary valvular* or *myocardial heart disease* with albuminuria from congested kidneys, or one of granular kidney with secondary dilatation and hypertrophy of the heart. It has already been said that the urine of chronic renal disease may in time assume all the characters seen in heart disease; nor can the pulse be absolutely relied upon. A localised systolic murmur at the apex may occur in both cases; diastolic or pre-systolic mitral murmurs, and much more aortic murmurs, are in favour of primary heart disease. However, the concentration of urea in the blood and the power of the kidney to excrete a concentrated urine after a dose of urea are most valuable in distinguishing the two conditions.

In their typical conditions there can be no difficulty in distinguishing chronic parenchymatous nephritis from primary contracted kidney. In the former there are general dropsy, scanty urine, much albumin, and casts of all kinds; in the latter we see little or no dropsy, much urine, a small quantity of albumin, few or no casts, and there may be a very long history. The resemblance between primary and secondary contracted kidney may be very close: in the latter the albumin is usually abundant, and the patient is younger.

**Prognosis.**—This is unfavourable, but much improvement may take place, and life may be prolonged in some cases to an advanced age. The greater the extent to which the heart is implicated, the less is the expectation of life; what has been said under secondary contracted kidney also applies here.

**Treatment.**—After the removal of any condition which can be safely regarded as the cause of the condition—*i.e.* alcohol, lead, constant exposure to cold, etc.—the objects should be (1) to diminish the call upon the excretory power of the kidney; (2) to reduce the strain upon the heart and vessels; (3) to remove anæmia; and (4) to treat special complications as they arise.

A quiet life, an easily digested and not too ample diet low in protein, the use of diaphoretics, and occasional purgatives will fulfil the first indication. An exclusively milk diet is unsuitable, because the protein content of such a diet is quite high. Alcohol should be entirely stopped, or amount at most to a glass of claret or dry sherry daily. Complete rest may be enforced when symptoms are serious, particularly if there are breathlessness when the patient is walking about and attacks of angina pectoris. At all times over-exertion and strain should be avoided. Exercise should be taken within the capacity of the heart. The patient should be warmly clothed, and residence in a genial climate is of great benefit.

Such hygienic measures may tend of themselves to lower the blood pressure. It must be remembered that the high blood pressure is probably itself in many cases a compensatory mechanism, so that the attempt to influence it directly with drugs, even if partially successful, may be bad practice. However, drugs may be used for this purpose if there is troublesome headache. Nitro-glycerine ($\frac{1}{100}$ minim once or twice daily and gradually increased), or sodium nitrate, or erythrol tetranitrate may be used.

Various preparations of iron should be given for the anæmia.

Antipyrin, caffein, and phenacetin are also useful. Potassium iodide or thyroid extract may be given for arterial disease.

Among the complications which require treatment are uræmia, vomiting, cardiac dilatation and failure. In uræmia the chief indication is to get rid of the excessive urea and other substances from the blood. For this purpose sweating should be promoted. A hot-air bath is sometimes useful, and pilocarpine injections have been employed. The bowels should be made to act freely, those purgatives being used that produce large watery evacuations, such as jalap. Irrigations of the colon may be used. Bleeding has often been of benefit, and this has occasionally been followed by blood transfusion from a suitable donor. Great care should be taken to see that the flow of urine remains free. Plenty of fluid should be taken by mouth. Intravenous injections of saline may be given if the blood pressure suddenly begins to fall. It is reasonable to add some sodium bicarbonate to the fluid to combat the acidæmia, and this drug may also be given by mouth.

For vomiting, effervescing mixtures, dilute hydrocyanic acid, a few drops of tincture of iodine in water every hour, or a cold compress or blister to the epigastrium may be tried. For the cardiac symptoms, which result from dilatation and feeble contraction of the ventricle, digitalis should be given, the bowels should be kept open, and generally the case should be treated like one of heart disease.

### SENILE ARTERIO-SCLEROTIC KIDNEY

This is the type of renal fibrosis most commonly met with. Nearly all elderly people, whatever the cause of death may be, have kidneys that show these changes. The kidneys are often slightly reduced in size. The capsules are adherent, and when stripped leave a granular surface. The arterial changes are those of atheroma and senile calcification. If the lumen becomes blocked at any point circulation is arrested locally, and death of kidney tissue follows. This is specially apt to occur, since the general blood pressure is not much raised, so that there is no force to drive the blood through a specially narrow channel. Microscopically the characteristic features are small infarcts, consisting of fibrous tissue in which obliterated glomeruli and tubules can be seen, arranged at the surface of

the kidney in the form of wedges, and close to them perfectly healthy glomeruli and tubules where the circulation remains intact. This type of kidney disease is not of much clinical significance, since there is usually enough kidney tissue left for the body's needs. The blood urea is not usually much more than 40 or 50 milligrammes per cent. The patients die of some other complaint, *e.g.* myocardial degeneration (*see* p. 325).

## KNOWN BACTERIAL INFECTIONS OF THE KIDNEY

### BACILLURIA

The presence of bacilli in the urine has been more than once referred to. Thus in typhoid fever the urine may contain Eberth's bacillus (*see* p. 86), in the paratyphoid fevers the corresponding bacilli (*see* p. 92), in pneumonia the pneumococcus; and in tuberculosis of the kidney the tubercle bacillus is found. In other cases, staphylococci, streptococci, the *Bacillus pyocyaneus*, and the *B. proteus* have been seen. Of special interest is the invasion by the *Bacillus coli communis*, which frequently happens from unknown causes and may be associated with streptococci and staphylococci; it is often at any rate a primary lesion—that is, such inflammatory lesions of the tissues as are found to accompany it are its results and not its causes.

**Ætiology.**—It occurs at all ages, but it is especially frequent in infants under two years of age. At all ages females are much more frequently affected than males, and in adults pregnancy is a common antecedent; but a common association in these, as in other cases, is some intestinal disturbance, such as constipation, diarrhœa, or colitis. There are three possible means of access of the colon bacillus to the urine: (1) probably the commonest, by way of the blood vessels; (2) from the colon through the tissue lymphatics; and (3) from the lower part of the urinary tract, and in particular from the urethra. The frequency of the condition in females suggests that the latter may really be a very common way of infection. However, it has been shown that unless there is some gross obstruction in the urinary passages, so that the urine remains stagnant, infection does not spread upwards in the lumen of the passages against the stream, but in the surrounding lymphatics, and in this way it may reach either the pelvis, causing *pyelitis*, or the capsule of the kidney (**9**). Consequently some primary lesion of the mucous membrane of the lower part of the urinary tract is essential in this mode of infection, or else the lymphatics would not be invaded.

**Condition of the Urine.**—The urine is often clear when passed, and has a specific gravity of 1,010 to 1,015. On standing it deposits a layer like mucus at the bottom, above this there is a haze or turbidity, and at the top a clear layer: or the urine is entirely turbid, and the turbidity does not clear on standing. The urine is nearly always *acid*, and it does not become clear either with heat and acetic acid or with alkalies. Moreover, it has a peculiar, offensive odour, which is not ammoniacal, but is probably due to sulphur compounds (methyl mercaptan and sulphuretted hydrogen). A small amount of albumin may be present, and on examination with the microscope there are generally some pus corpuscles, a few red blood cells, and numerous bacilli. These can be shown by centrifuging the urine, making a film of the deposit, and staining with methylene blue. Their nature can of course be further demonstrated by cultivation and bacteriological tests. In advanced cases pus cells and bacilli are more numerous.

**Symptoms.**—Bacilluria may be discovered in persons without any symptom at all, but at some time or other it is likely to cause either local or general disturbances. The local disturbances are due to irritation, and later inflammation of the urinary passages—the bladder, urethra, the pelvis of the kidney, and ultimately the substance of the kidney, constituting a suppurative nephritis. Thus, in small children incontinence of urine and frequency of micturition are

commonly met with ; and this may develop into a definite cystitis, in which fever may be absent, and the patient may not be ill. In a later stage, there is not only cystitis, but pyelitis as well, and the patient has pyrexia, with perhaps rigors. In a more advanced condition still, when the kidney is involved, there are all the usual signs of nephritis, namely, pronounced albuminuria, tube casts, severe pyrexial illness, prostration, uræmia, and a fatal termination.

The general symptoms associated with colon bacilluria, but not necessarily caused by it, are often referable to the gastro-intestinal canal, such as constipation or diarrhœa, or, the one alternating with the other, headache, giddiness, and furred tongue ; sometimes cerebral symptoms of a severe type have been present. It is probable that some hitherto unexplained cases of prolonged pyrexia in children are due to bacilluria.

**Diagnosis.**—This is based on the physical and bacteriological examination of the urine, and can give rise to little difficulty so long as it is remembered that obscure conditions of fever, or even cerebral symptoms, as well as local evidences of cystitis and pyelo-cystitis, may be due to this infection.

**Treatment.**—In early stages the disinfection of the urine may be attempted by the administration of hexamine (10 grains three times a day). Acid sodium phosphate in 30-grain doses may be used to keep the urine acid. Another method of treatment is to render the urine fully alkaline by adequate doses of potassium acetate or citrate. These two methods may be employed alternately.

Any intestinal disorder should be promptly treated. In more advanced cases the treatment by vaccines of *Bacillus coli* has given good results ; if streptococci or staphylococci are present a mixed vaccine may be employed.

## PYELITIS

**Ætiology.**—Inflammation of the pelvis of the kidney, or pyelitis, arises from several causes, of which the following have been recognised : (1) The action of turpentine and cantharides when given internally. (2) Gastro-enteritis, colitis, gall-bladder disease, appendicitis, febrile disorders, such as enteric and typhus fever, the exanthemata and pyæmia, as well as scurvy, diphtheria, and cholera. (3) It occurs, to a slight extent, in Bright's disease and diabetes ; in the latter probably as a result of the irritation by the saccharine urine. (4) Many cases are due to a definite local cause, such as the irritation of foreign bodies in the pelvis and infundibula of the kidney, especially calculi and gravel, but also hydatids, blood clots, and cancer. (5) Obstruction to the passage of urine may also lead to it by decomposition of the retained urine. (6) Inflammation may spread upwards in the lymphatics outside the ureter to the pelvis in cases of cystitis and other forms of inflammation in the urinary passages. (7) In the majority of cases of pyelitis there is no obvious primary focus, although it seems pretty certain that the infection reaches the kidney by the blood stream. Usually one kidney alone is affected. Such cases get well, but they often relapse. The colon bacillus is very often the causal organism, owing to the propinquity of the renal pelvis to the colon. Pregnancy predisposes to such an infection, since the uterus may press on the ureter, causing partial obstruction and dilatation of the pelvis above. Pyelitis is often associated with pyelo-nephritis and cystitis.

**Morbid Anatomy.**—An acute or chronic form may be distinguished. In *acute pyelitis*, the mucous membrane is swollen, its vessels are injected, and the surface is covered with muco-pus ; there are often small spots of hæmorrhage, and sometimes the inflammation takes on a diphtheritic form, patches of membrane adhering to the surface here and there. In calculous pyelitis there may be ulceration of the surface from the presence of the stone ; and such ulceration may, as already indicated, lead to perforation and perinephritis. *Chronic pyelitis*, in which the membrane has a white or ash-grey colour, is often the result of long-continued obstruction ; and accordingly there is at the same time dilata-

tion of the pelvis, infundibula, and calices, with flattening of the pyramids, and more or less atrophy of the renal structure. As a result of the pyelitis, pus and urine may accumulate to such an extent as to form a perceptible tumour (*pyonephrosis*); and the liquid so retained may from time to time, through a change in the obstructing conditions (*e.g.* shifting of a calculus), be discharged into the bladder, so that the renal tumour subsides, and the urine suddenly contains a quantity of pus. Sometimes a pyonephrosis will open like a perinephric abscess in various directions, such as into the loin, the iliac fossa, the bowel, the peritoneum, or the chest. In the renal pelvis there may be, besides pus and urine, blood, calculi, or other foreign bodies which have set up the mischief; and the urinary salts may be deposited, namely, urates in acid urine, and phosphates, if the urine is alkaline or ammoniacal, as it often is. The kidney is the subject of consecutive nephritis, either suppurative or interstitial, with more or less dilatation and atrophy. In some old cases the organ is so atrophied as to consist of little else than its capsule, and septa forming cavities which contain putty-like masses, the result of the inspissation of pus and the deposit of amorphous phosphates.

**Symptoms.**—*Acute Pyelitis.*—There is frequently some dull, aching pain in the loin, increased on pressure. A large tender lump may be palpable in the loin. There is fever, and at times rigors. Micturition may be frequent. The results of the inflammation generally show themselves in the urine. In early stages there are some mucus, a few pus-cells, epithelial cells from the pelvis and infundibula, and perhaps blood. The cells of the pelvic and infundibular epithelium are mostly conical, pyriform, tailed, or fusiform in shape. The urine is acid, and contains a mere trace of albumin. Where the pyelitis is secondary to infection elsewhere in the urinary tract, the symptoms due to the latter will also be present.

In the later stages the urine contains pus in notable quantities (*pyuria*). When such urine is passed it is turbid, and as it settles the pus forms a very pale yellow creamy deposit at the bottom of the glass, and mixes with the urine only just at the line of junction. The nature of this deposit can be determined by chemical tests, and by the microscope.

The usual *chemical* test is this: The supernatant liquid is poured off, and some liquor potassæ or liquor ammoniæ is added to the deposit; it quickly loses its colour, becomes translucent, and changes into a viscid, ropy liquid, which falls from vessel to vessel in a more or less coherent mass. If the urine should decompose and become alkaline within the body, the pus will undergo the same ropy change, and the urine will be mixed with this viscid, glairy fluid, instead of with creamy pus. This happens sometimes in pyelitis and pyonephrosis, if the urine retained in the dilated pelvis at length undergoes decomposition; and it happens frequently from the same cause in cystitis. Under the *microscope* the creamy deposit shows numerous pus corpuscles.

The urine contains a very small quantity of albumin, derived from the pus. It is often desirable to know whether the albumin present in such urine is solely due to pus, or is in part derived from diseased renal tissue. The pus should be separated as completely as possible by subsidence, or even by the use of the centrifuge; a large quantity of albumin must be due to something besides the pus. The presence of casts would, of course, suggest that the structure of the kidney was involved.

It has been already stated that the purulent urine may be retained by an obstruction of the ureter: during such retention, the urine in the bladder will be quite normal; and it will again become purulent when the obstruction is partially or wholly removed. In such cases a tumour forms in the flank, consisting of the pelvis and kidney distended with urine and pus, so long as obstruction exists. It is more or less rounded or oval, in some cases lobulated; lies between the costal margin and the crest of the ilium; is dull behind, and generally has the colon in front or to its inner side. There may be a line of resonance

between its dulness and that of the liver and spleen, but the areas of dulness are often continuous. The tumour is usually painful and tender; and if the obstruction of the ureter is sudden, the pain may be severe and paroxysmal. If the obstruction yields, the swelling will disappear for a time.

In *chronic pyelitis* the local pain and tenderness may be almost absent, so that there is difficulty in diagnosis. The symptoms are usually those of a chronic cystitis.

**Diagnosis.**—Pyelitis in its early stage must be distinguished by the local signs, and by the presence of pus cells in the urine. When pus is contained in acid urine, it is more likely to come from the pelvis of the kidney than from an inflamed bladder. The diagnosis as to the presence of cystitis, and whether one or other kidney is involved, may be furthered by the use of the cystoscope, with catheterisation of the ureters if necessary.

Pyonephrosis may be confounded with the numerous swellings which occur in the right flank, and which have been alluded to under Perinephric Abscess. Abscess and hydronephrosis are those which are most likely to give difficulty. In the former there is more pain than in pyonephrosis, more severe fever, fluctuation is more superficial, and the preceding hardness is less defined. The skin may be œdematous, and the urine is free from pus, unless the abscess has ruptured into the pelvis or ureter.

The **Prognosis** depends very much upon the primary cause. Pyelitis due to blood infection almost always gets well, although it may linger on for a long time. Pyonephrosis is a serious lesion; it may be fatal by perforation into the chest or abdomen, by exhaustion from continued discharge, or by the induction of lardaceous disease. Rarely the pus inspissates, and a cure results with the loss of one kidney.

**Treatment.**—The primary condition must be studied, and treated as far as possible (*see* Tubercle of the Kidney and Renal Calculus). Where the pyelitis is more or less distinct from its cause, and open to separate treatment, this consists, in acute cases, of rest; dry-cupping of the loins if there is hæmaturia or much pain; the free drinking of warm bland liquids, by which the urine may be diluted; and the administration of salines (potassium citrate or acetate), with small doses of opium if there is much pain. In other cases hexamine is given in doses of from 6 to 10 grains. It is important to make the urine acid. This may be done by prescribing boric acid with the hexamine, or acid sodium phosphate, 30 grains, may be given in doses alternating with the hexamine. In very chronic cases the balsams and allied drugs, oil of turpentine, oil of sandal-wood, copaiba, and benzoic acid, or salol may be given. In different cases vaccines of streptococcus or bacillus coli have been useful. A general tonic regimen may at the same time be desirable, a nutritious diet, fresh air in the country or at the seaside, quinine and cod-liver oil.

When the distended pelvis forms a tumour (pyonephrosis), as a rule the operation of *nephrotomy* should be performed, and the cyst opened, especially if there be constant pain, severe fever, and interference with the action of the stomach and intestine; or if the tumour is increasing in size, inflaming the surrounding tissues, or threatening to rupture. Under opposite conditions, and if the cyst empties itself into the bladder from time to time, palliative measures may be tried, such as rest in bed or on a couch, frequent hot baths, anodyne and emollient applications, gentle compression by belladonna plasters, and the avoidance of constipation and fæcal accumulation.

### SUPPURATIVE PYELO-NEPHRITIS

When suppuration of the substance of the kidney is secondary to lesions of the lower urinary passages, it is frequently associated with pyelitis, and hence is termed *pyelo-nephritis.* All those causes which produce obstruction to the

passage of urine, distension of the urinary passages, and cystitis, may ultimately result in suppurative nephritis; for instance, stricture of the urethra, prostatic enlargement and calculus; various diseases of the pelvic organs, uterus, ovaries, and appendages in the female, which involve the bladder or ureters; and paralysis of the bladder from spinal injury, spinal disease, or myelitis. Pyelo-nephritis may also result from pyelitis when the latter is due purely to a blood infection.

**Anatomy.**—The kidney is usually enlarged and softened. The surface is mottled pale yellow and red, and presents several small points of suppuration, some of which may be torn open when the capsule is stripped off. On section, the cortex shows the same mottled colour, the pyramids are usually red and congested, and the kidney has the appearance of one in a state of acute interstitial nephritis. The characteristic feature is the presence of numerous yellow streaks of pus, stretching in a radial direction from the surface inwards through the cortex, and even into the pyramids. They are sometimes wedge-shaped, or conical, with the base at the surface, in other cases simply linear. Under the microscope the kidney is seen to be acutely inflamed; there are numerous leucocytes in the intertubal tissue, glomerular inflammation and swelling and nuclear proliferation of the epithelium of the tubes.

**Symptoms.**—It will be understood that the patient who suffers from this form of suppurative nephritis is often the subject of stricture of the urethra, enlarged prostate, cancer of the uterus, or of cystitis from retention of urine in spinal disease. The first symptoms are often chills or rigors, with a rise of temperature to 104°, 105°, or 106°, and there may be a regular intermittent or remittent fever. Then follow the characteristics of a typhoid or septicæmic condition—loss of appetite, dry brown fissured tongue, nausea and vomiting, sometimes diarrhœa, sweating, and rapid emaciation. The patient is somnolent, in a more or less dreamy state, and eventually symptoms typical of asthenic uræmia may show themselves—coma, twitching of muscles and air hunger. The pupils vary, and have no special significance. The pulse is quick and feeble. Lumbar pain is sometimes present, and there is generally some tenderness on deep pressure in the loins. The state of the urine is determined by the preceding disease; it may thus contain pus, mucus, blood, or albumin; and it may be difficult to recognise any additional albumin, or even the hyaline casts, or the epithelial cells which result from an acute nephritis. In some cases, on the other hand, renal and pelvic epithelium and hyaline casts are found in sufficient quantity. When both kidneys are extensively affected, their functions are interfered with. There may be increased urea in the blood, acidæmia and a typical renal œdema. Morris notes that in cases with intermitting fever the quantity of urine is often greater during the febrile periods than between them.

The duration of the illness is not more than four weeks, and may be as short as three or four days.

**Diagnosis.**—The occurrence of pyelo-nephritis in cases of disease of the bladder and ureters is generally signalised by the fever and the typhoid condition. It is likely to be confounded with pyæmia, other forms of septicæmia, enteric fever, peritonitis, and ague. In *pyæmia* one looks for secondary abscesses, which are absent in pyelo-nephritis. The temperature of pyæmia shows a more extensive range, and the rigors are followed by profuse sweating. Milder forms of *septicæmia* resemble pyelo-nephritis; in the acute forms there are more restlessness and anxiety. Purpura and internal ecchymoses do not occur in pyelo-nephritis. In *enteric fever* the typhoid state is more slowly developed. Rose spots, typical motions, and the regular curve of temperature are in its favour; while rigors and an irregular fever would speak strongly for suppurating kidney. *Peritonitis*, which may arise from lesions of the bladder, is recognised by the absence of rigor, the greater pain, and more severe vomiting. The *malarial* patient in the intervals between the rigors is free from fever and almost well. In all these cases tests of kidney function may be of the greatest value in making a

diagnosis, but it must be remembered that suppuration may occur in the kidney with no alteration in its functions, if there is enough of the healthy tissue remaining.

**Treatment.**—That suppurative nephritis may supervene should always be borne in mind in the treatment of the urinary organs. In spinal cases it is necessary to try and prevent the onset of cystitis. Owing to war experience, the use of the catheter is now much restricted in these cases, since infection is introduced by such means. In cases where the spinal cord is completely divided, use may be made of the mass reflex which gradually develops under these circumstances. At periodical intervals the skin is stimulated. This leads to flexor responses in the limbs and contraction of the bladder, so that the latter is completely emptied. In other cases when cystitis is present the bladder should be irrigated with some antiseptic solution, such as salicylate of sodium (5 grains to 1 ounce), quinine (1 or 2 grains to 1 ounce), or borax (5 grains to 1 ounce); and urotropin (10 grains) or helmitol should be given internally. When suppurative nephritis has declared itself, treatment is much less likely to be of use; but the employment of vaccines of the organism concerned should be considered. The patient should be supported by nutritious but easily digestible food. The action of the skin should be promoted by vapour or hot-air baths. If there is much lumbar pain, hot sand may be placed on the loins, or dry-cupping may be employed. The bowels should be kept active. Internally, quinine, with small doses of opium or liq. morphinæ, seems the best remedy to give.

In cases where suppurative pyelo-nephritis is associated with acute retention of urine, *e.g.* in enlarged prostate, the outlook may be serious, if there is much retention of urea, since uræmia may supervene at any moment. It is important to relieve the obstruction to the flow of urine immediately.

## METASTATIC NEPHRITIS

Metastatic abscesses occur in *pyæmia* and occasionally in *malignant endocarditis* as the result of micro-organisms being conveyed by the blood vessels to the kidneys.

Pyæmic abscesses are generally scattered through the cortex of the kidney; are of small size, more or less elongated, and sometimes conical; and are surrounded with a red zone of vascularity. Their occurrence does not obviously add to the symptoms of pyæmia. Albuminuria may occur independently of them, and cannot therefore be held to indicate their presence.

In malignant endocarditis, the abscesses may arise from the breaking down of embolic infarcts of various sizes. These are conical, but generally have a different shape from the abscesses of pyelo-nephritis, the base being relatively broader. Embolic infarction may be indicated by pain and by blood and albumin in the urine; but albumin occurring alone in the course of endocarditis does not of itself indicate suppuration.

Sometimes, both in pyæmia and endocarditis, there may be one or two large abscesses, instead of several small ones.

## PERINEPHRITIS AND PERINEPHRIC ABSCESS

Perinephritis is the term used for inflammation of the cellular and adipose tissues around the kidney.

**Ætiology.**—It arises: (1) From injury—such as blows, kicks, or strains. (2) From the extension of inflammation from the kidney, the pelvis of the kidney or the ureter. This may be the result of suppurative pyelitis, pyonephrosis, tubercle of the kidney, or calculus, which either sets up pyelitis, or itself ulcerates through the kidney or pelvis. (3) From inflammation, especially suppuration, in more distant parts spreading to the perinephric tissues; for

instance, pelvic cellulitis, appendicitis, abscess of the liver or spleen, caries of the spine and psoas abscess, or inflammation of the gall-bladder.

**Pathology.**—The perinephric tissue in different cases undergoes all the changes that may take place in other inflamed tissues. In early stages it is vascular, œdematous, and infiltrated; then points of suppuration occur, and finally one large abscess is formed. The pus is free from odour, or very offensive; or it has a fæcal odour, from contiguity with the bowel. Sometimes shreds of gangrenous tissue are present. The kidney may be infiltrated or softened in the middle of the abscess. Occasionally the perinephric tissue is indurated by a more chronic process.

**Symptoms.**—These are to a certain extent those which accompany other inflammatory processes. The onset may be insidious, when there is nothing but some dull aching *pain;* in other cases it will be marked by rigor, with elevation of temperature, which continues uniformly high, or is intermittent in character. The pain is deep-seated, in the loin or side of the abdomen, and radiates to the hypogastrium, groin, or genitals; that is, over the distribution of the lumbar plexus. The pain in the loin is increased by pressure, and on bimanual examination a certain amount of fulness or resistance may be felt in that region. The patient may be unable to flex or extend the lumbar spine.

As the case progresses, a more or less extensive *tumour* occupies the space between the last rib and the crest of the ilium, uniformly dull, bulging the flank, causing œdema of the loin, and perhaps fluctuating. The leg of the same side is often flexed at the hip joint, and attempted extension causes pain. Attention has also been called to the peculiar way in which a patient stands who has perinephritis, and this even when the inflammation has not reached the stage of abscess: the body is bent over to the affected side, the hip is a little flexed, and the hand rests on the same thigh. A certain resemblance to hip-joint disease is thus often assumed.

The urine is not necessarily affected; if the inflammation has resulted from ulceration of the kidney, pelvis, or ureter, pus from the perinephric abscess may pass into the urine (*pyuria*). In other cases albuminuria may occur from pressure of the abscess on the renal vein.

When pus forms, it is generally situated at first between the kidney and the lumbar muscles, and may make its way in various directions. If externally, it usually presents between the edges of the latissimus dorsi and the external oblique muscles; or it may pass downwards and point under Poupart's ligament. In other cases it opens into the colon, ileum or stomach; into the ureter, bladder, or vagina; or into the peritoneum, causing peritonitis. Or it perforates the diaphragm and sets up pneumonia, pleurisy, and empyema; or, without perforation, it causes pleuritic effusions, or compresses the base of the lung by raising the diaphragm.

**Diagnosis.**—The conditions that are most likely to be mistaken for perinephritis or perinephric abscess, which is really a rare condition, are lumbago, spinal caries, cancer and tumours of the kidney, hydronephrosis and pyonephrosis, appendicitis, fæcal accumulations, splenic and hepatic tumours. The careful localisation of the lesion will distinguish it from spinal caries, hepatic and splenic tumours, and appendicitis. Fæcal accumulations, cancer of the kidney, and hydronephrosis are not accompanied by fever; nor is lumbago, and this is often a bilateral trouble. The lateral inclination of the body in standing is a useful guide, but hardly serves to distinguish it from hip-joint disease and psoas abscess.

**Treatment.**—This is mainly surgical. Local applications and opiates will relieve pain. If pus has formed it should be let out as soon as possible.

### TUBERCULOSIS OF THE KIDNEY

The kidneys may be infected by tubercle as a part of a general miliary tuberculosis. The liver, spleen, and lungs are often at the same time affected.

The tubercle appears in the form of minute grey or yellow deposits, 1 or 2 mm. in diameter, scattered irregularly, and, as a rule, rather scantily in the cortex and medulla of the kidney. A few may be seen on the surface, and others are revealed by section; they are round in shape or slightly elongated in the direction of the tubules. They present the characteristic minute anatomy of tubercle. The rest of the kidney is healthy, and, as a rule, there are no clinical symptoms attending their deposition. Albuminuria may occur, and Roberts mentions a case from a French source, in which an unusually abundant deposit of tuberculous granulations caused violent lumbar pains, with strong contraction and exquisite tenderness of the lumbar muscles.

The term "tuberculous kidney" is generally used for the condition that was in former days called strumous or scrofulous pyelitis or pyelo-nephritis. In the majority of cases tuberculous kidney is associated with other tuberculous lesions elsewhere in the body. It arises as an infection from the blood stream, though it is probable that it may occasionally result from a spread upwards along the lymphatics outside the ureter. It affects men more often than women, and occurs at all ages, but is least common in quite young children.

**Morbid Anatomy.**—The first change is the deposit of tubercles in the substance of the kidney, either cortex or pyramids. When first seen these are generally already yellow and cheesy; they enlarge, run together, break down into abscess cavities, and ultimately open into the calices and infundibula. But it is an important feature in the history of primary tubercle that the kidney is not alone affected; the disease commonly affects the urinary passages at the same time, and, it may be, every one of the urino-genital organs. Tubercles are deposited in the mucous and submucous tissues of the pelvis of the kidney, which ultimately break down, leaving ragged ulcers opening into the pelvis, and discharging pus, blood, tubercular *débris*, and portions of connective tissue into the current of the urine.

In extreme cases the kidney is almost entirely destroyed, so that scarcely any renal tissue remains. This is caused partly by the spread of the caseating process, and partly by the distension of the pelvis, in consequence of the ureter being obstructed. Thus, what has not been destroyed by tubercular ulceration is converted into dense fibrous tissue, and forms septa, separating the several abscess cavities from one another; and such cavities may ultimately come to be lined with a smooth membrane. Their contents are a semi-fluid cheesy material, or a putty-like mass, which contains an abundance of calcium salts. The capsule is thickened, and may be even like fibro-cartilage.

The *ureter* is similarly diseased; its wall is thickened and rigid from tuberculous deposit, which finally ulcerates. The thickening may be sufficient to obstruct the canal of the ureter, or the latter may get blocked by fragments of tuberculous matter, or by coagula of blood or pus coming down from the pelvis. If such an obstruction is at all complete, the urine and diseased products are retained in the pelvis of the kidney, distension takes place, and a pyonephrosis is the result.

The *bladder* is affected very like the ureter, either at the same time as or even before the kidney. Tubercle is deposited in the submucous tissue, and eventually the mucous membrane is covered with cheesy deposit, and much ulcerated. Sometimes the disease spreads into the *urethra*, and in men the *prostate*, *vesiculæ seminales*, and *testes* may become involved. The genital organs of women are much more rarely diseased in corresponding circumstances.

The disease is at first unilateral, but the kidney of the opposite side may be infected from the bladder. This extension, however, may be long delayed, or, indeed, may never occur. Another possible result is a general tuberculosis, with its inevitably fatal ending.

**Symptoms.**—In most cases the symptoms are those of pyelitis and cystitis combined, or pyelo-cystitis. There is more or less dull, dragging pain in the loin, which may be paroxysmal in character; and there is often some tenderness on

pressure. Severe colicky pains occur if the ureter becomes plugged by tuberculous *débris*. The kidney is not infrequently so much enlarged by tuberculous deposit, or by distension of the pelvis and calices, that it can be felt as a tumour in the flank.

The condition of the urine is most important. It is generally acid, with a more or less abundant deposit of pus, in which may be found pelvic and vesical epithelium, shreds of connective tissue, and *débris* of tubercle; while tubercle bacilli can be demonstrated if the urine be centrifuged and the deposit suitably stained, or their presence can be proved by cultivation or animal inoculation. Blood is often present from time to time, but not generally in large quantity. Albumin occurs in proportion to the amount of pus. Tube casts are rare. The amount of urea will depend on the opposite kidney, which may enlarge enough to eliminate the normal amount. Sometimes the urine is ammoniacal and ropy, from retention and decomposition in the pelvis; sometimes from the co-existing cystitis. With cystitis, also, micturition becomes frequent and often painful.

Fever is generally present at some period of the disorder, and eventually becomes persistent, with morning remissions and high evening temperatures. As the disease progresses, emaciation, loss of appetite, and prostration become marked. In many cases other organs—such as the lungs and intestines—are involved. The opposite kidney also may be affected with the same lesion, or with lardaceous disease. Death takes place from the exhaustion of prolonged suppuration or the tuberculous fever, from pulmonary or intestinal lesions, or from uræmia when the second kidney is seriously involved.

**Diagnosis.**—A tuberculous kidney is most likely to be confounded with renal *calculus*, since they may both cause severe renal colic. Hæmorrhage is more abundant in the latter, pus more continuously present in the former. In all cases of turbid or purulent urine, in which tubercle is possible, the urine should be examined microscopically and bacteriologically for evidence of the bacillus. Often the family history, the previous history of the patient, the co-existence of pulmonary phthisis, or other tuberculous lesion, such as tubercle in the epididymis, suggests or confirms the diagnosis. Sometimes the ureter is so thickened by tubercle as to be felt through the abdominal organ as a hard rigid cord. Palpation of the kidney cannot be relied upon, but the cystoscope may give valuable information both as to the presence of tubercle and the condition of the other kidney. By this means the mucous membrane of the bladder around the orifice of the ureter on the affected side is seen to be reddened, swollen, and abraded or ulcerated, and miliary tubercles may be seen scattered in the neighbourhood. The ureters may be catheterised and the urine from each collected separately and examined.

**Prognosis.**—The prognosis of tubercle of the urinary organs is like that of the lung, dependent on the virulence of the infection, the resistance of the patient, and the promptness and thoroughness of treatment. As recent methods of research render a diagnosis possible at a much earlier stage than formerly, the outlook of the patient is proportionately more favourable.

**Treatment.**—The strength of the patient must be supported by good food, and by tonics, such as iron, quinine, cod-liver oil, and extract of malt; and opiates and local applications should be used to relieve the pain. In early stages the use of tuberculin has led to relief of symptoms and delay in the progress of the disease. But pronounced local conditions will call for an operation either to let out pus, or to excise the organ entirely. This is generally the best course to take, since the tuberculosis is often limited to one side, and even if the ureter and bladder are involved the ureter may be also removed, and the bladder may recover. Moreover, if the cystoscope shows characteristic changes around the vesical orifice of the ureter, the kidney is probably too much damaged to escape destruction (Symonds). Removal is out of the question if the kidney of the opposite side is extensively diseased; or if tubercle has taken firm hold of other

viscera; or if the patient is much exhausted. A small abscess in the kidney may be incised and drained, with some hope of ultimate success; and for large collections of pus in connection with obstructed ureters, nephrotomy is also valuable by relieving tension and pain, and giving direct exit to the purulent secretions.

## HYDRONEPHROSIS

By this term is meant the distension of the pelvis of the kidney by retained secretion; and the retention is, as a rule, the result of an obstruction in one or other part of the urinary passage, whether the ureter, the bladder, or the urethra. If the ureter of an animal is tied experimentally, the pelvis dilates to some extent to accommodate more urine. This dilatation is relatively small, and does not constitute a hydronephrosis. The latter is produced under two conditions: (1) when the obstruction is complete, but intermittent; (2) when the obstruction is partial, so that a higher pressure than normal is permanently required to force the urine past the obstruction. Under these conditions a gradual, large dilatation of the pelvis, or hydronephrosis, is produced.

**Causes.**—Hydronephrosis occurs at all ages, and is more frequent in females than males. It may be congenital—that is, in actual existence at the time of birth, when it may be so large as to constitute a serious obstacle to delivery; or it may develop after birth, although it is due to congenital causes; or it may be entirely the result of disease occurring in later life. Among the *congenital* causes are various abnormalities of the ureter, such as twists upon its axis, folds, reduplications, and valvular arrangements of the mucous membrane, contractions, or conversion into a fibrous cord. Sometimes the ureter joins the kidney at an acute angle, or the opening into the bladder is thick and rigid, or a branch of the renal artery lies across its course. Another cause is an imperforate urethra. Congenital hydronephrosis is often associated with other congenital defects and malformations, such as club-foot, hare-lip, or malformations of the external genitals; and those affected with it are frequently still-born, or live only a short time.

The causes *in later life* are all those kinds of obstruction which have already been referred to—namely, in the ureter, impacted calculus, cicatricial stricture, cancer of the abdominal and pelvic organs, ovarian tumours, and peritoneal bands; in the bladder, villous growths and cystitis with hypertrophy of the walls; and in the urethra, stricture and enlarged prostate.

**Anatomy.**—Any long-continued obstruction leads to distension of the parts behind it. If it is in the upper part of the ureter the pelvis dilates, and the kidney undergoes the following changes of chronic interstitial (consecutive) nephritis: the colour is pale pink or pale yellow; on section it is found that the pyramids are flattened, while the cortex is relatively broad. Under the microscope there is found infiltration of the organ with leucocytes, chiefly around the Malpighian capsules, and in the intertubal tissue; there is also some glomerular change, with a slight alteration of the tubal epithelium. In certain cases the process may go on to contraction of the new tissue, and the production of cicatrices. If the obstruction is lower down—for instance, at the vesical orifice of the ureter—the ureter itself is involved in the distension. Such moderate degrees of hydronephrosis with consecutive induration of the kidney are common as the result of cancer of the uterus, vagina, and bladder in females. They, however, rarely lead to tumours that can be detected clinically, in many cases, no doubt, because death is brought about by the obstructive lesion too early for the extreme development of the hydronephrosis. Some dilatation of the pelvis is commonly produced in pregnancy by pressure of the uterus on the ureter. But where the cause is congenital, or is less directly fatal in itself, such as calculus, or stricture of the ureter, time is allowed for the full development of the pressure effects of the retained urine upon the renal structures. The pelvis becomes

distended, and the kidney more and more flattened out; and finally a large cyst is formed capable of containing 40 or 50 ounces, or even several pints of liquid, and consisting of a thin membranous sac, which may present here and there portions of the kidney substance, but in some cases is quite destitute of any trace of it. Inside the sac there are sometimes septa dividing it into separate cavities. If the ureter is involved, it may be enormously dilated, to the size, perhaps, of the small intestine or colon, or it may be entirely merged in the distended pelvis and kidney.

The liquid of a hydronephrosis varies with the amount of kidney substance still remaining; generally it is equivalent to a very dilute urine; it is pale yellow in colour, contains a small proportion of urea, uric acid and salts, occasionally a trace of albumin, or a little pus. Urea and uric acid are absent in some cases, and the fluid consists mainly of water with a trace of sodium chloride. Pus in any quantity is not present unless there has been previous pyelitis, and the condition is then practically a pyonephrosis.

Hydronephrosis may affect one or both kidneys, according to the situation of the lesion or lesions causing it. In congenital cases it is frequently double—for instance, imperforate urethra must involve both kidneys equally; and the milder forms caused by cancer of the pelvic organs in women are often double. On the other hand, a calculus can only produce a single hydronephrosis, though it is possible that such a cause may be in operation on both sides at the same time.

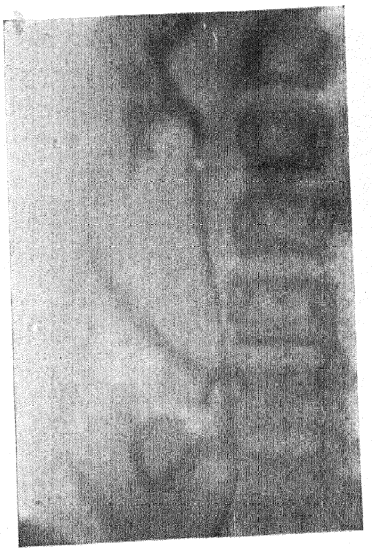
**Symptoms.**—A moderate degree of distension on one side, when the other kidney is healthy, may cause no symptoms whatever. If it is considerable, then a swelling is formed which becomes the prominent feature of the case, and has the usual characteristics of a renal tumour. It occupies one or other flank, extending from the costal margin to the crest of the ilium, and reaching, according to its size, towards the middle line, or even beyond it. A cyst holding 40 or 50 ounces may cause scarcely any prominence of the abdomen, but its presence will be detected by resistance to deep pressure, and by the difficulty of bringing together the two hands, placed one in front, the other under the last rib. With larger cysts, an unsymmetrical enlargement of the abdomen is produced, and the loin and flank are bulged; or in extreme cases there is uniform distension similar to that of ascites or ovarian tumour. Sometimes the cyst is limited to the upper part of the abdomen, and may resemble enlargement of the liver by hydatid. The tumour is smooth or lobulated, in some cases tense, in others quite flaccid; and fluctuation can be sometimes obtained. It is dull on percussion, the dulness reaching back to the loin, and forward perhaps nearly to the umbilicus; the colon lies in front of it, and may cause a resonant note.

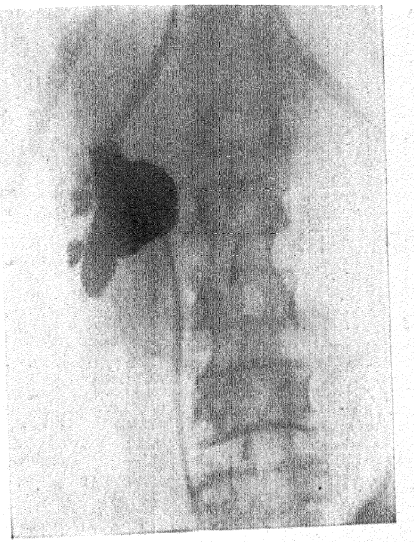
An important feature of the tumour of hydronephrosis is its liability to diminish in size suddenly, or even to disappear, from the escape of its contents into the bladder. Immediately afterwards the patient has an abundant discharge of urine; and the cyst again slowly fills. Slighter variations in size or tenseness may occur without any corresponding change in the urine being noticed. Local symptoms may be caused by the tenseness of the cyst, or its pressure on surrounding parts, such as pain, vomiting, dyspnœa, or interference with the heart's action. But these may be entirely absent; and pain is severe only when the obstruction is sudden and complete. The urine in hydronephrosis is not much altered. Its quantity may be natural, since the healthy kidney compensates for the deficiency of its fellow. It may contain a trace of albumin, or a little pus; the urea and salts are in average quantity.

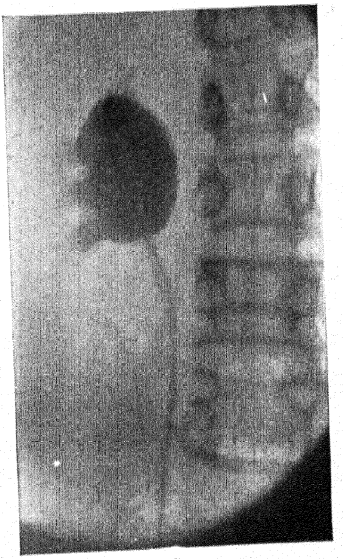
In cases of double hydronephrosis uræmia may occur from the retention of urinary constituents; the earlier indications of obstruction, when there is no tumour, are pains in the back or abdomen, partial suppression of urine from time to time, and increased frequency of micturition.

**Diagnosis.**—The tumour of hydronephrosis has to be distinguished from

# PLATE XXXII.







C.

D.

Radiograms of the Renal Pelvis. A, Normal Pelvis and Calyces. The lower border of pelvis with the upper end of the ureter and lower calyx forms a concavity facing downwards. B, C and D show increasing degrees of hydronephrosis. In B and C the calyces are hardly affected, while in D there is dilatation of both pelvis and calyces. (From cases described by Mr. H. P. Winsbury White.)

[*To face p.* 644

perinephric abscess, from pyonephrosis, hydatid of the liver or spleen, and, in extreme cases, from ascites and ovarian tumour. The history (*e.g.* of calculus) may be the same in hydronephrosis, *pyonephrosis*, and *perinephric abscess;* the first is generally of longer duration, without severe, or even any, constitutional symptoms, whereas the other two conditions are likely to show evidence of suppuration. Perinephric abscess also gives the local signs of acute inflammation. In the absence of general disturbances, pyonephrosis may be with difficulty diagnosed from hydronephrosis, and it may actually develop from it. *Hydatid* of the liver or spleen presses forwards or upwards, bulging the lower ribs, while hydronephrosis occupies the loin first; but a hydronephrosis due to calculus may occupy the right upper quarter of the abdomen just like hydatid of the liver. *Ovarian* tumour should be recognised by the history of the enlargement, the position of the uterus, and the absence of the colon from the front of the cyst In the rare case of a resemblance to *ascites*, the fluid withdrawn by paracentesis would contain urea and uric acid, and no albumin, or at most a mere trace, whereas in ascitic fluid albumin is abundant. In other cases also the aspirator and trocar may be used for diagnostic purposes. The spontaneous disappearance of the tumour, coincident with an increased flow of urine, is in all cases the strongest evidence of hydronephrosis.

**Prognosis.**—A simple hydronephrosis may cause little or no trouble for many years; the kidney may gradually undergo atrophy without the distension being such as to cause any serious trouble. The risk in such a case is that the other kidney may be at some time involved (*e.g.* by calculous obstruction), and then death may be brought about by uræmia. If the cyst reaches a great size, or becomes very tense, it may rupture into the peritoneum, or it may press on adjacent parts—the stomach and diaphragm—and cause death by interference with nutrition, respiration, or circulation; occasionally a cure results from the spontaneous discharge of the contents of the cyst, which never again collect. In one such case the fifth or sixth subsidence in the course of two years was accompanied by the discharge of a calculus, which became impacted in the urethra, and was removed by incision.

In double hydronephrosis, death results from the primary cause (*e.g.* cancer), or from uræmia.

**Treatment.**—Since recovery from hydronephrotic tumour sometimes takes place by the pressure of the liquid overcoming the obstruction, an attempt may be made to get the same result by friction and manipulation of the cyst. But sometimes it is too painful, or too tense, to make such a course desirable. It may then be aspirated. This should be done, on the left side, just at the anterior end of the eleventh intercostal space; on the right side, half-way between the last rib and the crest of the ilium, and 2 inches behind the anterior superior spine of the ilium. The fluid will probably accumulate again, and then the next step should be incision and drainage of the cyst. At this operation a way may possibly be found into the bladder, and the aperture dilated, or an obstructing stone may be sought for and removed. Sometimes this results in complete cure, the secretion ceasing and the wound closing; in other cases a fistula remains, which is often quite manageable. If the discharge is a serious trouble, or if it becomes purulent and threatens to exhaust the patient, the kidney must be excised.

## LARDACEOUS DISEASE OF THE KIDNEY

The nature and ætiology of lardaceous disease have been already described (*see* p. 489); phthisis, syphilis, continued suppuration, occasionally cancer and other cachectic conditions, produce the disease in the kidney as they do in other organs. But a slight amount of lardaceous change is found sometimes in cases of chronic nephritis without any of the above causes being present; and it seems as if nephritis itself may be a local cause of the degeneration.

**Morbid Anatomy.**—In the kidney the lardaceous change affects the vessels first. The glomerular tuft is often first altered, then successively the vasa afferentia, the vasa recta, the vasa efferentia, and the intertubal vessels. In some cases, however, the change can be found in the vasa recta before it is seen in the glomeruli. After the vessels, the basement membrane of the tubules is converted into the lardaceous material, but it is doubtful if the degeneration involves the epithelial cells of the tubules.

In the early stages of lardaceous change the kidneys present at first no appreciable difference, unless, perhaps, an undue distinctness of the glomeruli; but the application of iodine will bring out the vessels and Malpighian tufts by the dark brown or black colour (to the naked eye) which it gives them. If the change is very slight, it may be only seen with the help of the microscope in thin sections stained as formerly described. In advanced stages the kidneys are much enlarged—it may be to twice their normal bulk. They have a whitish-yellow colour, with some venules prominent on the surface; the capsule is slightly adherent. On section, the cortex is much widened, and has a pale yellow colour, more or less mottled with patches of white, while the pyramids are red or reddish-brown, forming thus a remarkable contrast to the cortex. Tincture of iodine stains the Malpighian tufts and their afferent arteries, and the closely packed vasa recta in the pyramids, so that the vessels look as if injected. On microscopic examination, it is seen that there is, in addition, much evidence of inflammatory change, to which the enlargement of the kidney is to be in great part referred. The tubal epithelium is swollen, granular, and fatty, the Malpighian capsules are thickened, and there are leucocytes extravasated into the intertubal tissue.

Sometimes the kidneys are almost normal in size, or even a little less and somewhat rough or granular upon the surface.

**Symptoms.**—The symptoms of lardaceous degeneration commonly appear in patients already obviously suffering from phthisis, from tertiary syphilis with periosteal nodes, gummata, or necrosis, from caries, suppurating sinuses, discharging empyema, or similar lesions. Moreover, evidence of its involving the liver and spleen is sometimes, but not always, present when the kidneys are first called in question.

The first symptom is certainly sometimes the occurrence of albumin in urine otherwise normal—of average quantity, and good, even high, colour. With this there may be no other indication of renal disorder, only the evidence of the cause, and perhaps the changes in the liver and spleen. In other cases there is pronounced polyuria, and this may be the first change, even before the appearance of albumin (Dickinson). The urine amounts to 70, 80, or 90 ounces daily, it is pale in colour, of low specific gravity, 1,008 to 1,014, deposits little or nothing, and contains a varying, often small, amount of albumin. Paraglobulin is sometimes present in abundance, though this is not peculiar to lardaceous disease. In the deposit may be at most a few hyaline or granular casts, and, rarely, some which give the lardaceous reaction.

A third condition of the urine is seen in the last stage, when the lardaceous change is obviously complicated with nephritis; and the urine is scanty, highly albuminous, with numerous granular and fatty casts.

As to the cause of the alteration in the urine, it may be here enough to say that the degeneration of the walls of the vessel seems at once to account for the passage of water and albumin, as long as there is no great extent of tubal nephritis.

Next to the urinary changes, dropsy is the most frequent symptom. It has all the appearance of a renal dropsy, and there may be ascites and hydrothorax. Of ifs complications, pericarditis and peritonitis occur not infrequently, and may be tatal; but the other results of Bright's disease—namely, cardiac hypertrophy, high arterial tension, retinitis, hæmorrhages, and uræmia—are quite rare, though it appears that they do sometimes occur. Diarrhœa may occur from co-existing lardaceous disease of the intestine. Death takes place from increasing dropsy,

exhausting diarrhœa, serous inflammations, or from the effects of the original disease.

**Diagnosis.**—If there is a sufficient cause, such as phthisis, syphilis, or suppurating lesion, and evidence of lardaceous change in the liver and the spleen, which are generally enlarged, smooth, and hard (though they may be considerably diseased without increase of size), then the presence of albumin in the urine speaks strongly for lardaceous degeneration of the kidneys. All the more is this the case if the urine does *not* present the features of ordinary nephritis—that is, if it is of normal quantity and colour, or if, being abundant, it has a larger quantity of albumin than is common in granular kidney. If the condition of the urine is compatible with an acute or subacute tubal nephritis, lardaceous disease can only be inferred from the presence of an exciting cause and the evidence of the change elsewhere.

**Prognosis.**—This is decidedly unfavourable, especially if the stage of dropsy is reached. But in earlier stages, and in cases where the causative condition can be more or less controlled—for instance, tertiary syphilis by suitable treatment, diseased joints and necrosis by various operations—the condition may last for some time without causing grave symptoms, and even it seems possible that practical cure may result.

**Treatment.**—This consists in removing the cause so far as it can be done. Beyond this the administration of potassium iodide, of cod-liver oil, and of mild preparations of iron, such as the syrup of the iodide, seems to be attended with some benefit. Complications must be treated in the same way as in the different forms of Bright's disease.

## RENAL HÆMORRHAGE

In the majority of cases in which blood appears in the urine it has escaped from the substance of the kidney and not from the mucous membrane of the urinary passages or bladder below; and the condition of hæmaturia has been described as one of the important symptoms of nephritis. But hæmaturia is frequent in other diseases of the kidney, such as tubercle, cancer, calculus, bilharziasis, extreme venous congestion, general diseases involving the kidney, whether definite infections or not, such as typhus, the enteric fevers, yellow fever, black-water fever, acute yellow atrophy of the liver, purpura hæmorrhagica, and others.

Thus hæmaturia becomes a symptom, about which it has to be determined, first whether the blood comes from the kidney or from the urinary passages below it, and secondly what is the pathological condition of the kidney or bladder which has caused it.

It must, of course, not be forgotten that the urine may be coloured red by hæmoglobin without the presence of actual blood, and by some other red pigments (*see* pp. 539, 608).

The diagnosis of the different occurrences above mentioned is repeatedly considered in the various sections, and it will be seen that so far as a renal hæmorrhage is concerned there is little in the characters of the urine itself which will help, but that the diagnosis depends chiefly upon the associated circumstances. In some cases, there are at the time no such circumstances: for instance, in children hæmaturia may be the first and only symptom of calculus, without any pain or other trouble; and in cancer of the kidney blood may appear in the urine without pain till much later. In due course, however, a solution of the problem is generally reached; but there are rare cases in which hæmorrhages from the kidney of considerable extent and frequently repeated have occurred without an explanation being afforded at any time; and such cases have been called *essential hæmaturia* or *nephrostaxis* (στάζω, to let fall drop by drop. (Cf. Epistaxis, Gastrostaxis)).

A patient of Sir Frederick Taylor's, a lady of middle age, had attacks of

hæmaturia for fifteen or more years, each lasting from five to eight weeks, with intervals of six to nine months in which the urine was normal. Eleven years ago a laparotomy was performed and the kidneys were found to be normal. The necessary instruments showed that the bladder was healthy, and that the blood came from both kidneys. She had had other troubles, especially attacks of diarrhœa, and abdominal pain, but they had no certain relation to the renal hæmorrhage; and there had been other local disturbances which did not help to explain the hæmaturia in any way.

Some cases have been also published in which attacks of true hæmaturia have been apparently brought on repeatedly by exposure to cold, or to the strain of exercise (Edgeworth, Brasher).

In any similar case the possibility of the existence of such diseases as acute nephritis, renal or vesical calculus, cancer of the kidney or villous growth in the bladder must be carefully excluded before the diagnosis of nephrostaxis can be accepted.

## PARASITES IN THE KIDNEY AND URINARY ORGANS

The parasites invading the urinary organs are the *Echinococcus granulosus (hydatid)*, the *Schistosoma (Bilharzia) hæmatobium*, and the *Strongylus gigas*. The last is exceedingly rare, and need not here be described.

### HYDATID DISEASE OF THE KIDNEY

The life history of the *Echinococcus granulosus* and the development of hydatid cysts have already been described among the diseases of the liver. Hydatid cysts are very rare in the kidney; they form either in the substance of the gland, or between it and its capsule, and they grow to a variable size. They undergo the same changes as they do in the liver, and lead to corresponding local difficulties. As the cyst grows it gives rise to a tumour, which is generally globular and tense, and exerts considerable pressure on surrounding parts. Not infrequently it ruptures into the pelvis of the kidney, and the daughter-cysts, either whole or in fragments, escape and are discharged with the urine, if they are small enough to pass down the ureter. The cyst may rupture into the intestine, or it may, after compressing the diaphragm and the base of the lung, open into the bronchi. Such ruptures may occur spontaneously, or be brought about by a blow or other injury. In other cases the cyst suppurates, or it becomes converted into the putty-like remains which have been previously mentioned (*see* p. 494)

**Symptoms.**—One of the symptoms, and it may be the only one, is the presence of the tumour formed by the cyst. It is situate in the loin, with the colon in front of it; it is generally more or less globular and tense, and occasionally, but not always, gives the so-called hydatid thrill on percussion. The cyst may not be large enough to be detected, and may rupture in its early stage. If it bursts into the pelvis of the kidney, the urine will contain daughter-cysts, or shreds of them, or a milky detritus, in which the characteristic hooklets may be found. The cysts, or any portions of them, may become impacted in the ureter, causing renal colic; or in the urethra, after passing through the bladder. If rupture takes place into the intestine or bronchi, cysts, or portions of them, will be got rid of by these passages, or the discharge of them may soon cease, and recommence after a longer or shorter interval. Pyelitis and cystitis may result from the passage of hydatids; but if no rupture takes place the urine is quite normal. Suppuration of the mother-cyst produces an abscess, which becomes manifest by increasing pain, tenderness, the implication of the surrounding tissues, and characteristic fever.

The **Prognosis** is fairly favourable, since a free discharge of the cyst and its contents is so often possible, and, indeed, a good many recoveries have been

recorded. The duration of hydatid of the kidney is variable, and may be as long as even thirty years.

The **Diagnosis** depends upon the presence of a renal tumour, combined with the discovery of cysts or hooklets in the urine. This latter is not in itself conclusive as to its position in the kidney, since a hydatid cyst behind the bladder may rupture into it. In doubtful cases a rectal examination should be made. A history of renal colic accompanying the discharge of cysts or fragments would be strongly in favour of their origin in the kidney. The tumour formed by the hydatid is most likely to be confounded with *hydronephrosis;* this may sometimes be distinguished by the typical variations of size; for, though a hydatid may also empty itself, this will be most likely accompanied by the appearance of daughter-cysts, or scolices and hooklets, in the urine. But the resemblance may be so close as only to be solved by the use of the exploring needle, when the hydronephrosis will yield a urinous fluid, the hydatid a clear or opalescent fluid with a minute quantity of salts and a mere trace of albumin, and perhaps under the microscope some scolices and hooklets. Serum tests in hydatid disease have been described (*see* p. 495).

**Treatment.**—The withdrawal of the fluid by aspiration has yielded good results in the case of the kidney, as it has in the case of the liver. But the more certain method of cure is to cut down upon the tumour, through the loin if possible, to empty the cyst by tapping, and then incise it, and stitch the membrane to the edges of the wound in the parietes. If the contents of the cyst are being discharged by the bladder, this operation is not necessary. Impaction of cysts in the ureter requires the treatment proper to renal colic: opium, hot baths, and soothing local applications.

## BILHARZIASIS

(*Schistosomiasis*)

In different parts of Africa (Egypt, Natal) the inhabitants are liable to a form of endemic hæmaturia, which is due to the invasion by parasites of the mucous membrane of the urinary passages. In other parts of Africa, South America, West Indies and China similar parasites occur which infect the intestinal canal and produce abdominal symptoms.

The latest researches (Leiper) into the life-history of these parasites show that in the infected districts of Egypt and elsewhere fresh-water snails act as intermediate hosts. The eggs which are voided in the urine or fæces of infected individuals give rise to ciliated embryos, which swim about and enter the snails, and multiply as sporocysts in the liver, eventually producing cercariæ (larvæ) by internal budding. After four or six weeks these larvæ make their way through the intermediate host into the water, where they can survive for one or two days. The natives of Egypt bathing, or washing, or standing in the pools or canals inhabited by the snails, are infected through the skin, or through the mucous membrane of the urethra. Three such parasites are now recognised, differing in their eggs, in their larvæ or cercariæ, in the molluscs they infect, and in the clinical symptoms and pathological conditions which they cause in man. They are unisexual flukes belonging to the order *Distomidæ*. The one which causes the lesions in the genito-urinary tract was first described by Bilharz, and is called *Bilharzia hæmotobia* (or *Schistosoma hæmotobium*). It is elongated in shape, the male being about $\frac{1}{2}$ inch and the female $\frac{3}{4}$ inch in length. In copula, the females lie in a groove on one side of the male, called the gynæcophoric canal. The eggs are $\frac{1}{170}$ inch in length and $\frac{1}{1050}$ inch in thickness, and present at one end a sharp terminal spine. The snails, *Bullinus contortus* and *B. dybowski*, are the intermediate hosts in Egypt, and *B. africanus* in South Africa.

The parasites have been found in the minute veins of the bladder, ureter pelvis of the kidney, uterus and rectum, and in the portal vein and its tributaries

they lie in smooth-walled spaces, which are, no doubt, altered veins. As a consequence, extensive inflammatory changes occur in the mucous membrane and submucous tissues of the bladder, ureter, and pelvis. The mucous membrane of the bladder, especially posteriorly, presents patches from ¼ to 1 inch in diameter, which are swollen, vascular, ecchymotic, and covered with tough mucus or yellowish exudation; or there are warty prominences encrusted with urinary salts. The eggs of the bilharzia are found in great numbers in the mucus and exudation on the surface, and in the mucous and submucous tissues. In the ureter similar changes occur, especially in the lower half. In the Egyptian, such complications as septic cystitis, pyonephrosis, urinary calculi, vesical carcinoma and fistulæ are seen.

The second species of bilharzia is the *Bilharzia* or *Schistosoma mansoni*, which has for an intermediate host in Egypt and other parts of Africa the snail, *Planorbis boissyi*. The eggs have a spine placed a little distance from the extremity, that is, lateral; the cercariæ differ from those of *B. hæmatobia* in their suckers and in the length of their tails: and the adult worms differ from the other form in the arrangement of the alimentary canal, in the number of the testes and in the position of the ovary. In South America this form is also common, and is then transmitted by other species of *Planorbis*.

It is this form which appears to be responsible for the remarkable changes which occur in the intestinal canal of the human sufferer. Thus, in the colon are seen numerous polypoid growths or adeno-papilloma, sessile or pedunculated; and these may be accompanied by a diffuse colitis. The rectum is sometimes almost blocked by similar adeno-papillomas, which may be mistaken for piles; the mucous membrane is much thickened, and beneath it are large hæmorrhages. The liver may show cirrhosis.

A third species of bilharzia occurs in China and Japan and is known as *Bilharzia japonica* (*Schistosoma japonicum*). The adults, which live in the portal vein, are smaller than the two preceding species, and are characterised by the smoothness of their cuticle. The eggs, which have no prominent spine, find their way out in the fæces, but are apt to become disseminated throughout the body and have often been found in the brain. This infection produces severe disturbance in man, with fever, urticaria and enlargement of the spleen; eventually cirrhosis of the liver, ascites and cholæmia are produced. Severe dysenteric symptoms result from the intestinal lesions. The intermediary host is a spirally-shaped fresh-water snail known as *Onchomelania japonica*. This bilharzia worm infects dogs, cats, and cattle as well as man.

**Symptoms.**– Clinically bilharziasis is divisible into two stages. The *toxæmic stage* occurs four to ten weeks after infection. The symptoms are pyrexia of variable length, marked urticaria all over the body, enlargement of liver and spleen, eosinophilia and abdominal pain and diarrhœa. After the disappearance of this stage there is a latent period of from six weeks to two and a half years, but eggs are being passed all this time. Then follows the *localised stage*, which is vesical or intestinal, depending on the parasite. In vesical bilharziasis the symptoms are hæmaturia, sometimes pain in the penis or perineum, and, in severe cases, anæmia from loss of blood. The hæmaturia is mostly of vesical origin; the urine is passed clear, and is followed by about a teaspoonful of blood. Micturition is frequent. Passage of blood and mucus per rectum also occurs. There may be secondary anæmia.

In intestinal bilharziasis there are attacks of diarrhœa, accompanied by passage of blood and mucus, but rarely by tenesmus. There is sometimes emaciation. It must be remembered that simultaneous double infections are common, and that symptoms of both types may be present together. In many cases the symptoms are only mild, though they may last for years. On the other hand, the various complications already mentioned may supervene and cause a fatal result.

**Prevention.**—Bathing in rivers and fresh-water pools should be prohibited. Obviously the great desideratum is the extermination of the snails, as of the mosquito in countries subject to malaria, but this may be impossible in countries where they are very widespread. Where the domestic animals are not infected efficient treatment of patients with antimony tartrate will lessen the disease by cutting off the supply of eggs from the intermediate hosts (**10**).

**Treatment.**—Intravenous antimony tartrate kills the eggs deposited in the tissues, and afterwards kills the parasite. It should be used in all but debilitated subjects or those who cannot tolerate it; in these cases intravenous emetine, 1½ grains, repeated fifteen or twenty times in one and a half to two months, may be tried instead.

Recently sodium or potassium antimony tartrate, sterilised by boiling, has been recommended as a specific. Intravenous injections are made alternate days, or three days a week. The first injection is ½ grain dissolved in 5 to 10 c.c. of sterile distilled water, and this is increased by ½ grain at each injection until the maximum dose of 2 grains (or 2½ grains) is reached, the total amount making a course being 20 to 30 grains. In children under ten half quantities are employed. It is important to prevent the drug from getting into the subcutaneous tissue. The injections may cause a metallic taste and dryness of the mouth, with a feeling of constriction in the throat, tightness in the chest, and later colicky pains in the abdomen, and possibly giddiness, headache, sickness and diarrhœa. The presence of these symptoms calls for judgment in graduating the next dose (**14**), but are to a great extent obviated by using freshly distilled water as a menstruum for the drug, and by injecting only a small quantity of fluid, at the maximum 10 c.c. On account of the extensive systemic fibrosis produced, the Japanese form of the disease is not so amenable to treatment as are the other two.

## NEW GROWTHS IN THE KIDNEY

The following tumours are met with in the kidney: Adenoma, fibroma, forming small round nodules in the pyramids, leukæmic deposit, masses of lymphadenoma in Hodgkin's disease, cavernous angeioma, papilloma, epithelial carcinoma, and embryonic tumours. The fibroma in reality starts as a myoma, and fibrous tissue is deposited as the tumour grows older. Embryonic tumours and carcinoma are, as a rule, the only ones large enough to become of clinical importance. Small accessory suprarenals are also seen in the upper pole of the kidney. They are of no clinical importance

### EMBRYONIC TUMOURS

These are generally primary, and frequently occur in quite young children or infants. The organ is enlarged to an immense size, often filling half the abdomen. It presents the usual characters of a renal tumour, filling out the loin, but increasing downwards and inwards towards the umbilicus, having the colon in front of it, and rounded or oval, with no sharp edges or notches. Histologically the tumour recapitulates the development of the kidney, being composed of tubule cells and stroma containing unstriped muscle fibres. It may contain round cells and spindle cells; and its consistence varies, sometimes being hard, at others so soft as to invite exploration for fluid. The tumour grows rapidly, causes neither pain nor hæmorrhage, but kills finally by exhaustion and emaciation. It is often bilateral.

### EPITHELIAL CARCINOMA

This is primary or secondary. *Secondary* cancer occurs occasionally in the course of cancerous disease of other organs, such as the female genitals, the rectum, other abdominal viscera, or the female breast. The nodules are small,

and their presence is not generally productive of special symptoms. As a rule, both kidneys are involved. *Primary* carcinoma occurs mostly in persons of middle or advanced age, and is more frequent in men than in women. There are two types of primary carcinoma. In the first place, there is the carcinoma of the renal pelvis, consisting of squamous or transitional cells. The tumour keratinises very quickly, becoming a typical epithelioma. It is found in association with calculi; and it may be assumed, as in the analogous case of hepatic cancer and biliary calculi, that the calculi have acted as a constant source of irritation. It mostly affects one side only. It causes considerable enlargement of the kidney, but rarely to the same size, proportionately to the body, as does sarcoma in children. Hæmorrhage and softening often take place, as in cancers elsewhere. In the second place, there is the tumour which arises from the substance of the kidney itself. The cells very readily undergo fatty degeneration and hydropic distension, and when this is the case, they are called *Grawitz* tumours, or, from the close resemblance of their elements to those of the fasciculate layer of the suprarenal cortex, have been called *hypernephroma*, and they have been thought to arise from suprarenal remains in the substance of the kidney; but they present features which are not present in the suprarenal structures, and are more probably epithelial growths from the cells of the convoluted renal tubules. They are often encapsuled, and present on section irregular division into lobules by fibrous tissue, with scattered areas, some of bright yellow colour, others of red or brown colour, in addition to cysts the walls of which may be calcified. The tumour cells vary in size, often reaching the dimensions of giant cells; and they have a tubular or alveolar arrangement, or are grouped radially round vessels. Clinically these tumours are especially important, as they readily invade the pelvis of the kidney and give rise to hæmaturia.

In other cases the carcinoma does not resemble the suprarenal, but has cells of relatively small size with a large nucleus and a protoplasm darkened by numerous granules.

**Symptoms.**—These are tumour, hæmaturia, and pain. The characters of the *tumour* have already been in part indicated; it may be irregular or nodulated; its mobility may be limited by adhesion; manipulation will elicit tenderness Rarely the tumour pulsates, and a *bruit* may be heard in it. *Hæmaturia* is frequent; it is intermittent, variable in amount, but generally moderate. Albumin does not occur without blood, unless there is independent Bright's disease. Cancer cells are sometimes recognised, but they may be lost among blood corpuscles; or epithelium from the bladder or pelvis may be mistaken for them: and on the whole they cannot be relied upon for a diagnosis. Apart from blood and cancer elements, the urine may be perfectly normal in quality, density, and colour. *Pain* is variable, often absent in the early stages, but likely to be very severe when the tumour reaches a large size. It is situate in the loin and hypochondrium, or shoots down the groin to the thigh; it is not associated with retraction of the testicle. If blood coagulates in the pelvis of the kidney, the passage of the clots by the ureter may be accompanied by the pain of renal colic (*see* Renal Calculus). Extension of the growth to the lymphatic glands may lead to pressure upon the roots of the spermatic veins and the production of a *varicocele;* or *œdema of the legs* may occur.

The remaining symptoms of cancer of the kidney are anorexia, nausea, vomiting, irregularity of the bowels, either constipation or diarrhœa, and progressive emaciation and anæmia, as in other malignant affections. The duration varies with the kind of cancer— it may be from six months to two years, or even more.

**Diagnosis.**—Cancer of the kidney may be recognised by the co-existence of pain, hæmaturia, and tumour of the renal region; but, in the absence of hæmaturia, the nature of the tumour has to be very carefully investigated. It has to be distinguished, first, from enlargements of other organs; secondly, from other diseases of kidney. From enlargement of the *liver* it is distinguished on the right

side by the presence of a band of resonance between its upper margin and the ribs; if it is adherent to the liver, the edge of that organ may still often be felt on the anterior surface of the tumour. The presence of bowel (colon) in front of the tumour is also characteristic of its renal origin. The same holds good for the left kidney as compared with the *spleen;* and the latter is recognised by its sharp notched edge, its smooth surface, and its generally uniform enlargement in a downward and inward direction. An *ovarian* tumour grows up from below, whereas a renal tumour begins above and grows downward. *Mesenteric glands* lie nearer the middle line than the kidney, and form very nodular masses; isolated enlarged glands may give a clue to the nature of the larger mass. If cancer of the *colon* simulate renal cancer, intestinal symptoms are mostly present. *Accumulated fæces* on the left side would show more variability in size and consistence, and the diagnosis can be cleared up by the use of an enema. *Perinephric abscess* gives infiltration of the skin, local pain and tenderness, and febrile reaction. The development of varicocele in an elderly man should lead one to examine the loin for a tumour; and the association of a tumour of a bone with hæmaturia should suggest the existence of hypernephroma.

The diseases of the kidney which may resemble cancer are pyonephrosis, hydronephrosis, hydatid, cystic degeneration, and tuberculous disease. From the first three it should be distinguished by its solid feel; but a cancer may exceptionally be very soft, or a renal cyst may be small and tense, so as to give no certain sense of fluctuation. A tense uniform globular surface would be in favour of a cyst, but a lobulated hydronephrosis may be mistaken for new growth. By the time that *tuberculous disease* causes enlargement enough to resemble cancer, it is practically a pyonephrosis. *Cystic* disease commonly involves both kidneys, and has not the other local signs of cancer; the pale abundant urine, with a trace of albumin, should distinguish it. In cases of doubt as between cancer and cystic enlargement, the aspirating needle may be safely used; and X-rays and the cystoscope will often assist the diagnosis.

**Treatment.**—If cancer of the kidney is recognised early enough, its removal by operation should be attempted, otherwise the treatment must be mainly palliative; the objects being to relieve pain by opium and local applications, to keep up the strength by an easily digestible diet, and the moderate use of wine or small quantities of brandy.

## CYSTS IN THE KIDNEY

The following forms of cystic disease of the kidney occur: (1) Small cysts in granular kidneys already described; (2) extensive cystic change known as *cystic disease* or *polycystic kidney;* (3) simple cysts; (4) dermoid cysts, which are exceedingly rare, and have the same characters as elsewhere; and (5) hydatid cysts, which have been dealt with under Parasites in the Kidney.

### CYSTIC DISEASE

*(Polycystic Kidney)*

This is a congenital disease, and may lead to enormous abdominal distension of the fœtus, with serious difficulty in parturition. Less advanced degrees of it are compatible with life, and it may be found at any age. The condition is usually bilateral. In adults the organs weigh from 1 to 5 or 6 lbs. It is more common in males than in females.

**Pathology.**—On section the kidneys are seen to consist almost wholly of cysts of various sizes, containing a fluid which is clear or turbid, yellow, pink, red, or purple, sometimes viscid, colloid, or purulent. Always albumin, and sometimes blood discs, leucocytes, and cholesterin, are found in the fluid, but

urea and uric acid are generally absent. The cysts are surrounded by fibrous tissue, in which only remnants of renal tissue can be found; they are lined with epithelium. The pelvis, ureter, and bladder are healthy, or the pelvis may be dilated. The pathology of these kidneys is still open to doubt. The view of Shattock has been widely accepted that a fault has occurred in the development of the kidneys. The latter are formed from two different sources. The pelvis and collecting tubules arise from an upgrowth of the ureter which comes from the Wolffian duct; the rest of the kidney comes from the "intermediate cell mass," derived from the mesoblast of the cœlom. When these two fail to meet each other, the secretion of the kidneys does not escape, and cysts are formed above the junction.

**Symptoms.**– In congenital cystic disease the kidneys may occupy the greater part of the abdomen, and press upon the diaphragm; and death may happen *in utero*, or during birth; or the child may survive a few months, or in the event of the disease being unilateral, a few years, when at length death may result from uræmia. The change is often associated with other congenital malformations, both of the urinary organs and other parts.

In adults the symptoms are often very obscure, but resemble those of chronic interstitial nephritis. The urine is generally abundant, of low specific gravity, pale in colour, and contains a little albumin and some granular casts. Sometimes there is hæmaturia. Hypertrophy of the heart and high arterial tension and raised urea in the blood occur in advanced cases. There may be lumbar pain, but dropsy is generally absent. The kidneys are often large enough to be easily felt. They occupy the usual positions of the kidneys, are rounded, firm or elastic in different degrees, and descend on inspiration. One may be larger than the other. In a patient under Sir Frederick Taylor, there was daily intermitting pyrexia for several weeks preceding death. The termination is often by uræmic coma; or there is cerebral hæmorrhage, or pulmonary complication, such as bronchitis, œdema, or pneumonia.

For **Diagnosis** one must rely upon the presence of the greatly enlarged kidneys (for the change is generally double), associated with the characteristic symptoms of chronic interstitial nephritis; but the disease is quite rare.

**Treatment** must resemble that of contracted kidney.

### SIMPLE CYSTS

Cysts of considerable size are sometimes found. They may be 3 or 4 inches in diameter, and exceptionally very much larger. They arise from the cortex, and project on the surface. Their contents are a clear limpid or gelatinous fluid, containing a little albumin and some salts, but no urea or uric acid. The remainder of the kidney may be quite healthy. Such cysts may be unrecognised during life; if very large, they form tumours which may require to be tapped and treated surgically on the same principles as hydronephrosis.

## MOVABLE KIDNEY

The name *movable kidney* is given to one that is readily displaced from its normal position, and can be moved more or less freely in the abdomen. This unusual mobility may be congenital or acquired.

*Congenital mobility* is due to the presence of a *mesonephron*—that is, the kidney is partially or completely surrounded by peritoneum (like the colon), and is thus free to move about among the abdominal viscera. This condition is quite rare It is sometimes distinguished as *floating* kidney.

*Acquired mobility* is much more common. It affects females more often than males; and the right kidney is movable thirteen or fourteen times as often as

the left. Sometimes both are affected at the same time. The age of the patient is mostly between twenty and fifty. It mainly results from conditions which stretch or relax the tissues and structures surrounding the kidney, especially the fatty capsule and the peritoneum Perhaps the most frequent cause is repeated pregnancy, by which the peritoneum is dragged upon and stretched, and fails after delivery to recover its normal tension. But movable kidney is not confined to those who have borne children. Emaciation by reducing the fat surrounding the kidney may be a cause sometimes. Many patients have a pendulous abdomen, and the general want of tone in the abdominal and pelvic tissues which constitutes Glénard's disease (*see* p. 416). An increase of size of the kidney from any cause must favour it. Tight-lacing has been charged with it, but it frequently occurs independently.

**Symptoms.**—The most common subjective symptom is a sensation of weight, or dragging, or pain in the loin or side of the abdomen affected; and this may be constant, aggravated by walking or exertion, and relieved by lying down. From time to time there may be severe attacks of so-called *strangulation* of the kidney (*Dietl's crises*), consisting of great pain and tenderness in the renal region, with scanty, high-coloured, and even bloody urine. There may be nausea or vomiting, and malaise, but generally not much pyrexia. Such an attack, which subsides in the course of a week or more, is probably due to twisting or kinking of the renal vessels by the movement of the kidney.

Rarely does a movable kidney produce any considerable pressure on surrounding organs, for its very mobility renders this unlikely; but œdema from pressure on the inferior vena cava has been recorded. Some gastric disturbances, such as nausea, flatulent distension, etc., are probably due rather to the nerve connections of the kidney and of the stomach than to pressure on the duodenum, as suggested by some. Lastly, many patients suffering from displaced kidney are nervous, hysterical, neurasthenic, or hypochondriacal.

The evidence of movable kidney lies in its detection by palpation of the abdomen. In the majority of cases it is felt only in the flank of the side affected. Here a smooth, firm, but not hard, rounded tumour may be felt, of the size of the kidney. If it lies between the last rib and the crest of the ilium, it can be pushed more or less in all directions, but most easily upwards towards the thorax, when it may get entirely out of reach, leaving the flank normal. Often when the patient lies down nothing is felt until she takes a deep breath, when the kidney glides down, and may be secured by dipping the fingers in above its upper extremity. If the organ be pressed firmly, or grasped, the patient experiences a sharp pain, or sickening sensation. The examination should be made with both hands, one pressed firmly in between the last rib and the crest of the ilium, the other on the front of the abdomen. In some cases the kidney rises towards the front of the abdomen, or rests in the iliac fossa, or can be pushed over towards the middle line or beyond it. A further examination in the erect posture may help our estimate of the extent of the displacement. Apparently no difference in respect of mobility exists between the kidneys which possess a mesonephron and those which do not. It should be noted that a transitory albuminuria may result from too free manipulation.

Hydronephrosis from repeated kinking of the ureter is a possible result of this complaint; except for this, mobility of the kidney is rather a discomfort than a serious disease, and does not tend to a fatal result.

**Diagnosis.**—A movable kidney is likely to be confounded with a small ovarian cyst, with distended gall bladder, with tumour of the omentum, mesentery, stomach, pancreas, ascending or descending colon, with retained fæces, or with enlarged spleen. *Ovarian tumours* are movable only in directions determined by their pelvic connections, and cannot be pushed up into the loin. A *distended gall bladder* has dulness continuous with that of the liver, but may be extremely movable. Its cystic nature may be recognisable. *Tumours of the*

*alimentary canal* or *pancreas* have rarely a perfectly smooth surface, vary in shape and size, and are more continuously painful. An enlarged *spleen* is never behind the intestines; the lower it lies, the more it gets to the front of the abdomen, and it is always close under the parietes.

**Treatment.**—The object of treatment, if symptoms are severe enough to require it, is to retain the kidney in its normal position, and so to prevent the weight, dragging pain, and other sensations, as well as the strangulation which may result from kinking of the vessels of the ureter. Abstention from violent exercise may be enough in some cases, and rest in the recumbent posture will give temporary relief. But as getting about again brings on the troubles, it is desirable to attempt support of the kidney by some kind of pad, truss, or bandage. A spring truss, with a large pad pressing on the front of the affected loin, may be used; but is likely to lead to atrophy of the tissues upon which it presses. Better is a broad bandage, extending from the groin to the sixth or seventh rib, with a large pad sewn into it, in such a position as to press into the right flank and thus prevent the fall of the kidney An air pad, which can be inflated while in position, is often effectual. A further desideratum is to strengthen the abdominal muscles by suitable gymnastic exercises. The treatment of the symptoms of strangulation consists in complete rest, the use of poultices and hot fomentations to the loin and abdomen, and opium or morphia by injection or suppository.

## RENAL CALCULUS

(*Nephrolithiasis*)

The following are the varieties of urinary calculi. The first five are the commoner forms; the others are much more rare:

1. *Uric Acid.*—Hard, round or oval in shape, smooth or finely tuberculated, sometimes facetted from contact; of yellowish, fawn, or reddish colour. They vary in size, from that of poppy seeds to that of mustard seeds or peas, and are occasionally very much larger. Frequently they exist in great numbers.

2. *Sodium Urate.*—Soft, not generally of large size.

3. *Calcium Oxalate, or Mulberry Calculus.*—These are very hard, rough or irregular on the surface, and of blackish-brown colour; when smaller they are smooth, rounded, grey or brown in colour. Generally they are solitary.

4. *Mixed Calcium and Ammonium-magnesium Phosphate, or Fusible Calculus.*—The mixed phosphates are precipitated in urine rendered alkaline by ammoniacal decomposition, such as occurs when the secretion is retained in the bladder or in a dilated pelvis. They rarely form the nucleus of a stone, but are deposited upon other calculi of uric acid or oxalate, upon foreign bodies (*e.g.* in the bladder), and upon the inflamed mucous membrane of the bladder or of the renal pelvis. They may thus enormously increase the size of vesical stones, and in the pelvis may form concretions, which are moulded to all the infundibula and calices (*dendritic calculi*). The deposit is white, soft, and friable; and fuses under the blowpipe into a kind of enamel.

5. *Calcium Phosphate.*—White and chalky, rather smooth on the surface, with an earthy fracture, varying in size from that of a pea to that of a hen's egg.

6. *Calcium Carbonate.*—Small, very hard, smooth, grey, yellowish or bronze-coloured, and varying from minute grains up to stones the size of a hazel nut.

7. *Cystine.*—Usually egg-shaped, the surface granular, glistening with crystals of yellow colour, looking translucent on section with indications of a radiating structure, and rather soft in consistence. They become green on exposure. With a lens the hexagonal form of the crystals may be seen.

8. *Xanthine.*—In physical characters like uric acid calculi, but of a cinnamon colour, soluble in liquor ammoniæ and liquor potassæ. They are extremely rare, and have not been found in the renal pelvis.

9. *Urostealith.*—Soft, greasy concretions, which have been found in a few cases; one was shown to consist of about one-third cholesterin and fat, one-third uric acid, and some oxalates.

10. *Indigo.*—Once found in the renal pelvis by Ord as a calculus weighing 40 grains.

The majority of calculi consist of more than one of the above substances, and sometimes there are found alternating layers of uric acid, oxalate, and phosphates, laid one upon another under varying conditions of the urine. Phosphates and carbonates are deposited in alkaline urine; the remainder of the calculi above enumerated form in acid urine.

Urinary calculi vary much in size; they may be 2 or 3 inches in diameter, or they may consist of very small particles, and are then known as *gravel.*

The centre or nucleus (*i.e.* the first-formed portion) of most calculi is uric acid; but within that, calcium oxalate or sodium urate has been found. Some calculi are deposited upon a nucleus of blood clot, mucus, or renal casts, and the ova of *Bilharzia hæmatobia* may form the starting-point of renal stones. Most calculi are formed in the urinary tubules, and some even in the epithelial cells (Ralfe); and the frequency of uric acid and calcium oxalate as components of stone is, no doubt, determined by their relative insolubility. Another important factor seems to be the presence of some "colloid matrix," such as may be formed by mucus, blood, or perhaps the protoplasm of the epithelium cell; since it has been shown by experiment that in the presence of viscid solutions a chemical precipitation does not take place rapidly in a crystalline form, but more slowly in the shape of granules, spheroids, and laminæ, which has been called *submorphous.* Such colloid matter, therefore, determines the form of the precipitated matter, and may also bring about the precipitation in a secretion overcharged with the relatively insoluble salt. According to Ralfe there is often an impairment of vital power in the renal cells, so that they fail to secrete uric acid or oxalate, and hence these substances are actually deposited in the cells, the cell substance acting as "colloid." Ultimately the calculus grows by accretion of other deposits upon it. Roberts showed that the precipitation of uric acid from the urine was favoured by great acidity of the urine, by excess of uric acid, by decrease of the urinary pigment, and by decrease in the urinary salines; and that it was retarded by the opposite conditions.

Renal calculi are probably usually formed in the pelvis of the kidney, though there is evidence that in some cases they may begin in a cyst in the cortex and be discharged into the pelvis (**15**).

**Ætiology.**—Calculus is very much more frequent in the eastern part of England than in the middle or western parts; a difference which is not explained by the existence of a chalky soil in the former, since the majority of calculi from patients in these parts are of the uric acid variety. Stone is more common in males than in females, and perhaps more in early or late life than middle age. Ralfe pointed out that these were periods of vital impairment. For instance, febrile illnesses are common among children, and many suffer from malnutrition; on the other hand, in old age, besides the obvious waning of the powers generally, there is often local impairment from urethral stricture, or prostatic enlargement, or diminished expulsive powers.

**Symptoms.**—(1) When the stone exists in the pelvis of the kidney, it may be entirely latent, or it may give rise to lumbar pain, hæmaturia, albuminuria, or the passage of pus from the induction of pyelitis; and these changes may result from the presence of very small stones, or of the deposit known as gravel. (2) If it falls into the ureter, it may become impacted or move along with great difficulty, causing *renal colic,* hæmaturia, and, in certain circumstances, *obstructive suppression.* (3) The later effects of renal stones, either in the kidney, or after impaction in the ureter, are pyelitis of all degrees, pyelo-nephritis, perinephritis' perinephric abscess, hydronephrosis and pyonephrosis, the symptoms of which have already been described.

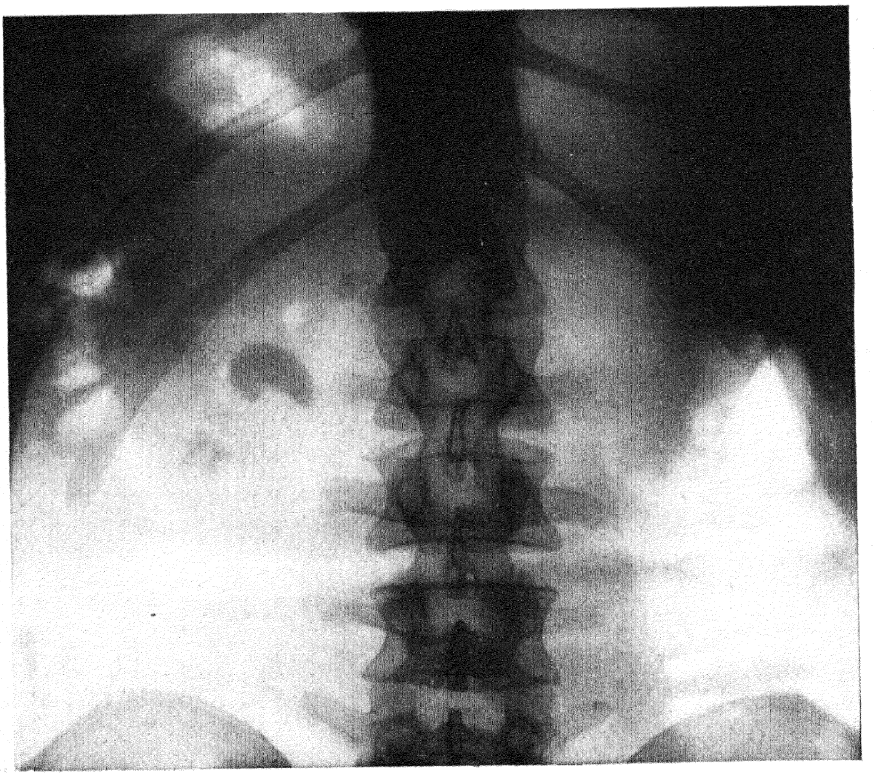
Gravel and calculus are frequent causes of *lumbar pain*, which is often regarded as "lumbago" or muscular rheumatism. The lumbar pain or aching may be rendered worse by jolting or shaking. If the symptoms are of long duration, albumin, pus, or mucus may be passed, and from time to time blood in varying quantities. Occasionally, small calculi or gravel are discharged with the urine. The subsidence of symptoms entirely after a long period of activity may be due to the calculus becoming encysted.

*Renal colic* is associated with the spasmodic contraction of the muscular fibres of the ureter, irritated by the impaction or the passage of the calculus. It is comparable with biliary colic, and is characterised by intense pains, rigors, nausea, and vomiting. The pain is situated in the loin and flank of the same side, and radiates downwards and inwards to the groin and testicle; sometimes to the thighs, and even to the heel and sole of the foot; at others to the abdomen, chest and back. In the severer attacks the patient is doubled up with the pain, or writhes on the floor, and bursts out into profuse perspiration, or he becomes pale and collapsed, with quick, feeble pulse; but the temperature may be raised. With this there are nausea and vomiting, often a rigor, and sometimes even general convulsions. The testicle on the same side is retracted, and is swollen and very tender. The pain may be less for a time, but soon returns, and altogether it may last a few hours or a day or two, until at length the stone is passed into the bladder, or returns to the pelvis, when there is a sudden relief, and only an aching, smarting sensation in the side is left. The pain may, however, cease when the calculus still remains impacted in the ureter. During the attack, micturition is frequent and painful, and the urine is scanty, coming, perhaps, only by drops; and it may contain blood; and, indeed, a diagnosis of cystitis may be wrongly made. If the bladder be examined with the cystoscope, signs of irritation of the mucous membrane about the orifice of one or other ureter may be observed, and the urine flowing from either orifice may come too slowly, or may be turbid, or blood-stained. By examination of the abdomen the kidney may be found to be tender; or the stone may be detected in the ureter, and its course watched from kidney to bladder.

Such an attack may occur spontaneously, or may be brought on by some movement which appears to dislodge the calculus from its position. Renal colic may recur in the same patient. This, of course, must depend on the number and size of the stones; obviously, if a stone gets back into the pelvis it may set up renal colic on again becoming impacted. Exceptionally a large number of stones in the pelvis of the kidney may be detected by a crackling sensation on palpation.

*Obstructive suppression* is distinguished from the suppression which results from acute congestion or acute Bright's disease, and the symptoms in marked cases are strikingly different. It arises when both ureters are simultaneously compressed, as occurs in women when cancer of the pelvic organs invades the base of the bladder; or when one kidney has been disorganised, or excised, or otherwise placed *hors de combat*, and a calculus becomes impacted in the ureter of the healthy organ. This condition is sometimes called *latent uræmia*. In some cases no urine is voided; in others, a certain amount may be passed in small quantities at long intervals, but it is clear, watery, of very low specific gravity—*e.g.* 1,006—and contains an extremely small quantity of urea or other solids: and there is no albumin, unless there is blood, or unless the urine is modified by the cystitis which accompanies cancer of the bladder. The patient's condition is not at first materially altered. He may eat as usual, but he loses muscular power, and becomes sleepless, and after some five or six days he is seized with muscular twitchings or jerkings, affecting the arms, legs, and trunk. The pupils are contracted, the temperature of the body falls, the breathing is slow, panting, and laborious, the mouth and tongue are dry, and there is great thirst, and there may be troublesome vomiting. The muscular twitchings

PLATE XXXIII.



Radiogram showing two Renal Calculi in the Left Kidney. The upper one is kidney-shaped, and situated in the pelvis. The lower one, which is much less opaque, and is at an earlier stage of development, is situated near the lower pole of the kidney. The rounded outer borders of both kidneys are seen outlined against the gas in the colon. They extend below the twelfth rib on each side. The iliac crests are seen at the bottom of the picture. The triangular shadow which extends upwards from the crests represents the psoas muscles. (From a plate by Mr. W. Lindsay Locke.)

[*To face p.* 659.

continue, and the patient becomes restless, indifferent, and drowsy, and coma may eventually occur. Death, as a rule, ensues from nine to eleven days after the commencement of the obstruction, and is very rarely postponed beyond this. Recovery may take place if the obstruction is removed by the passage of the calculus or by the breaking down of any new growth. There may be obstructive suppression of a less dangerous kind in the course of a double hydronephrosis; and Bradford shows that symptoms identical with the above may arise when the functions of the kidney, hitherto healthy, are disturbed by distant lesions, presumably acting reflexly, or by toxic influence.

**Diagnosis.**—The typical symptoms—lumbar pain, hæmaturia, and albuminuria—may be caused not only by a medium-sized calculus, but also by fine gravel and uric acid crystals, which will readily pass the ureter; and in these cases testicular pain and frequency of micturition may also be present. A severe attack of colic, associated with hæmaturia and testicular pain and retraction, is very strong evidence of calculus; but other foreign bodies besides calculus may

## ERRATA

P. 659. *For* Plate XXXI. *read* Plate XXXIII.

P. 666 *For* Plate XXXII. *read* Plate XXXIV.

P. 694. *For* Plates XXXIII.—XXXVI. *read* Plates XXXVI.—XXXIX.

P. 740. Morbid Anatomy of Multiple Neuritis. The secondary histological changes in the muscles are described under Progressive Muscular Atrophy on p. 800.

over the kidney is justified in some cases. It appears to be quite clear not only that a calculus may cause pain in the opposite flank at the same time as pain on its own side, but also that in exceptional cases the pain has been felt only on the side of the healthy kidney. Stone in the kidney may also cause suprapubic pain, and simulate stone in the bladder very closely. These pains must be set up reflexly, and they show how closely the nervous connections of all parts of the urinary tract are related to one another.

Some differences have been noted in the symptoms dependent on the chemical nature of the calculus. *Uric acid* calculi produce the least pain, which is dull and oppressive, with a sense of weight. The urine is acid; bleeding is frequent, not excessive, and periodic, apart from exertion. The mucous deposit is yellowish, or rusty. *Calcium oxalate* causes acute pain referred to a particular spot, as well as shooting pain in the ureter, shoulder, or epigastrium. The urine is less acid, bleeding is abundant, and the mucus is glairy and greenish. *Phosphates* cause severe unremitting pain, attended with exacerbations. The urine has been at some time alkaline, and there is much muco-pus or flocculent mucus, which may be tinged with blood, though free bleeding is rare.

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The influence of the seat of the hæmorrhage upon the character of the urine has been mentioned (*see* p. 616).

Any disease leading to hæmorrhage from the kidneys may for a time be mistaken for calculus, since hæmaturia may occur without other symptoms being prominent. In *cancer* of the kidney hæmorrhage is often more abundant, and more continuous. Gelatinous red lumps appear in the urine after the blood; and sometimes cancer cells may be found by the microscope. The discovery in due time of a tumour will help the diagnosis. Calculus is more likely in a young patient, but either may be present in middle or old age. *Tubercle* of the kidney may closely simulate calculus, by lumbar pain, frequent micturition, pus in the urine, and even blood. In the former there may be a family or personal history, or present indications of tubercle; hæmaturia and renal pain are less prominent and characteristic. *Cystitis* is simulated by the frequency of micturition which occurs in renal calculus, especially if hæmaturia is absent. Previous attacks of lumbar pain, and the acid reaction of the urine, if pus is present, are in favour of a renal origin.

In difficult cases diagnosis may require manual examination of the kidney in the loin, the use of the cystoscope, and the Röntgen rays, which will often show the presence of a calculus (*see* Plate XXXI.). However, uric acid and cystine calculi are not opaque to X-rays. A metallic sound may be passed into one or other ureter, so as to detect the stone by contact. Lastly, an exploratory incision in the loin over the kidney is justified in some cases. It appears to be quite clear not only that a calculus may cause pain in the opposite flank at the same time as pain on its own side, but also that in exceptional cases the pain has been felt only on the side of the healthy kidney. Stone in the kidney may also cause suprapubic pain, and simulate stone in the bladder very closely. These pains must be set up reflexly, and they show how closely the nervous connections of all parts of the urinary tract are related to one another.

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**Treatment.**—The frequent occurrence of gravel, which is nearly always uric acid, should be met by the administration of drugs to render the urine less acid, or even alkaline. The most efficient are the citrate, acetate, and bicarbonate of potassium or sodium, which may be given in 30 to 60-grain doses in 3 or 4 ounces of water two or three times daily, or in one larger dose before the night's rest, during which time the tendency to uric acid precipitation is greatest (Roberts). Another important point is to give copious draughts of water so as to dilute the urine and flush out the urinary passages. This will be of value not only when deposit is taking place in the kidney itself, but also if a small calculus is impacted in the ureter, as it may be dislodged by such means. Plain water or any kind of mineral water or natural water may be used. Where uric acid is suspected, a purin-free diet should be given, which means that all kinds of animal food made up of cells should be avoided. Alcoholic liquors should also be avoided. Ralfe recommended turpentine in capsules every morning or twice daily. Benefit is often derived from residence at Vichy, Vittel, Contrexéville, Ems, Carlsbad, Salzbrunn, Tarasp, Neuenahr, or Wildungen, where the waters are alkaline or saline. As a preventive of stone exercise is desirable.

If a calculus has actually formed and is stationary in the pelvis of the kidney, it is doubtful if it can be dissolved by any medical treatment. If there is no chance of the stone being passed naturally, *nephrotomy* or *nephrolithotomy* should be performed, or *nephrectomy* if the kidney is hopelessly damaged.

*Treatment of Renal Colic.*—Anodynes are here required, both to relieve the intense pain, and because they may also relax the spasm of the ureter, and so facilitate the escape of the stone. If the pain is severe, a morphia injection should be at once given; or morphia or opium may be given internally, or in suppositories, or chloroform or ether may be inhaled. Papaverine hydrochloride, one of the opium alkaloids, in doses of $\frac{1}{3}$ to $1\frac{1}{3}$ grains, has produced good results. Locally, hot poultices, hot fomentations, belladonna applications, or the hot bath should be used. The patient should be at rest, and warm diluent drinks, barley water, etc., should be taken from time to time.

## FUNCTIONAL ALBUMINURIA

It has been already shown that albuminuria may occur in a number of morbid conditions, of which nephritis and renal degenerations, acute illnesses, infective diseases, and venous congestion are the most important. But it is sometimes present in persons who appear to be in perfect health, and show no sign of disease on further examination. It may occur without any apparent cause, or it may be brought on by an exercise of function which in other individuals is quite harmless —*e.g.* muscular effort, or exposure to cold, or assumption of the erect posture. It may be of short duration, quickly passing away to recur from time to time, or it may persist for months.

Different cases of this disturbance have been described under the names physiological albuminuria, albuminuria of adolescents, intermittent albuminuria, remittent albuminuria, cyclical albuminuria, postural albuminuria, and functional albuminuria.

Maclean examined the urines of 50,000 soldiers and found albuminuria in 5·62 per cent. of them. In 2·55 per cent. the albuminuria was well marked. Many of these cases were probably examples of functional albuminuria. Numerous observations have also been made on the presence of albumin in the urine in groups of individuals, such as infants from one to six days old, schoolboys, bank clerks, soldiers, or workers in factories; and invariably albumin is found in a certain percentage, which varies from 5 to 30. In life insurance practice it is common experience that a large proportion of applicants between eighteen and thirty years of age, believing themselves to be perfectly well, have a small quan-

tity of albumin in the urine. The term albuminuria of adolescents represents a fact of great importance, the explanation of which is still under discussion.

**Pathology.**—The albumin is, as a rule, small in quantity. The interesting observation has been made that in cases of functional albuminuria, or non-nephritic albuminuria, as they have been called, there is a high percentage of globulin. Serum globulin consists of euglobulin and pseudo-globulin, and it is the euglobulin fraction that is present in the urine. This substance is often combined with lecithin and other lipoid substances, and occasionally the urine becomes opalescent when they are present in large amount. In the albuminuria of nephritis there is six times as much albumin as globulin, but in these cases the amounts are about equal. Euglobulin may be discovered by adding 33 per cent. acetic, drop by drop, to urine until a white precipitate is formed.

The following types, mostly in young people, require separate description.

**Athletic Albuminuria.**—Albumin in small, decided, or even considerable amount is sometimes found in the urine of those who have recently rowed or run a race, or have undergone other excessive physical exercise. There is no doubt that the albuminuria is only a temporary disturbance, indicative of deficient blood supply to the kidney, because so much blood is required for the muscles and skin.

**Dietetic Albuminuria.**—This is due to the ingestion of large quantities of protein food, *e.g.* 100 to 200 grammes of protein, for periods of two or three weeks. Red cells may be found in the urine due to the renal irritation from the high protein diet (**12**). Albumin is occasionally associated in the urine with crystals of calcium oxalate, which may possibly irritate the kidneys, and thus cause the passage of albumin; and such excess of oxalates may be produced by particular kinds of diet (*see* p. 606).

**Orthostatic Albuminuria.**—This includes the cases described as postural albuminuria, and probably also the cyclical, remittent, and intermittent forms. It occurs in persons in good health, more often youths or young adults; and it is characterised by the presence of albumin at certain times of the day, while it is absent at others. Thus, in the early morning it cannot be detected; it is present from about 9 a.m. to 5 or 6 p.m., and again disappears from the urine passed at night. The albuminuria is obviously determined chiefly by the assumption of the erect position, and by the accompanying exercise during the day; and it disappears as a result of the recumbent position at night. The amount of food taken has no influence upon it. Sonne has shown that in these cases the important factor is the presence of lordosis, which occurs naturally in the erect position. The left renal vein is compressed between the spine and the aorta. The albuminuria is caused by the circulatory disturbance. The condition is relieved when the patient is lying down. In ten cases of this kind, by catheterising the ureters he showed that it is only the left kidney which gives rise to albuminuria. This condition has been classed as *lordotic albuminuria*. The albuminuria of pregnancy may be partly a left-sided lordotic albuminuria (**13**).

**Paroxysmal Albuminuria.**—This may undoubtedly occur as a phase of paroxysmal hæmoglobinuria, in which the hæmoglobin breaks up into hæmatin and globulin (*see* p. 539). The patients have malaise, a sallow tint, and subsequently albuminuria; the urine contains at the same time excess of urea and of urobilin.

**Diagnosis.**—In order to distinguish these cases from the graver instances of Bright's disease, a single examination of the urine is seldom sufficient. If albuminuria is discovered, as it often is, in the routine examination of the urine for life insurance, or admission to a school, a city office, or to the public services, where the individuals are presumably healthy; or if it is found in a young person who is at most only a little languid and out of sorts, a diagnosis of Bright's disease should not be hastily made, but the urine passed at different periods of the day should be tested to see if the albuminuria is determined by any of the

factors—diet and position—alluded to, and especially to see if it is at any time absent. In chronic Bright's disease the quantity of albumin fluctuates during the day, and sometimes it is increased by exercise and diminished by rest; but its entire absence at night and during the early morning is very characteristic of the functional forms. The presence of euglobulin, as shown by the addition of acetic acid, is in favour of functional albuminuria. The functions of the kidney may be tested. If normal, this points in the same direction.

**Prognosis.**—The future of these cases is on the whole favourable, and in the large majority the albuminuria entirely disappears, though it may persist for two or three, or even seven or ten years.

**Treatment.**—Except in the dietetic form, no treatment is required. In this case the protein in the food should be diminished. Where calcium oxalate seems to be responsible, dilute nitro-hydrochloric acid may be given, and meat, excess of vegetables, and wines should be excluded from the diet.

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# DISEASES INVOLVING BONES AND JOINTS

## GOUT

This name is given to a form of acute arthritis associated with an increase of uric acid in the blood and a deposit of crystalline sodium biurate in the joints and other tissues. In the majority of cases the feet, and especially the great-toe joints, are first attacked, whence the classical name *podagra*; but the joints of the hand (*chiragra*) and other articulations are subsequently, and much less commonly, affected first. In the intervals between the attacks symptoms occur in other parts of the body, and in the course of time pathological conditions develop which point to the disease being general rather than local. For the symptoms remote from the joints the terms *atypical*, *irregular*, *abarticular*, *metastatic*, *retrocedent*, and *visceral* gout have at different times been used. Of the visceral affections the most important is primary contracted kidney, sometimes called gouty kidney (*see* p. 630).

**Ætiology.**—Gout is well known to be strongly hereditary, so that the descendants of a gouty stock are liable to outbreaks of the disorder at an early age and with comparatively little exciting cause. It is more common in men than in women, and is a disease of middle life or advanced age, though it does occasionally, in the hereditary cases just referred to, appear as early as the age of twenty; and it has been seen in boys who were only eight, nine, or twelve years old. It has been regarded as a disease of the rich, from which the poor escape; but this is not true, as the disease is often seen in its most typical form among hospital patients and others in poor circumstances. The influence of wealth is related to diet, which is the most important ætiological factor; and the ingestion of large quantities of food, especially those kinds which are rich in purin bodies, with abundance of alcoholic liquors, directly contributes to that condition of the blood and tissues which is the essence of gout. Of alcoholic beverages, malt liquors and the stronger wines, like port and sherry, seem to be more prejudicial than distilled spirits. The effects of dietetic excess are aggravated by a sedentary life; and, as a rule, an occupation is prejudicial in proportion as it tempts to one or necessitates the other. Prejudicial also are occupations which, like house-painting, type-founding, etc., expose the operatives to lead intoxication, and hence increase in them the liability to gout (*see* pp. 630, 681). In those who are predisposed to it, or who have already had manifestations, an attack may be brought on by an aggravation of the dietetic excesses, or by any departure from the strictest regularity hitherto found necessary, by anxiety and mental worry, and by injury. It is supposed that injury due to pressure of the boot on the metatarso-phalangeal joint of the big toe makes that joint so liable to attack.

**Anatomical Changes.**—In a joint which has been the subject of gouty inflammation, the cartilage is found to be covered with a bright white incrustation, either in patches or more or less completely. If perpendicular sections be made of this, it is seen to be due to a deposit of minute acicular crystals of sodium biurate in the substance of the cartilage. These form in the stratum of cartilage just beneath the surface, leaving at first a thin layer quite healthy, and in later

stages the deposit extends irregularly and by small more or less isolated patches into the deeper parts of the cartilage. In advanced cases the cartilage is quite destroyed and eroded down to the bone. Collections of biurate crystals take place also in the other tissues of the joints, for instance, in the ligaments, and in the cutaneous structures, so that the movements of the joints are considerably impaired; and in some cases this is assisted by some of those changes—osteophytes, etc.—which occur so commonly in rheumatoid arthritis. In bursæ, in tendon sheaths, in the cartilage of the ear, or in the skin of parts not immediately over the joints, the essential change is also the accumulation of biurate crystals. In the gouty kidney minute yellowish-white streaks, which are due to similar crystals, may be seen, especially in the pyramids. The kidneys are often of the primary contracted type (*see* p. 630), and the arteries may be thick and show degeneration. Exceptionally, true gouty deposit has been found in other situations—*e.g.* on the spinal meninges, and on the meninges of the cerebellum.

**Chemical Pathology.**—The uric acid of the urine is derived from two sources: (1) From the purin bodies in the food (*exogenous*). These substances (xanthin, hypoxanthin, etc.) are contained in tea, coffee, cocoa, in the nuclei of all cells, and so they are present in meat, sweetbreads and other cellular organs. The purins derived from this source are not altogether excreted as uric acid in the urine. A large amount is destroyed in the body. Still it is possible to raise very considerably the amount of urates in the urine by partaking largely of such rich foods. (2) From the wear and tear of the tissue cells of the body (*endogenous*). If a man takes a purin-free diet, *i.e.* lives on bread, vegetables, milk products and eggs, the uric acid in the urine is purely endogenous; and as this remains constant in amount for an indefinite period on a diet of this kind, it shows that the body has the power of synthesising purins. Histidin and arginin may be one source (Hopkins and Ackroyd). It has also been found that the endogenous uric acid falls to about a half on an almost protein-free diet (**1**), and also on a carbohydrate-free diet (**2**). These facts afford a basis for the prevalent view that a gouty person should not indulge too freely in protein or carbohydrate foods.

In gout the blood contains an excess of uric acid. This was first shown by Sir Alfred Garrod by his "thread test." This consists in placing 2 drachms of serum obtained from a blister in a shallow watch glass, adding 10 or 12 drops of acetic acid, placing in the serum three or four threads, and setting it aside for thirty or forty hours at the ordinary temperature. At the end of this time, if the threads be examined under the microscope, crystals of uric acid will be found to have formed upon them. The blood of a normal man contains from 0·5 to 2 milligrammes per cent. Before an attack of gout the value is 4 to 6 milligrammes per cent. But, in spite of this high figure, the amount of uric acid excreted in the urine is below normal. Further, if a gouty person is given purins in the food there is great delay in their excretion in the urine. This points to the failure of the excretory power of the kidney as being responsible for the high uric acid in the blood. An attack of gout is associated with a sudden rise in the excretion of uric acid in the urine, which reaches its maximum on the second or third day of the attack. At the same time uratic deposits are laid down in the joints and other places. The uric acid in the blood falls. Atophan (diphenyl-quinolin-tetracarboxylic acid) has been found to ward off attacks of gout; and it does so by increasing the uric acid excretion in the urine, and at the same time the uric acid in the blood and tissues falls (Folin). It also facilitates the excretion of uric acid if purin foods are given to a gouty person.

It must be remembered that the uric acid of the blood may be increased in diseases, such as leukæmia and severe nephritis, in which gouty attacks are absent, so that gout cannot be due merely to this cause; there must be an additional factor.

The acute articular inflammations of gout are attributed to irritation by the crystalline deposit of sodium biurate, which is found in the tissues; and possibly

the visceral attacks are due to a deposition, at least temporary, of the same biurate crystals.

**Symptoms.**—*The Gouty Attack.*—In the majority of persons gout first shows itself by an attack of acute inflammation in the metatarso-phalangeal joint of one great toe. Various premonitory symptoms are noted in different cases; in some there may be an unusual feeling of health or exhilaration; but more often there are the following: mental depression; disturbed sleep; odd sensations, itching, or cramps in the limbs; tinnitus aurium; salivation, gastralgia, vomiting, or flatulence; alterations in the quantity and colour of the urine, which is mostly scanty and deposits urates. These symptoms may have been troublesome for a day or two, when the patient is awakened, commonly about two o'clock in the morning, with pain in the great toe. The pain becomes worse and worse, and the patient finds it impossible to get ease. At the same time there may be a little chill, or even a rigor, and some fever. After some hours of excruciating pain this at length abates, and the patient may fall off to sleep; when he awakes again he finds the affected joint red and swollen. It is exquisitely tender; the skin is tense and shining, and if it can be touched, pits slightly on pressure. The veins around it are slightly distended. During the day the patient may be free from severe pain, but towards evening there is a recurrence of all the early symptoms—that is, of severe pain and some febrile reaction—which remit towards morning, to return again the following night. The joint continues swollen, and the swelling extends in the cutaneous tissues some distance up the foot; the colour is a dull, dusky red. When at length the inflammation subsides, which it does in from five to ten or fourteen days, the skin desquamates in large thick flakes, and gradually assumes its normal colour. In exceptional cases one or two toe-nails may be shed; on the other hand, in mild attacks desquamation does not occur.

The general condition of the patient is one of slight febrile reaction, with more or less gastric disturbance. The temperature is not much raised; the thermometer may reach 101°, but is rarely so high as 102°, and then only for a short time. The tongue is thickly furred, and the patient has no appetite, but much thirst, nausea, and sense of distension at the epigastrium; the bowels are confined, and the motions are deficient in bile. Sometimes the sense of distension and the tenderness extend to the hepatic region. Phlebitis with thrombosis of veins is not an uncommon accompaniment of the acute attack, and uræmia may also occur if the kidneys are diseased, and there is usually a considerable leucocytosis, due to an increase of polymorphonuclear cells.

When an attack of gout is at an end, the patient often feels better than he has done for a long time before; and he is, as a rule, free from any reminder of his condition for a period of several months, or even two, three, or more years. His second attack may be in the same joint as the first, an almost exact reproduction of it; or it may occur in the opposite foot, or in one ankle, or in the wrist or hand. A third attack often comes at a somewhat shorter interval than that between the first and second, and the periods of repose diminish in length as time goes on. Ultimately a great many joints have been at one or other time affected, and with repeated attacks they undergo changes which result in considerable deformity, so that the old gouty subject becomes crippled in somewhat the same way as the sufferer from rheumatoid arthritis (*see* p. 668).

When the disease has reached this stage it is in reality a *chronic gout*. If the small joints of the toes and fingers are often first affected, ultimately all the joints of the extremities may become the seat of gouty deposit, the shoulder and the hip less frequently. In the hand the joints are enlarged, are more or less fixed in different positions of flexion or extension, and in severe cases there is deviation of the fingers to the ulnar side of the hand. Similarly the foot may be fixed in a condition of talipes, or the knee or elbow in a flexed position. The swelling about the joints is often assisted by the existence of white deposits called *tophi*, which

at first lie close under a thin shining skin, with dilated venules. But subsequently the skin may yield, and the creamy or chalk-like deposit may escape in small quantities at a time; or, more rarely, suppuration takes place around the deposit, and leads to its more rapid elimination. These tophi are not confined to the affected joints; they are seen under the skin of the fingers adjacent, in the bursæ (for instance, over the olecranon), in the tendons, and with considerable frequency in the cartilage of the helix of the ear. If the creamy juice from one of these deposits be examined under the microscope, it will be found to consist of innumerable minute acicular crystals, which are composed mostly of sodium biurate, with a small proportion of calcium urate or phosphate, and sodium chloride.

When the kidneys are diseased, the blood pressure will be found to be high, the volume of the urine will be increased, and it will contain albumin and casts.

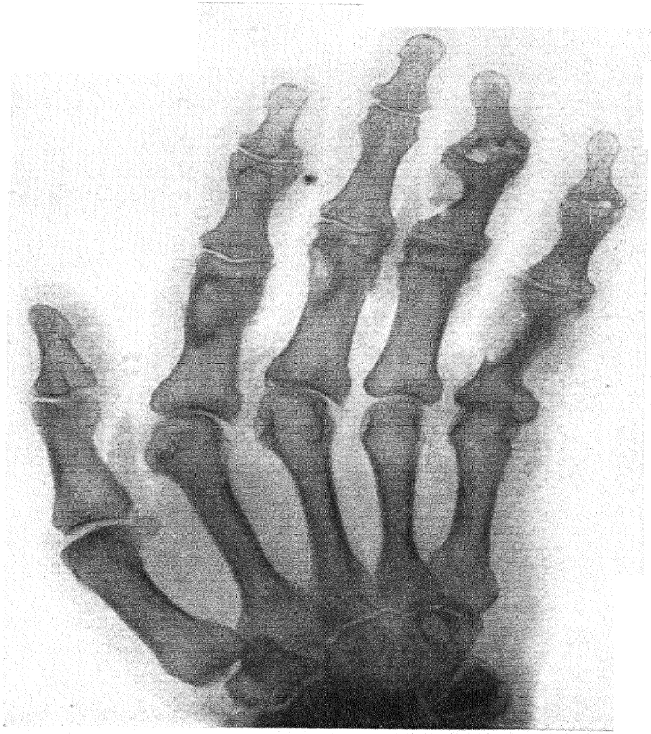
Gout sometimes occurs in the form of subacute or even chronic arthritis in two or more joints without any preceding typical acute attack in the great toe or hand. There is often more effusion into the synovial sac, less cutaneous redness, and a general resemblance to the polyarthritis of acute rheumatism.

*Irregular Gout.*—People who suffer from gout very often suffer from other diseases as well. Some of these, such as renal disease and phlebitis, are undoubtedly associated with gout; but others are almost certainly not related to gout at all, though in the past they have been considered as manifestations of a gouty diathesis, under the heading *irregular gout*. These disturbances, which it is necessary again to emphasise have nothing really to do with gout, are very various, and consist of inflammatory lesions and functional disorders in many organs in the body. The former include gastric and intestinal catarrh, bronchitis, conjunctivitis, iritis, gouty urethritis (which is, according to Ebstein, a prosta torrhœa), neuritis, cirrhosis of the liver, and chronic eczema. Amongst the functional disorders attributed to gout are migraine, vertigo, attacks of asthma and angina, muscular cramps, and lumbago.

**Diagnosis.**—The typical gouty attack occurring at night in the great toe can scarcely be confounded with anything else. It is distinguished from *acute rheumatism* by the dark red, shiny, tense swelling of one joint, the absence of general sweating, and the slight constitutional disturbance. Later illnesses implicating many joints show a closer resemblance. There is generally a history of many previous invasions of single joints, and less fever or sweating than in rheumatism; but the condition of the joints themselves cannot be relied upon as it can in early attacks. The swelling and redness of the back of the hand in gout may be such as closely to resemble *abscess*, but fluctuation can scarcely be obtained, and the history will mostly protect against errors. *Pyæmia* may be suggested by multiple gout, but rigors would be more severe, and the general disturbance more intense. In all cases the ears should be carefully examined for tophi, and, if necessary, microscopic examination of part of the tophus should be made to see if sodium biurate crystals are present, or the murexide test may be employed. In cases of doubt the uric acid in the blood may be estimated by Folin's method. A low value excludes gout, but it does not follow that if the value is high gout is necessarily present.

Radiographic examination of the chronically affected joints may be of value. Since sodium urate is not opaque to X-rays, clear areas in the neighbourhood of the joints may be seen. They have sharply defined edges, as if punched out, and are often round, varying in size up to that of a sixpence. Small pot-holes may be seen in the articular surface. The structure of the bone may appear altered, the striæ becoming irregular, and the shape of the bone may be deformed. This is ascribed to the irritation produced by the uric acid. Finger-like projections or exostoses may be seen projecting from the shaft (*see* Plate XXXII.).

**Prognosis.**—Gout once declared is likely to be repeated unless the conditions, dietetic or otherwise, which have led to it are altered. Acquired late in life, and properly treated, it may not materially shorten life; but it tends to



**Radiograms** illustrating Gout Note the sharply defined clear areas in the shafts of the proximal phalanges of the second, third and fifth digits of the right hand, the alterations in the striæ and occasional deformities; also the pot-holes in the articular surfaces between the second and third phalanges of the fourth and fifth digits of the right hand, and between first and second phalanges of the fifth left digit. Numerous finger-like exostoses are visible on the shafts. (From a case of Dr. George Graham's.)

[*To face p.* 666.

granular kidney, atheromatous arteries, and cerebral hæmorrhage. The risk of these sequelæ is more or less in proportion to the frequency of joint attacks, and hereditary gout developed early in life is very likely to be fatal by uræmia or apoplexy before old age is reached.

**Prevention.**—The quantity of food taken should always be moderate. Many patients are too fat, so that a loss of weight will be an advantage. Restriction of both protein and carbohydrate foods is generally regarded as beneficial. But, most important of all, a diet containing a minimum of purin bodies should be chosen, since an attack of gout may be precipitated by such food. The foods which contain no purins are milk, eggs, butter, cheese, white bread, rice, sago, fruits, cabbage, lettuce, cauliflower. Potatoes contain 0·02 gramme per kilogramme, peas 0·4, oatmeal 0·5, beans 0·6, asparagus 0·2, codfish 0·5, plaice 0·7, salmon 1·1, halibut 1, chicken 1·3, mutton 0·96, veal 1·10, pork 1·20, ham 1·10, beef 1·10 to 2, liver 2·7, sweetbread 10. Thus white fish and mutton are the most suitable among fish and red meats, and liver and sweetbread are highly unsuitable. Potatoes should be taken in moderation, on account of the large amount of starch they contain. Personal peculiarities may have to be consulted, so that indigestion is avoided, and with this proviso fat may be taken in moderation. Alcohol in any form is best avoided by those with a tendency to gout; but wines are free from purin bodies, and, on the other hand, beer, tea and coffee contain small amounts, the last two as methyl purins (caffein, thein). A light claret or light dry sherry is the least harmful, or a small quantity of good brandy or whisky well diluted. The stronger sherries, port, burgundy, and champagnes must not be taken. As in diabetes, saccharin may be usefully employed to sweeten beverages such as cocoa or coffee. The advantage of exercise in promoting digestion and assimilation is sufficiently well known. It may be supplemented by the morning bath with the use of the flesh brush or by the occasional use of the Turkish bath with shampooing, or more scientific massage. Alkaline and saline waters are of service, and more still residence at the spas, where the influence of the waters is combined with a regulated diet, fresh air, and pleasant surroundings.

Atophan, described above, is the most valuable drug for preventing attacks of gout. It is given in 15-grain doses three times a day for three days of each week. Salicylates and sodium benzoate act similarly in increasing the excretion of uric acid in the urine and so preventing a harmful accumulation in the blood and tissues. The bowels should also be kept free.

**Treatment.**—In acute gout the affected foot should be kept raised, and supported on a chair or couch, if the patient is not in bed. It should be wrapped in cotton wool, and in severe cases some anodyne application, such as belladonna liniment, tincture of aconite, tincture of opium, or a lotion of atropine and morphia, may be previously applied sprinkled on lint, and some oiled silk or thin gutta-percha laid over all. Leeches, hot poultices, and ice applications are to be avoided. The diet must be at once restricted in younger patients to milk and farinaceous foods, but in older persons and those broken down by previous attacks it may be more liberal, but still mostly fluid and easily digestible. Alcohol should, if possible, be entirely withheld. Medicinally colchicum has a decided effect in most cases. It may be given as wine or tincture in doses of 15 to 25 minims every six or four hours, in combination with bicarbonate or citrate of potassium, or citrate of lithium. Atophan is useless in relieving the inflamed joint. The bowels should be kept active with calomel. If pain is very severe, morphia may be given by subcutaneous injection or internally.

## INFECTIVE ARTHRITIS

It will have been observed that in several of the infectious diseases arthritis has been mentioned as a complication; the most common case of a multiple arthritis,

namely, rheumatic fever, is now recognised as an infective disease; and it is probable that rheumatoid arthritis and osteo-arthritis are in many cases also due to microbic invasion. In the following diseases a multiple arthritis may occur as complication or sequela: typhus, scarlatina, small-pox, typhoid, influenza, dengue, pneumonia (pneumococcal arthritis), streptococcal septicæmia, pyæmia, gonorrhœa, bacillary dysentery, syphilis, both congenital and acquired, tubercle, erysipelas, erythema multiforme, and erythema nodosum. Whether these are due in every instance to the specific organism of the disease, or to a secondary infection, is not always clear.

Infective arthritis may be a simple synovitis, which soon recovers; or it may result in fibrous ankylosis or in suppuration. In some diseases, such as rheumatic fever and scarlatinal synovitis, recovery is the rule; in others, like pyæmia, septicæmia, and pneumococcal arthritis, suppuration is frequent or invariable; in others, again, like typhoid fever, influenza, and erysipelas, every one of the three results is sometimes seen.

**Diagnosis.**—It is here only necessary to point out in what a large number of instances a polyarthritis due to other infections has been called "rheumatic," or diagnosed as acute rheumatism.

The symptoms are pain and tenderness, with or without swelling of the joints, and it may be only in the history and associations of this arthritis that means of recognising its origin can be found. The fact that salicylates give relief to the pain and lower the temperature is not evidence that the disease is specifically rheumatic fever.

**Treatment.**—The milder forms subside with rest, severer forms require splints and fixation, and where suppuration is recognised the joint should be opened and drained. Passive movements should be carried out as soon as the acute stage of the inflammation has subsided, so as to prevent fixation.

## RHEUMATOID ARTHRITIS AND OSTEO-ARTHRITIS

Under these names will be described the disease which has at different times been called *rheumatic gout*, *chronic rheumatic arthritis*, *chronic rheumatism*, *arthritis deformans*, and *nodose rheumatism*.

**Ætiology.**—Many believe that two different diseases are included under the terms *rheumatoid arthritis* and *osteo-arthritis*, and that the former is an inflammation resulting from infection and that the latter is a degeneration. Certainly these names are given to conditions that clinically are very unlike one another: but there are intermediate forms, so that in a classification it is impossible to say where one ends and the other begins. These differences may not be due so much to the exact cause of the disease in individual cases as to other factors, such as the age of the patient, the nature of his tissues and the strains to which they are subjected (Strangeways).

At present it is believed by many in this country that this disease is inflammatory, being the result of infection from some primary focus elsewhere in the body, though there is a difference of opinion as to whether the joint changes are caused by toxins circulating in the blood, or whether the living virus itself gains access to the joint. Cultivations from the synovial fluid are usually sterile, but it is argued that the micro-organisms may be tucked away in the proliferating tissues at the sides of the joint. In most cases the primary foci are in the teeth, consisting of apical abscesses or pyorrhœa alveolaris (*see* p. 392). Less commonly the tonsils are at fault. Other possible causes are adenoids, suppuration in the nasal sinuses or middle ear, uterus, male urethra, urinary tract, open wounds, and the colon, the latter if there is derangement of function. Most cases are probably of streptococcal origin, but the *pneumococcus* and *gonococcus* may also be concerned, and perhaps others, such as *B. coli*. There is also a tuberculous form, described by Poncet. Others believe that the infective or toxic origin has

been exaggerated and that in many cases it is caused by some biochemical change or endocrine disturbance. Certain it is that such factors may produce alterations in the joints, *e.g.* the spinal changes in hunger osteomalacia (*see* p. 695). The fact that the sugar tolerance is diminished in rheumatoid arthritis also seems to point in the same direction (**4**). The subjects of hæmophilia also are liable to frequent attacks of synovitis, which are probably due to effusions of blood and are not infective; and these may be caused by the slightest injury. Articular pains also occur as parts of so-called "serum disease," when antitoxic serum is used as treatment. Arthritis also occurs in pulmonary osteo-arthropathy. The multiple arthritis of certain nervous diseases, locomotor ataxy and syringomyelia, may be inflammatory, but it has special features (*see* later).

This disease occurs at all ages, and in all conditions of life; but it is more common after forty years of age than before, in women than in men, and in the poorer classes than in the wealthy. Cold and damp may dispose to its occurrence or excite fresh attacks. Traumatism in the form of continued pressures, or constant strain, also predisposes to the disease, especially in the cases in which one joint is chiefly affected.

**Morbid Anatomy.**—In the early stages there may be effusion into the joint, the synovial membrane is vascular and thickened, and the articular cartilage undergoes softening. This softening begins with proliferation of the cartilage cells, and the matrix splits into fibres perpendicular to the articular surface; then the cartilage cells burst into the joint, and leave a soft, ulcerated surface. By a continuation of this process the cartilage is completely destroyed, the bony surfaces come into contact, and the effusion is absorbed. The adjacent surfaces, from continued pressure, acquire a dense, hard white surface (*eburnation*), owing to increased bone formation, and may be more or less grooved or fluted on the surface; further, what was formerly a rounded or convex surface becomes flattened down. At the same time bony outgrowths may appear at the edge of the articular surface and form separate projections (*osteophytes*) or unite to form a kind of ridge or "lip" around the joint, or in some cases (*e.g.* in the knee) may contribute to produce large plates of calcareous matter, which surround the joint like plates of armour. The synovial membrane also forms fringes or villous outgrowths. These may break away and form *loose bodies* in the joint; some of them contain cartilage (**3**).

The term *rheumatoid arthritis* is often used for the more acute cases, where there is chiefly synovial effusion and thickening with formation of fringes and villi and some erosion of cartilage. The term *osteo-arthritis* applies to the more chronic type of case, where there is not much fluid, but where the cartilage has disappeared, so that the bony surfaces articulate on one another. This has been regarded as a degeneration rather than an inflammation. But all these types of change may sometimes be seen in different parts of the same joint. Limitation of movement arises from muscular spasm and atrophy, from fibrosis of the capsule, from alteration in shape of the articular surfaces, or from fibrous, cartilaginous or bony ankylosis. Although often described, there are no peri-articular inflammatory changes round the affected joints (Strangeways).

In the more acute forms the ends of the bones and even the shafts are seen by the Röntgen-rays to be more transparent than in health, owing to absorption of bone. This atrophy is probably the effect of both inflammation and disuse. At the edge of the articular cartilage there may be proliferation, as shown by the bony surface being ill-defined. In old-standing cases exostoses, osteophytes, or "lipping" are seen on the edge of the bone.

The changes in the arthropathies of tabes and syringomyelia (Charcot's joints, *see* p. 791) are similar to those just described; but there is much greater destruction of tissue, with the formation of a large joint cavity filled with fluid. Infection may be the primary cause, but trophic influences may play a part. An even more probable explanation is that the great destruction is due to over-use of the

inflamed joint, because the patient has lost the sense of pain which would normally make him keep it at rest.

**Symptoms.**—These will, of course, vary with the tissue primarily affected, with the acuteness of the onset and the number of joints involved.

In a minority of cases, and these chiefly in young persons, the disease is acute, polyarticular, and largely synovial. This has been called acute rheumatoid arthritis. It occurs especially in young women, begins with acute fusiform swelling of the proximal interphalangeal joints, with pain and tenderness; and subsequently the metacarpo-phalangeal joints, wrists, elbows, shoulders, and joints of the lower extremities are involved, as well as those of the spine and jaw. There is febrile reaction with quick pulse for some weeks; and when this subsides the joints are still limited in their movements, and relapses of fever and swelling of the joints may occur. At the same time the patient becomes pale, sweats profusely, and shows pigmentation. There is depression of spirits and loss of appetite. With each relapse the movements become more restricted, and, finally, ankylosis may result. In most cases there is no cardiac complication, but endocardial murmurs are occasionally heard.

In older persons the disease may occur with a somewhat rapid onset and affect many joints. Those of the hands and fingers are swollen, painful, tender on manipulation, and stiff on movement. The skin is shiny and slightly reddened. These symptoms subside and recur from time to time, each recurrence leaving more change behind it; or the disease may be more chronic from the commencement. The stiffness is often most marked in the morning, so that movements are at first painful; yet if they be persevered in, the pain will gradually wear off. Similar symptoms may be noted, in different cases, in other joints, such as the wrist, elbow, and shoulder, the hip, knee, ankle, and foot. The sterno-clavicular articulation, the joint of the jaw, and the vertebral joints may be also involved. Sometimes effusion can be readily recognised, and the joint is tense and elastic. In other joints, or in later stages, creaking or grating can be elicited on passive movement. As time goes on movements become more and more limited. These cases are often described as osteo-arthritis. Usually no constitutional disturbances peculiar to the disease are noticed in these cases, though the other diseases of elderly people are frequently present.

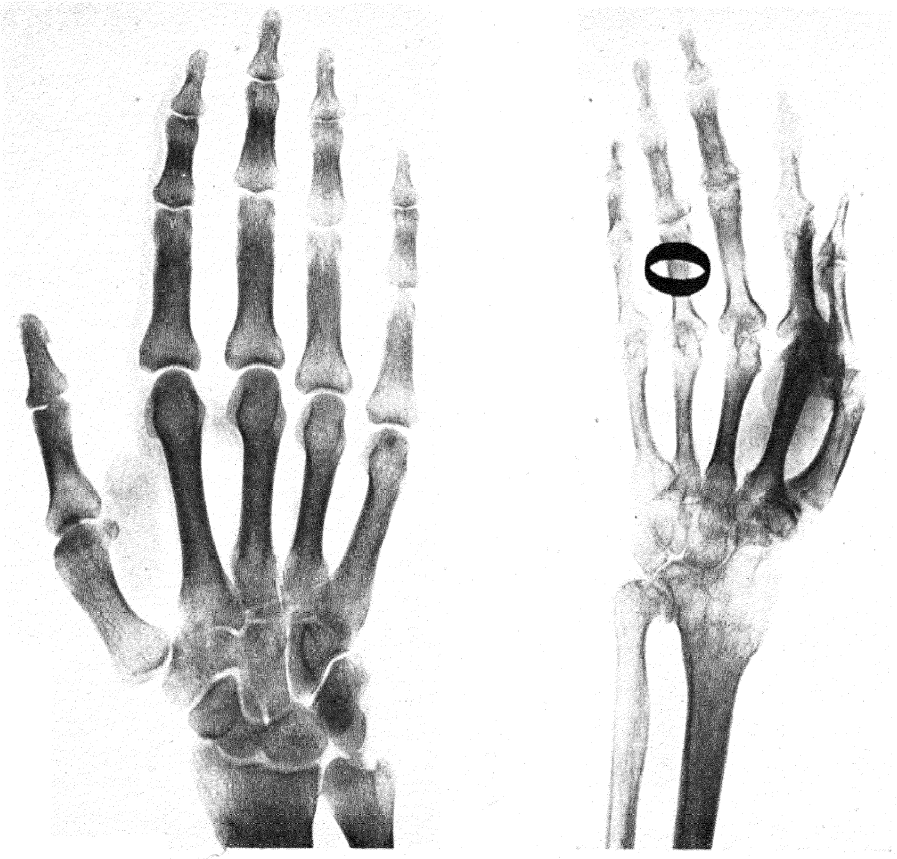
The disease is often liable to attack one joint and to cause very extensive changes with destruction of cartilage, and many osteophytes (monoarticular osteo-arthritis). In the hip joint the pain and stiffness are followed by very limited movement, eversion and apparent shortening of the limb, and flattening of the buttock. The knee and the joint of the jaw are others that may be involved alone.

Very characteristic deformities take place in consequence of the joint changes and the muscular atrophy which is associated with them. Thus the fingers, instead of remaining in line with the metacarpal bones, deviate to the ulnar side, and the joint at the base of the index finger is often greatly swollen; the metacarpo-phalangeal joints are commonly flexed, the first phalangeal joints are over-extended, and the second are flexed. But other forms occur in which these joints are differently placed, and these varieties probably depend on the extent to which the interosseous and other muscles are affected. The lower ends of the radius and ulna project at the back of the wrist. The muscles commonly observed to be atrophied are the interossei in the hand, the muscles at the lower end of the femur, and the deltoid over the shoulder joint (*see* p. 914). The nerves also in connection with the joints are affected by chronic neuritis, causing pains in the limbs.

*Heberden's nodes*, the small nodules or knobs which may form at the sides of the terminal phalanges, are bony outgrowths, and are characteristic of the early stage of the chronic disease (osteo-arthritis).

*Spondylitis Deformans.*—This is essentially a bony ankylosis of the vertebræ

PLATE XXXV.



1. 2.

FIG. 1.—Radiogram of a normal hand of a man, aged forty-one.

FIG. 2.—Radiogram of the hand of a woman suffering from advanced rheumatoid arthritis. The heads of the metacarpal bones show marked atrophy since the rounded ends have disappeared, and osteophytes are present. The radius and carpus are ankylosed. The thumb is bent inwards, so that the thenar eminence is seen behind the metacarpal of the fore finger.

[*To face p.* 671.

occurring in older patients, often associated with osteo-arthritic changes in other joints, and probably due to the same cause. The back is stiff, and there is often kyphosis. Neuralgic pains, probably due to pressure on the spinal nerves, may be present, and sometimes the ribs are immovable, so that breathing is entirely diaphragmatic.

In children a multiple arthritis, beginning before the second dentition and observed in the knees and wrists, is often associated with enlargement of the lymphatic glands and spleen, anæmia, sweating, varying pyrexia, and arrest of the bodily development (Still's disease).

There is considerable variability in the progress of the local symptoms in rheumatoid and osteo-arthritis; they may subside for long periods, even when untreated, and then burst out again.

**Diagnosis.**—In chronic *gout* the articular changes may closely resemble those of rheumatoid arthritis. They have to be distinguished by the history of the first acute attacks, and the presence of urate deposits (tophi) in the ear, and in the affected joints; if necessary, the uric acid in the blood may be determined. In the acute cases other forms of acute or subacute polyarthritis must not be forgotten, such as congenital syphilitic arthritis and gonococcal arthritis; the localisation is often different, and the history will help. Rarely, repeated attacks of acute rheumatism produce permanent changes in the joints. Arthritis affecting one joint only may be confounded with tuberculosis. Disease of the *hip* in elderly people must be distinguished from sciatica and lumbago. This is not easy in the early stage of the disease. Reliance should be placed on (1) local pain and tenderness on pressure over the joint, and pain on movement; (2) referred pain simultaneously along the sciatic and anterior crural and obturator nerves, since these nerves all supply branches to the joint: (3) limitation of movement. The joint changes accompanying *tabes dorsalis* and *syringomyelia* generally begin with an abundant painless effusion into the joints, which are greatly enlarged, and there is subsequently great mobility, with destructive changes. They may be distinguished by these features or by other evidence of tabes dorsalis (Argyll-Robertson pupil and loss of knee jerk) or of syringomyelia (dissociated sensation).

Radiograms give valuable information with regard to ankylosis, the presence of osteophytes, and the deficiency of calcium, as shown by increased translucency of the shadow of the bones.

**Prognosis.**—No cure is possible when permanent changes in the joints have occurred, but in the very early stages a cure is possible. However, even in the later stages of the disease very great improvement takes place with careful treatment. In the acute form it is remarkable how altered in outward appearance the joint may be, and yet there may be complete recovery; but it takes a long time.

**Prevention.**—Care must be taken to prevent the occurrence of septic foci. In particular the teeth must be attended to with regularity.

**Treatment.**—The first step is to see if there is a possible primary focus. It is impossible to exclude the teeth and gums as sources of infection after a superficial examination; the required information may be obtained by taking dental radiograms (*see* p. 392). Suppuration at the root may exist when the tooth looks perfectly healthy, does not cause pain, and is not tender on percussion. The affected tooth should be extracted, and cultivations made from the apical abscess so as to prepare an autogenous vaccine. Apical abscesses are sometimes sterile. In severe pyorrhœa alveolaris the teeth should be extracted, but in milder cases improvement often takes place with less radical treatment. When the discharge of pus is free the danger is not so great as when it is pent up behind obstruction. Tonsils, adenoids, and the other possible sources of infection already mentioned should be attended to, and vaccines may be used. If no foci are found, suspicion often falls on the alimentary canal, particularly the

colon, and vaccines prepared from organisms, usually *streptococci*, found in the fæces, have often been used. The test of a vaccine is its power to produce a focal reaction in the joints, and it should be continued for a year or longer until an injection produces no more pain in the joints (Beddard). Intestinal antiseptics, such as salol, bismuth salicylate and β-naphthol, have been used, especially if there is diarrhœa. Another line of treatment is the so-called "protein shock therapy," in which intravenous injections of some foreign protein are used, for instance 50 to 100 millions of a coli or typhoid vaccine or 10 c.c. of a 10 per cent. solution of Witte's peptone in water. The joints become swollen and painful, and subsequently there is improvement. This method may be used if no primary focus can be found. Intra-muscular injections of sulphur are also given.

Apart from specific treatment directed towards the cure of the disease, there are certain general measures and local measures of a palliative kind that must be considered. The diet should be carefully ordered so as to avoid indigestible articles, but should not be stinted; indeed, the patients require, as a rule, good feeding, and meat may be given freely, as well as vegetables. A moderate use of alcoholic drinks may be allowed. The patient should be well clothed in flannel, a warm dry atmosphere should be looked for, and changes of temperature avoided. Various health resorts and spas fulfilling these requirements have been found beneficial, such as Buxton, Bath, Harrogate, and Strathpeffer, at home, and Aix-les-Bains, Aix-la-Chapelle, Baden-Baden, and Wiesbaden, abroad. Internally arsenic in full doses, aspirin, sodium salicylate, guaiacol, iodide of iron, and cod-liver oil are most valuable, and they must be continued, with such intermissions as may be desirable, for weeks or months. Thyroid extract has given good results in some cases. The joints themselves should be kept at rest in splints during the active stage of the disease; tincture of iodine may be applied.

After pain has subsided massage and passive movements will help to restore the mobility of the joints and the muscular nutrition; it is a good plan to apply heat to the joint before massage is carried out, as the mobility is increased by such means. Peat baths, mud baths, electric light baths, hot air baths and hot water baths, including the whirlpool bath, may be used. Electricity may be given in the form of diathermy, the continuous galvanic current, high frequency, and ionisation with salicylic ions. Sometimes it is found impossible to get a freely movable joint, and then fibrous ankylosis in the most useful position should be aimed at. In the case of the knee joint bony ankylosis is preferable, and in certain cases the joint has actually been excised in order to bring this about.

### INTERMITTENT HYDRARTHROSIS

This is a rare disease, in which at periodical intervals an effusion takes place into the joints. The knees are most commonly affected and the length of the cycle is usually rather less than a fortnight. It may continue for months or years; but the attacks have been known to cease during pregnancy. It is remarkable how constant the cycle is in any individual case. The cause of the conditions is obscure, but it may be a manifestation of angeio-neurotic œdema, and it is to be noted that the circumscribed œdematous swellings of the latter disease have sometimes been found to recur at regular intervals. If the two are related, then intermittent hydrarthrosis must be regarded as a *toxic idiopathy*, and treated accordingly (*see* p. 224).

## HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

"Clubbing" of the fingers is the earliest stage of this disease. The terminal digit is swollen and globular; the nail is enlarged and more convex than usual.

The soft parts may be involved alone, but the phalanx is often broad and thick. In fully developed hypertrophic pulmonary osteo-arthropathy the clubbing of the fingers is associated with enlargement of the bones of the extremities, particularly those of the hands and feet, and the distal ends of the long bones. There may be effusion into the joints, preceded by stiffness.

"Clubbing" occurs in two groups of cases: (1) Among people who live at great altitudes, *i.e.* the Andes (**6**), and in other cases of prolonged oxygen deficiency, such as chronic pulmonary disease, *e.g.* fibroid lung, phthisis empyema and bronchiectasis, but also in mediastinal new growths, in compression of the subclavian vein by aneurysm, when unilateral clubbing has been found, in methæmoglobinæmia or sulphæmoglobinæmia, and in congenital heart disease (*see* p. 353), and, rarely, acquired heart disease. (2) In this group are included a varied assortment of cases, cirrhosis of the liver (*see* p. 487), infective endocarditis, carcinoma of œsophagus, pyloric stenosis, etc. Clubbing has disappeared with relief of the primary cause. Pathological and skiagraphic observations show that in developed pulmonary osteo-arthropathy there is a thin deposit of new bone under the periosteum of the long bones, and especially of the lower thirds of the radius, ulna, tibia, fibula, metacarpals, metatarsals, and proximal and middle phalanges of the hands and feet. The deposit is such as to fill up the concavity between the proximal and distal ends of the bone, and to make, for instance, in the case of a metacarpal, the middle thicker than the ends. Sometimes the joints (wrists, ankles and interphalangeal joints) contain fluid, and the synovial membrane is thickened. In clubbing of the fingers and toes the primary change is œdema of the tissue between the nail and the phalanx, which separates the connective tissue fibrils from one another (**5**). In later stages this is replaced by fat and fibrous tissue. In early cases, after compressing the sides of the fingers for some little time, a boggy feeling is experienced on compressing the base of the nail, as if it was lying on thick fluid. Two factors probably play a part in the production of the œdema—desaturation of the arterial blood with oxygen and peripheral stasis. The clubbing in the second group of cases can hardly be explained in this way.

## OSTEOMALACIA

(*Mollities Ossium*)

In osteomalacia there are degenerative changes in the bone and bone marrow, so that the bone becomes soft and fragile.

**Ætiology.**—It is a disease of adults, beginning mostly between the ages of thirty and forty; and it affects women almost exclusively. Little more is known of its ætiology, except that it often begins during pregnancy, and is aggravated by it, and that it has been much more often observed in some parts of the world (along the Rhine, in Westphalia, Switzerland near Bâle, North Italy, and Calabria) than in others. Hunger osteomalacia is described elsewhere (*see* p. 695).

**Morbid Anatomy.**—The bones are found to be so soft that they can be cut with a knife or indented with the finger. Nearly the whole of the bone is converted into a soft greasy mass or pulpy material, except perhaps a thin shell of compact tissue just under the periosteum, or even there may be nothing left but the thickened periosteum itself. Microscopically the change seems to be, first, a confusion of the natural minute structure of the bone; the Haversian systems become fused together, and then absorption of the salts takes place from the innermost rings round a canal, so that the substance is gelatinous and transparent. As the same change proceeds in the rings placed farther out, the innermost rings are entirely absorbed, the lacunæ also alter their shape and size, and finally the Haversian systems are destroyed. The bone becomes spongy

and porous (*osteoporosis*), and the enlarged medullary spaces are filled with a marrow which is fatty or lymphoid, with giant cells and osteoblasts. In this there are recent hæmorrhages and pigment resulting from former effusions of blood. In some places the medulla may be gelatinous or watery. Chemical analysis of the bones shows that the inorganic constituents are reduced from 68 to 30 per cent.

**Pathology.**—The disease is probably due to disturbed internal secretions. The disease occurs during pregnancy, and is aggravated with each succeeding pregnancy, and it has been arrested by the removal of the ovaries. Thus these organs seem to be implicated; but this theory will not explain infantile, or senile, or male cases. The thyroid and suprarenal bodies are also thought by some to be concerned, perhaps acting through their connection with the ovaries, at least in females.

**Symptoms.**—The first is pain, which is felt in the bones of the lower extremities, the pelvis, spine, ribs, face, and not so often in the upper extremities. The bones are tender on pressure, and there is great pain on movement. Consequently there is a characteristic stiff and waddling gait, and the patient uses his arms as much as possible to help him with his movements. He is languid and is disinclined to do anything. He may become bedridden. The joints are not painful. In course of time a change of figure may be noticed; the patient loses height from rounding of the spine or bending of the limbs: then, some day or other, a bone breaks with very little apparent cause, or on slight exertion. As a rule, the fracture heals but imperfectly, and subsequently other bones break. Moreover, the long bones show a remarkable degree of flexibility, so that they can be bent into very strange positions, and the more superficial bones may be indented with the finger. The thorax becomes deformed from the softness of the ribs, and dyspnœa may occur in consequence. In rare cases the bones of the skull may be softened.

With this there may be fibrillary twitchings and cramps, and the occurrence of definite tetany has often been described. Though the general health may be preserved at first, exhaustion at length supervenes.

The disease runs a chronic course, and generally lasts from five to ten years. Death takes place often from inability to breathe on account of the softness of the ribs or from broncho-pneumonia favoured by the deformity, sometimes during parturition from the obstruction which the distorted (rostrate) pelvis offers to the passage of the fœtus.

The **Diagnosis** is not difficult when the changes in the bones have become manifest. Earlier symptoms may be mistaken for *rheumatism*. Spontaneous fracture also may take place in *sarcoma, carcinoma*, and *multiple myeloma* of the bones.

**Treatment.**—Removal of the ovaries has been advocated, and several times performed: the existing deformities remain, but further progress is checked. Improvement has also occurred after the subcutaneous injection of adrenalin ($\frac{1}{2}$ to 2 c.c. of 1 in 1,000 solution twice daily). Otherwise recovery is exceedingly rare. The treatment should be tonic, as by quinine, iron, and especially cod-liver oil. Foods containing lime and phosphorus should be given, such as milk, eggs, fish, fresh meat, peas, beans and cereals; and phosphorus is said to be of value in doses of $\frac{1}{20}$ to $\frac{1}{12}$ grain daily, either as oleum phosphoratum or pil. phosphori (B.P.), continued for one or two months.

## ACHONDROPLASIA

This is a congenital disease of the bones and cartilages, which results in permanent stunting of growth and other deformities. It is probably due to some *hormone* defect. The thyroid is normal. Some cases are hereditary, and the disease has been known from very early times. It has no doubt been often mistaken for

rickets, which, indeed, may sometimes complicate it. At birth the limbs, especially in their upper halves, are noticed to be abnormally short. The child may be of full weight, but growth is slow, the limbs continue to be short, and the stature is consequently small, though the vertebral column is of normal length. There is a projection of the buttocks which gives the appearance of lordosis and a waddling gait. The head is generally large, somewhat like a rachitic head, with a prominent forehead, but a depressed bridge of the nose. The limbs often show transverse furrows, as if the soft parts were too long for the bones. The hands present a characteristic deformity in that the index and middle fingers diverge from the ring and little fingers when the hand is open (*trident hand*). The deformities are due to defects in ossification of the cartilages of the long bones and of the innominate bones, together with a premature synostosis of the bones of the basis cranii. The cranial bones which are formed in membrane are normally developed, as well as the bones which remain cartilaginous till a late period of fœtal life, viz., the sternum, patella, carpal and tarsal bones, and the costal cartilages. The thyroid is normal; and the subjects of the disease have a good intelligence, and are muscularly and sexually well developed. The disease does not usually tend to shorten life.

The changes occurring in the cartilages are of three types: (1) hypoplastic, with diminution of the cells; (2) hyperplastic, with excessive proliferation and enlargement of epiphyses; (3) softening, with defective columnar arrangement of the cells and increased vascularity.

No treatment is of any avail.

## OSTEOGENESIS IMPERFECTA

As the name implies, there is in this rare condition deficient ossification of the bones, with consequent undue fragility, especially shown in the long bones; and there is an abnormal formation of the skull, which is typical of the disease (Cameron). The shafts of the long bones are curved and tortuous, and there is some shortening, which is more marked in the proximal halves of the limbs. Fractures, relatively painless, take place with great facility both in the ribs and in the limbs; the bones may unite again under a callus which is thrown out in a circular form. In the skull there is a persistent wide frontal suture, continuous with a large anterior fontanelle; and the posterior fontanelle remains also large. The bones are thin and yield to pressure, and instead of forming in each case a single bone, they present a mosaic of small irregular separate pieces of bone. In the lateral region the skull may be still unossified, and this leads to a characteristic bulging in the temporal region just above the ear, the concha of which is pushed outwards by it. The orbits are oval with the long axis vertical, and during life the eyeballs are turned downwards so as to produce some resemblance to the condition seen in chronic hydrocephalus. Other points of interest in some of these cases are the blue colour of the sclerotics, the existence of continued pyrexia, and the occurrence of more than one case in a family.

## OSTEITIS DEFORMANS

(*Paget's Disease.*)

The ætiology of this disease is unknown. Perhaps the most likely suggestion is that it is due to some defect of internal secretion; but syphilis has been considered a cause. It affects both sexes about equally, and is observed in the latter half of life.

**Morbid Anatomy.**—The long bones of the limbs are often attacked in the early stages of the disease. They become thicker. There is an increase in the size of the marrow cavity. At the same time the compact bone becomes looser in texture

and more spongy and much thicker than normal. The consequence is that the bones are light, even though they are large. The bones of the lower limb bend under the weight of the body. Similar thickening and porosity are noticed in the bones of the skull. There is kyphosis of the spine. The bones of the face, hands, feet and pelvis and the clavicles and ribs may show similar changes in slight degree. Arterio-sclerosis and myocardial degeneration are often present.

**Symptoms.**—The onset is insidious. The patient complains of pain and tenderness in the affected parts. His legs may become gradually bowed and his back bent. The increase in the size of the skull is noticed because he finds that he must wear a larger hat. Often the disease begins in one limb, and the corresponding limb on the opposite side is affected subsequently. In the later stages the patient's attitude is characteristic, with bent back, bowed legs, and large head projecting forwards. He can only waddle along with the help of a stick. The disease is slowly progressive, but does not shorten life, except that in a few cases sarcoma has developed in the affected bones. The bones are not liable to fracture.

**Diagnosis.**—The disease must be distinguished from *acromegaly*, in which the soft parts are affected as well as the bones; *osteo-arthritis*, in which the joints are affected, but not the shafts of the bones; *osteomalacia*, in which the bones are soft and liable to fracture; and *syphilis*, in which there is no general enlargement of the skull. The history of the disease and the Wassermann reaction will also help.

**Treatment.**—This is symptomatic. If the Wassermann reaction is positive, anti-syphilitic measures should be adopted.

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# SOME CHRONIC INTOXICATIONS

## ALCOHOLISM

UNDER this term are here described the results of the more or less continued use of alcohol, not the immediate effects of a considerable overdose, known as drunkenness or intoxication. The symptoms of the latter are familiar, and usually subside as the alcohol passes through the system. Occasionally, however, from the rapid imbibition of large quantities of spirit in those unaccustomed to it, a fatal result may ensue. It is preceded by unconsciousness, with pupils sluggish or fixed, and generally dilated; small pulse, cold clammy skin, stertorous respiration; and sometimes delirium or convulsions. Very low internal temperatures have been recorded, as if the patient had been lying exposed out of doors (*see* p. 23).

### DELIRIUM TREMENS

This commonly occurs in those who habitually drink freely, who may have been not infrequently drunk, and who have recently been taking unusual quantities continuously for some days. The symptoms may also be brought on by sudden deprivation of alcohol in persons who from long-continued excess have acquired unusual tolerance. Delirium tremens is also sometimes determined in habitual drinkers by an injury, such as the fracture of a bone, or by the onset of pneumonia, erysipelas, or other acute disease, or by some mental shock, without evidence of any amount of drinking beyond the daily average.

**Symptoms.**—The first symptoms are disturbed sleep, restlessness, irritability, and loss of appetite; and with the disturbed sleep there are unpleasant dreams and some wandering. In the morning the patient may be more rational, but the delirium returns at night; the next day the delirium continues, and shows its characteristic features. The patient is constantly talking, addressing either those about him or imaginary persons whom he supposes to be present. He talks on his business, or on other affairs, frequently changing from one subject to another. He may be recalled to his senses for a moment, but soon relapses. In the pursuance of his thoughts he may try to get out of bed, or pull the bedclothes about. With this there is very marked tremor; the hands shake with quick oscillation whenever he moves them; the lips and tongue tremble when he speaks. As the condition gets worse he has very definite hallucinations and illusions. Objects in the room or figures on the wall-paper are conceived to be animals or insects; he is constantly seeing cats, dogs, rats, mice, or black-beetles running after him or crawling about the bed; he looks under the bed, or behind the curtains, or peers behind any bystander, and is suspicious of injury from those about him. He may, under some such false impression, strike those who are near; but, as a rule, he can, with a little firmness, be controlled, and it is often easy to turn his thoughts for a moment in another direction.

Other symptoms accompany this mental and muscular disturbance. The face is flushed, the conjunctivæ are suffused, the tongue is thickly furred and becomes dry, as the disease progresses. The pulse is quick, soft, at first full, afterwards small and feeble; the temperature rises to 102° or 103°, and there is occasionally hyperpyrexia (106° or more). The skin is generally moist or even profusely perspiring; and the urine is accordingly dark-coloured, scanty, and of high

specific gravity. It may contain a small quantity of albumin, and albumoses are present in a small proportion of cases. According to Gowers, there may be indications of early optic neuritis

About the end of the third day, with considerable constancy in favourable cases, some improvement begins to show itself. Hitherto sleep has been entirely absent, but now the patient falls into a slumber which may last eight or ten hours, and he wakes much refreshed, the delirium and trembling are less, and gradually improvement takes place in all respects. In more serious cases the face loses its colour and becomes pale and earthy, the pulse is quick and feeble, the delirium is less active, and the patient more prostrate; he lies on his back muttering and semi-comatose. Finally death takes place, preceded, it may be, by convulsions or hyperpyrexia. In some instances the symptoms are comparatively mild, and subside in a day or two; in others the long-desired sleep is not directly curative, but is repeated more than once before recovery is ensured.

Death occurs from exhaustion; from cardiac failure, and this is sometimes sudden; or from pneumonia.

**Morbid Anatomy.**—There is some congestion of the cortex of the brain; and minute hæmorrhages may be present in the cortex of the central and frontal convolutions, and also, though less, in the cerebellum and spinal cord. Thickening with opacity of the membranes of the brain is a chronic condition not responsible for the acute symptoms.

**Diagnosis.**—It may be simulated by *meningitis* and *general paralysis of the insane*. The former is generally distinguished by early convulsions, or later on by paralysis, sometimes, but not always, by headache, or by the longer duration of the symptoms. In the latter a carefully studied history ought to help to a right conclusion. In any case of delirium tremens it is possible that pneumonia or fever may be present.

**Treatment.**—The patient should be kept as quiet as possible in a darkened room undisturbed by visitors. If he is restless and gets out of bed, male attendants are desirable who may with a little management keep him under control. Extreme violence may require a strait-waistcoat, or a sheet stretched over the trunk, legs and arms, and fixed to the sides of the bed, or broad padded leather bands, by which the shoulders, wrists, and ankles can be fixed down to the bedstead, thus leaving the chest free from restraint. These methods should, however, if possible, be avoided. The likelihood of the patient doing injury by getting at knives or forks, by drinking lotions or liniments, or by jumping out of the window must be borne in mind. Food must be given every two or three hours in small quantities; it may be milk and beef-tea, and it is better if it can be peptonised by the use of liquor pancreaticus or similar preparations. The use of drugs has to be approached with great caution. Hyoscine hydrobromide would seem to be the most satisfactory narcotic, and it is stated that it can be given in doses of $\frac{1}{33}$ grain without harmful effect. Sulphonal in 30-grain doses is also advised. Stimulants are better avoided; but they may become absolutely necessary in the stage of prostration, when ammonia, ether, or alcohol may be given.

## CHRONIC ALCOHOLISM

The effects of chronic alcoholism are seen most markedly in various nervous symptoms, in impaired digestion, and in cirrhosis of the liver; it no doubt also contributes to fatty and to atheromatous degeneration of the arteries, and to diseases of the kidneys. These different parts of the body may be affected separately or together; but, as a fact, the term *chronic alcoholism* is more commonly applied to those cases in which the liver alone, or the kidneys alone, are the seat of disease. In all cases it is important to observe that it is not drunkenness or intoxication which leads to this condition, but the constant imbibition of considerable quantities of beer, wine, or spirits, possibly without

the individual getting drunk on any single occasion. The daily amount of alcohol and the duration of the drinking habits required to produce this effect in different persons are very variable.

**Morbid Anatomy.**—In chronic alcoholism the membranes of the brain are commonly opaque and thickened, and the convolutions are shrunken and atrophied. The changes found in peripheral neuritis, hepatic cirrhosis, etc., are described elsewhere.

**Symptoms.**—Muscular tremor is one of the first indications of the nervous system feeling the effects of the poison. The hands are unsteady, and the tongue trembles when it is protruded. The patient is restless and irritable, he sleeps badly, and wakes unrefreshed, with a feeling of prostration that tempts him to take stimulants at once. Sinking feelings are frequently complained of, which require, according to the patient, to be met by fresh doses of the favourite drink. As things get worse the patient is unequal to any sustained mental effort; even the simplest business transaction must be preceded by a glass. Sensory disturbances may be also present, such as buzzing or rushing in the head, vertigo, muscæ volitantes, flashes of light, or diffused headache. There may be severe neuralgic pains in the legs, possibly the early signs of neuritis. In later stages the mind is seriously involved. Judgment, intellectual capacity, volition, and the moral sense are all weakened. The patient becomes hesitating and vacillating, unable to follow out any definite line of action, but unscrupulous in his attempts to get stimulants at all times. Multiple neuritis (*see* p. 741), epileptic fits, irregular paralysis or anæsthesia, and some forms of insanity, which may be mania, melancholia, or dementia, also occasionally result from continued excess (*see* Korsakow's Disease, p. 741).

With the occurrence of the first nervous symptoms the functions of the stomach are often disturbed. The patient vomits in the morning directly he rises from bed, he is quite unable to eat any breakfast, and his appetite generally is deficient. The tongue is covered with thick yellowish-white fur, and the breath is fœtid. The eyes are suffused, and the face may be tinged with yellow.

In course of time the minute venules of the cheeks become dilated, the nose is red and thickened, and sometimes rosacea develops. The face becomes more and more bloated, and the blotching with dilated venules is more marked. In this stage, or a little earlier, the liver may be found to project 2 or 3 inches below the costal margin, and the urine frequently contains albumin. The blood pressure is often raised, and may reach 180 or 200 mm. Hg.; and either as a result of this pressure, or from fatty or fibrous degeneration of its muscular substance, the heart becomes dilated, and the circulation fails. The patient by this time presents many of the clinical features of chronic Bright's disease. Glycosuria of moderate degree is occasionally present also, and women of the child-bearing age are likely to be sterile.

Finally, in many cases the obesity which is the early result of alcoholism gives way to the converse condition; and it is not uncommon to see the patient with wasted chest, arms, and legs, and the abdomen large from retained fat, or cirrhosed liver and ascites; the feet are perhaps œdematous, while the urine is scanty (*see* Cirrhosis of the Liver, Chronic Interstitial Nephritis, and Gout).

In addition to the diseases directly resulting from alcohol, these patients are liable to succumb with great rapidity to any acute illness, such as pneumonia or erysipelas.

**Treatment.**—Complete abstention from alcohol is the obvious remedy, but is more easily prescribed than enforced. The patient's goodwill should be enlisted, but is never to be relied upon, and for this reason treatment is seldom of value unless the subject is kept at first under constant supervision. This can be most easily obtained in a nursing home or institution. In the majority of cases the alcohol can be withdrawn suddenly, but in cases of great excess long continued gradual deprivation is a wise precaution against delirium tremens. Insomnia

is the most troublesome symptom of withdrawal, and must be treated effectually by means of full doses of paraldehyde or medinal. Hypodermic injections of strychnine and atropine are also recommended. When the craving has been overcome an attempt should be made to discover the psychological causes which led to the habit, and plans should be made for the future *régime*. This should include as its first principle complete and permanent abstention.

## LEAD-POISONING

(*Plumbism*)

This occurs among those who have to do with lead or salts of lead, either in the preparation of those substances or in industries in which they have to be used. The latter are especially printing, plumbing, type-founding, type-setting, glazing of china and pottery, house-painting, coach-painting, and manufacture of electric accumulators. It also arises accidentally as the result of impregnation of drinking water with lead from service pipes or cisterns. This occurs when the water is soft, but not when it is hard. Lead may cause poisoning when taken as an abortifacient. It may thus enter the system by the respiratory mucous membranes, by the alimentary canal, or by the skin. Legge and Goadby show that the first is by far the most common way, lead salts being inhaled as a fine dust. Susceptibility to lead-poisoning varies greatly. Women are more susceptible than men. The tendency to be attacked is increased by starvation, ill-health, exposure to cold, and indulgence in alcohol, and by the pre-existence of gout, syphilis, or disease of the kidney, and it is also hereditary.

**Pathology.**—Lead is absorbed from the lung and from the upper part of the bowel, and is excreted by the large intestine and kidneys and perhaps by the skin in sweat and saliva. It is found in the brain, liver, spleen, and bones, though in small quantity; and the cæcum and colon are blackened by its sulphide.

The paralytic symptoms are mainly due to a multiple neuritis, which has been found to be more marked in the peripheral branches than in the larger nerve trunks, and is usually absent in the parts near the nerve roots; but more widespread inflammatory changes may be present, involving the anterior motor cornua, and sometimes the brain. In the muscles the usual degenerative changes are found (*see* p. 800). The lead has obviously a prejudicial effect upon the blood-forming organs, as shown by the anæmia.

**Symptoms.**—The effects which it produces on its victims are—(1) colic; (2) paralysis and other nervous symptoms; (3) "blue line" on the gums; (4) anæmia; (5) changes in the urine; (6) interference with the function of reproduction.

1. *Lead Colic.*—This is a form of intestinal colic. After a few weeks of impaired health, as shown by headache, sick feeling and pallor, the patient is seized with severe spasmodic pain in the lower part of the abdomen or at the umbilicus; the abdominal muscles are contracted, and the pain is rather relieved by firm pressure in many cases, but often the abdomen is tender, and it is generally retracted. On examination the intestines may be felt to be contracted, forming irregular masses in different parts of the abdomen. Sometimes there is vomiting, and the bowels are nearly always confined. The pain diminishes for a time and then recurs, and it is generally relieved in the course of one to three or four days. It is probably due to spasmodic contraction of the intestine, with some diminution of secretion from the intestinal mucous membrane; and some think that vasomotor constriction has an important share in it. Oliver notes that during colic the pulse is slow and hard, and the urine scanty. *Vomiting* is sometimes a troublesome symptom before the occurrence of colic.

2. *Lead Paralysis.*—This is often described under the head of multiple neuritis, but is probably due to a selective incidence of the poison upon the motor cells

of the spinal cord. The characteristic feature is the distribution of the paralysis, which is in most cases confined to the upper extremities, and though it may affect one arm before the other, is usually symmetrical. The first muscles to suffer are the long extensors of wrists and fingers, so that when the arms are held out with forearms pronated, the hands and fingers hang down. Hence the affection is sometimes known among workmen as "the dangles." The index and little fingers, possessing additional extensors of their own, suffer less than the ring and middle fingers. If the hand and the first phalanges are supported in the horizontal position, the remaining phalanges can be extended, showing that the lumbricales and interossei are still active. The supinator longus as a rule escapes. As the result of long-continued weakness backward displacement of the carpus sometimes occurs, and a prominence forms on the back of the wrist.

While this is the most characteristic form of lead palsy other groups of muscles are sometimes affected. Of these the commonest are—(1) the intrinsic muscles of the hands; (2) the muscles of the shoulder girdles, especially biceps, brachialis anticus, deltoid and spinati; and (3) the anterior tibial group in the lower limbs.

Although there may be complaint of muscular pains at the onset, there is no peripheral sense of numbness or tingling, nor any cutaneous anæsthesia.

The affected muscles waste, and on electrical examination show the reaction of degeneration. The supinator longus being as a rule spared, the tendon jerks are unaffected.

*Cerebral Symptoms: Saturnine Encephalopathy.*—These may be in the form of hemiplegia, or of hemianæsthesia. More severe cases, not infrequently fatal, occur in which convulsions, delirium, and coma, with, perhaps, optic neuritis and some fever, are the symptoms. Such cases often run a very acute course, and appear to be more frequent in females (Oliver). Anæmia is the first symptom, and then colic, headache, vomiting, diplopia, or defective vision from optic neuritis. In a few days the patient is convulsed, becomes comatose and dies. In other instances there is mental disturbance, amounting to insanity, which is either acute mania or melancholia, or progressive mental failure and muscular weakness, with, perhaps, convulsions.

Ocular lesions and symptoms are frequent, and include optic neuritis with or without hæmorrhages, neuro-retinitis, primary and consecutive atrophy, inequality of pupils, diplopia, and a bilateral amblyopia without change in the fundus, similar to uræmic amaurosis.

3. The *blue line* on the gums, or *lead line*, has mainly a diagnostic importance, showing that lead has been taken into the system. It is a dark slate-coloured or black finely dotted line, which forms in the gum close to the teeth, and consists of a deposit of sulphide of lead in the tissues around the vessels; this results from the union of lead with sulphur provided by albuminous substance (partly contained in "tartar") at the edge of the gum. Where teeth are absent there is no blue line, and also if the teeth are kept exceptionally clean; or it may be seen only in portions of gum rising between the teeth. Sometimes a blue patch is seen on the inside of the cheek opposite a decayed tooth. The blue line may exist without any other symptoms of plumbism; it persists for from eight days to three months, or even much longer, after all entry of lead into the system has ceased. The lead line must be distinguished from a blue discoloration which accompanies a marked degree of pyorrhœa alveolaris.

4. Sufferers from lead-poisoning are generally *anæmic*, often remarkably so, with a sallow earthy look. The red corpuscles and hæmoglobin are diminished, the colour index being less than 1. There may be a moderate leucocytosis, in which the lymphocytes, large mononuclears and eosinophils are relatively increased in numbers. Many of the red corpuscles contain basophil granules. The anæmia is often the earliest indication of impregnation with lead.

5. Albuminuria is a result of lead-poisoning in two or three ways. It may

occur temporarily in lead colic; and it is often the result of more definite renal change, in the form either of tubal nephritis, as in cases of encephalopathy (Oliver), or of granular kidney in more chronic conditions of plumbism. The relations of lead, gout, and granular kidney are very close. Gout, at least in the south of England, is common in those who suffer from chronic lead-poisoning, which causes, it is said, diminished excretion of uric acid. And it has also been observed that sufferers from gout are very readily affected by lead.

The urine may also contain indican, and lead itself in combination; but the latter is often in exceedingly small quantity.

6. Lead-poisoning affects the functions both of menstruation and of gestation. Menorrhagia is very common, but occasionally there is amenorrhœa or dysmenorrhœa. In pregnant women there is a large proportion of miscarriages and still-births; and of children born alive many are under weight, and many die within a year. Amongst those who survive convulsions, imbecility, and idiocy are above the average proportion.

**Diagnosis.**—This depends for the most part upon the history of lead-poisoning, upon the presence of a lead line upon the gums, and upon the discovery of lead in the excretions. The history may have to be investigated with the greatest care, as lead may get into the system by the most unexpected means. If paralytic symptoms are present, a history of colic is often obtainable, and in nearly all cases the lead line is present, though in very cleanly persons it may only be found between the teeth, or possibly not at all. The common type of lead palsy is readily distinguished from musculo-spiral paralysis by the escape of the supinator longus in the former and the simultaneous affection of both arms. The rarer forms have to be distinguished from progressive muscular atrophy by means of the other signs of lead poisoning.

Lead is present in the fæces in larger amounts than in the urine, so that the former should be used when lead is being tested for. The fæces are incinerated in a porcelain crucible of high quality and treated with nitric acid. The excess of acid is evaporated off, and the lead precipitated with sulphuretted hydrogen as the black sulphide after dissolving the residue in dilute acetic acid.

**Prognosis.**—Recovery, though slow, is, with proper treatment, usually complete. If at the end of two years from the commencement of treatment any weakness still remains, it is likely to be permanent. Severe cerebral symptoms endanger life, but if death does not occur, recovery may be complete.

**Prevention.**—It is unnecessary to say much on this point. The protection of lead workers is a matter for the employers of labour, or, failing them, for the State. Thorough ventilation in all industrial processes, absolute cleanliness in the intervals of work, and periodical inspections of the workers by a medical man to detect the earliest signs of lead intoxication are important considerations. A lemonade containing sulphuric acid is often provided to precipitate the lead as sulphate, and so prevent its absorption. It is obvious that cisterns or pipes used for drinking water should not be constructed of lead, nor should recourse be had to this metal for the preservation of anything, such as food, drink or tobacco, which is to be afterwards taken into the system.

**Treatment.**—The patient should give up his occupation, or prevent in whatever way may be necessary the introduction of more lead into the system.

*Colic* generally yields readily to a full dose of opium combined with a simple purgative; an ounce of castor oil with 15 or 20 minims of tincture of opium is a common and successful prescription. Sometimes it may have to be repeated; a strong saline purge (magnesium sulphate) may be given, or a drop of croton oil may be added to the castor oil. Hypodermic injection of morphia may be used instead of opium if the pain is very severe. Amyl nitrite inhalation and other vaso-dilators are also recommended. Hot fomentations or poultices should in the meanwhile be applied to the abdomen. Iodide of potassium is believed to hasten the elimination of lead, and may be given in 5-grain doses three times

daily for two or three weeks afterwards, but it seems in some cases to have put lead into circulation with ill effect that was previously latent and harmless.

In *paralytic affections* appropriate splinting is of the first importance, the paralysed muscles being placed in the position of relaxation. Massage and movements are also essential. The electric current should be used daily to exercise such muscles as cannot be voluntarily controlled. Potassium iodide should also be given. For *acute cerebral attacks* Oliver recommends the inhalation of nitrite of amyl and lumbar puncture with removal of from 1 to 3 ounces of fluid.

*Anæmia* must be met by suitable treatment with iron preparations in addition to what is necessary to prevent further impregnation, such as removal from work and the use of potassium iodide.

## CHRONIC MERCURIAL POISONING

This is chiefly brought about by the inhalation of the vapour of mercury in those who work in quicksilver mines, in making barometers and thermometers, and in other manufactures. Many such persons are affected with the anæmia, salivation, and stomatitis which are the familiar results of overdosing with mercury used as a drug.

The characteristic of mercurial poisoning is the tremulous movements of the limbs and body known as "mercurial tremors," or "trembles." They come on suddenly or gradually, generally after exposure for some years to the metallic vapours. They affect the arms first, and then spread to the legs and the muscles of the rest of the body. The movements are at first brought out only by excitement, later whenever the patient makes any muscular effort, and finally they become constant, and even persist to a certain extent during sleep. The tongue is tremulous, and speech is hurried, abrupt and jerky. Ataxia and even paralysis may be present in bad cases, and the patient may be quite unable to walk without support. In some cases there are tonic spasms of the flexors of the forearms after violent movements, or hard work. Anæsthesia, vertigo, "loss of nerve," emotional disturbance, delirium, hallucinations and mania may also occur. In the earlier stages there is some resemblance to disseminated sclerosis, but there is no nystagmus; and if the movements become constant, paralysis agitans is, to a certain extent, simulated. Mercury also causes tubal nephritis.

Recovery may take place if the disease is not too advanced.

**Treatment.**—Removal from the fumes of mercury is essential. Tonics and iodide and bromide of potassium are of most value as drugs; and sedatives, such as opium, chloral and belladonna, may be useful.

## CHRONIC ARSENICAL POISONING

This is less common than lead-poisoning, but it may arise in the following circumstances: among persons employed in arsenic factories, and in industries involving the use of arsenical pigments; among persons using such coloured articles as, for instance, certain grey and green wall-papers; occasionally from the use of large doses of arsenic in medical treatment; and in accidental impregnation of beverages with arsenic, such as occurred in the Manchester epidemic of peripheral neuritis, when an impure sulphuric acid was used in the process of malting. Poisoning by arseno-benzol has already been described.

**Symptoms.**—In the case of exposure to arsenical dust, which is highly irritating, the effects are manifest on the skin and accessible mucous membranes; when the arsenic is taken internally, the alimentary mucous membrane, the peripheral nerves, and the skin through the circulation are affected. In the first case the prominent feature is irritation of the skin resulting in a form of eczema or dermatitis, which is seen especially in the warmer or moister parts, such as the

axillæ, between the scrotum and thighs, at the edges of the nostrils, and the eyes. Redness of the conjunctiva and smarting of the eyes, sore throat and irritation, with frequent hawking, are also present. Later there are symptoms of general poisoning, but these are more prominent when the poison has been injected in small doses frequently. There is gastro-intestinal irritation, with perhaps sickness, diarrhœa, and abdominal pains. With this are often combined emaciation, weakness, muscular cramps, and frontal headaches. Peripheral neuritis is a characteristic feature (*see* p. 741), and a disorder of the skin of which pigmentation and thickening of the epidermis, especially on the palms and soles, or *keratosis*, are the prominent features. Transverse ridges and furrows appear on the finger-nails; and in acute cases curved white lines (*leuconychia*) have appeared, showing interrupted nutrition at the time of the poisoning.

**Treatment.**—This consists in removing the cause, when the symptoms will subside; medicinally iodide of potassium may be given in 5-grain doses. Peripheral neuritis may, however, persist for several months.

## CHRONIC MANGANESE POISONING

Commercially manganese is used in making chlorine, paint, varnish, enamel, and linoleum, for the marbling of soap and in the manufacture of steel.

The clinical features of manganese poisoning resemble paralysis agitans and suggest that there is a lesion in the corpus striatum. There are emotional disturbances with periods of uncontrollable laughter or weeping, mental languor, muscular rigidity and tremor. The gait is shuffling, and there is a tendency to fall down. There are no signs of disease of the pyramidal tracts (**1**).

### REFERENCE

1 J. R. Charles .. .. .. *J Neurol. & Psycho-pathol.*, 3, p. 262

# DISEASES DUE TO CERTAIN DEFICIENCIES IN DIET

## ACCESSORY FOOD FACTORS

(*Vitamins*)

THE growth of the young animal depends on two sets of factors: (1) those that are inherent in the organism and are influenced by heredity; (2) those factors in the environment that exert an influence on the organism from without. Food is the most important of these. Till recently it has usually been considered sufficient to calculate the diet solely on the energy requirements of the individual, while a protein ration was allowed more than sufficient to cover the loss by wastage of the tissues and the amount required for the formation of new tissue in the growing organism. Human experience with scurvy through many centuries has shown that something else is wanted, and we find Bachstrom writing in 1734, "This evil" (scurvy) "is solely owing to a total abstinence from fresh vegetable food and greens, which is alone the true primary cause of the disease. . . . Recent vegetables are found alone effectual to preserve the body from this malady and most speedily to cure it, even in a few days, when the case is not rendered desperate by the patient's being dropsical or consumptive." The general principle that it is impossible to keep animals alive on a diet of purified proteins, fats and carbohydrates with the necessary inorganic salts was enunciated by Hopkins (1906). The special substances that must be present in the diet are called *accessory food factors* or *vitamins*. Apart from the antiscorbutic factor, which will be considered later, the most important are a substance present in certain fats (*fat-soluble A*) and another substance present in the germ and outer layers of certain seeds (*water-soluble B*). It is possible that other substances will be added to this list; there is, indeed, some evidence that pellagra is due to a dietetic deficiency, viz., to the want of *tryptophane*. Although this is not yet certain, it has been thought best to include this disease in the present group. Rickets, again, is due to a deficiency of a vitamin, *antirachitic X* or the *calcifying vitamin*, which is similar to, but not identical with, fat-soluble A.

Not much is at present known as to the way in which vitamins act, whether they are required for the nutrition of all cells, as is commonly supposed, or whether they are necessary in the higher animals for the activity of lymphoid tissue (Cramer, Drew and Mottram), or for the correct balancing of the various important metallic ions in the body (7).

**Fat-soluble A.**—Butter, cream, cod-liver oil and other fish oils, and egg yolk provide the most abundant sources of this substance. It is also present in the green leaves of plants and the embryos of certain seeds. It is completely absent from certain vegetable fats and oils such as olive oil, cotton-seed oil, cocoa butter, linseed oil, and also lard (specially prepared pig fat), although bacon, mutton and beef fat contain plenty of it. It is completely destroyed when oils are "hardened" by the action of hydrogen—a process widely employed in the preparation of edible fats; but it can stand being heated to 100° C. for an hour or two. Its presence in milk fat is in part dependent on a good supply of this substance in the food of the cow, and this is satisfied when there is plenty of green food available. In the absence of this substance, young rats cease to grow. They

become specially liable to infection, and often suffer from xerophthalmia. It is claimed by Bloch and Monrad that this disease when occurring in children is due to a lack of this factor in the diet, and is cured by cod-liver oil (**8**). Interesting observations have been recently made on the effect of diet on the spread of dental caries. The diet which gave the best results in preventing caries, besides having the ordinary qualities of a normal diet, was rich both in calcium and in the calcifying vitamin (**9**).

**Water-soluble B.**—This is also known as the antineuritic or anti-beriberi factor. It is very widespread in nature. Its richest supply is the germ of rice and wheat, but the outer covering of the grain which forms bran and the *aleurone* layer just beneath it also contain the substance. Egg yolk, ox liver, yeast, and pulses contain plenty of it. A commercial preparation of yeast called *marmite*, used for soups, is a convenient source of the substance. It is only very slowly destroyed by being kept at 100° C., but much more quickly at 120° C. Hence no serious loss of the substance need be feared in the baking of bread or biscuits; on the other hand, tinned foods are to be regarded as quite free from it. When rice is "polished," the germ and the whole of the external covering of the grain (pericarp) are removed, and so the whole of the antineuritic factor is lost. When young rats are fed on "polished" rice growth stops immediately, and inco-ordination of the back legs occurs, so that the animal can no longer walk about. Pigeons also develop a polyneuritis, and this is regarded as the same disease as beri-beri. When severe symptoms have been produced experimentally they can be rapidly cured, sometimes in a few hours, by giving adequate amounts of the antineuritic factor.

**Antiscorbutic Factor.**—The richest supply of this accessory substance is provided by raw cabbage leaves, raw swede juice, fresh lemon and orange juice; other substances, such as carrots, beetroot, potatoes, onions, grapes, apples, milk and fresh meat, contain comparatively small amounts, while flour, eggs and dried pulses contain none at all. However, if dried pulses, like lentils, peas and beans, are soaked in water and allowed to begin germination for two or three days, they acquire antiscorbutic properties. The vitamin is very sensitive to drying and to high temperature. Dried foodstuffs will not prevent scurvy, and cabbage loses 90 per cent. of its power when boiled in water for an hour, and the loss occurs more quickly still if alkali is present. Hence vegetables should not be cooked for longer than twenty minutes, and soda should not be added.

When young guinea-pigs are fed on a diet containing no antiscorbutic factor, they eventually lose weight and develop symptoms resembling those of human scurvy, and they can be cured by giving food containing the vitamin. Observations have also been carried out on monkeys, in which the disease resembles human scurvy still more closely.

## BERI-BERI

(*Kakke*)

This is an endemic disease consisting essentially of a multiple peripheral neuritis, which causes paralysis and anæsthesia, with cardiac dilatation and dropsy in varying degrees. It is due to the absence of the water-soluble B factor from the diet.

**Ætiology.**—It has been observed chiefly among rice-eating populations, in Japan, China, and the Malay Peninsula. But it may occur anywhere, when a diet consisting chiefly of cereals is eaten, if the germ and external covering have been removed in their preparation. It has been recorded among sailors. It is not so common among wheat-eating as among rice-eating people; but outbreaks do occur, as, for instance, in Newfoundland and Labrador, where the population subsists largely on white bread during the winter and summer.

During the siege of Kut an outbreak of the disease took place among the British troops when they were subsisting chiefly on wheaten flour, but disappeared when they were compelled to eat barley flour, or *attar*, which is a coarsely ground whole-wheat flour used by the Indian troops. On the other hand, scurvy occurred among the Indian troops. The British troops escaped this owing to their large ration of fresh horseflesh, which would not be eaten by the Indian troops. An infantile type of beri-beri is well known in districts where the disease is endemic. White bread is largely eaten in England, but there is sufficient of the accessory factor in the rest of the diet to prevent the onset of the disease. Exercise is to some extent a preventive of beri-beri (**4**).

**Morbid Anatomy.**—*Post mortem*, in addition to the œdema and anasarca seen during life, there are ecchymoses under the serous membranes in the muscles, and in the sheaths of the nerves; the lungs are engorged and œdematous; the right side of the heart is dilated, and in chronic cases hypertrophied; and its muscular fibres may show fatty degeneration. In acute cases the mucous membranes of the pyloric end of the stomach, of the duodenum, of the small intestine and of the colon, are congested or present punctiform hæmorrhages; and the liver and kidneys are congested. In the peripheral nerves is found degeneration of the axon and medullary sheath, going on to total destruction, with cellular infiltration of the perineurium and endoneurium. The muscular fibres suffer loss of striation, atrophy and degeneration with increase of the inter-fibrillary connective tissue.

**Symptoms.**—The disease takes eighty or ninety days to develop on a diet of "polished" rice. The symptoms are, for the most part, weakness or paralysis of muscles, muscular atrophy, and sensory disturbances, which are characteristic of multiple neuritis (*see* p. 740); but there are added in this form certain features not commonly seen in the familiar cases of neuritis due to alcohol, diphtheria, or plumbism—namely, œdema of the legs, or even extensive anasarca, and severe or fatal dyspnœa from cardiac failure or œdema of the lungs

There is, however, considerable variety in the effects produced by these lesions: in some, anasarca is pronounced or extreme (*wet beri-beri*); in others, the muscular atrophy is the chief feature, and the patients are thin and emaciated (*dry beri-beri*); and intermediate conditions occur.

The disease often begins slowly with a state of languor, with weakness of the legs and knees, pains in the calves, slight dyspnœa or tachycardia and œdema of the legs; or the occurrence of weakness and numbness of the legs and pains in the calves may be almost sudden. As the disease develops there are loss of power and muscular atrophy beginning in the extensors on the front of the leg, and then affecting the other muscles of the leg and thigh, as well as later the extensors of the hand, the biceps, and, it may be, the abdominal muscles, the diaphragm, and the intercostals. The knee jerk is generally soon lost, and later the Achilles jerk. When the legs are chiefly affected, the patient has a gait characteristic of foot-drop: the heel is lifted high to clear the ground, and the toes come down before the heel. The weakness of the legs may be shown early in the disease by the following method. While standing the patient bends his knees and separates them so that he assumes a squatting position with the buttocks a few inches from the ground. He is then unable to raise himself from this position, though he may try to do so by the use of his hands upon his thighs, like a patient with pseudo-hypertrophic paralysis (*see* p. 916). This is called the squatting test. Anæsthesia is early noticed in the skin over the tibiæ, and may extend to other parts of the limbs and trunk. Hyperæsthesia of the muscles, especially of the calves, tenderness of nerve trunks, and painful cramps occur, as they do so often in multiple neuritis from other causes. Laryngeal paralysis, shown by loss of voice, and pharyngeal paralysis, by difficulty in swallowing, may also occur.

A little œdema over the shins appears to be almost invariable. When the

anasarca is extreme the urine is scanty. There are in most cases palpitation and dyspnœa, and bruits are heard at one or more of the cardiac orifices. The pulse is soft and rapid. The temperature is generally normal or even subnormal.

Acute cases occur in which after a sense of depression, epigastric pain and nausea, cardiac symptoms rapidly develop with palpitation, dyspnœa, oppression over the heart, and throbbing of vessels, and later scantiness of urine and dropsy. Paralysis and anæsthesia are then observed, and with increasing cardiac weakness the patient may succumb in from one to three or four days.

The more usual cases last from five weeks to twelve months or more, and recovery is often very slow in spite of the use of a presumably normal diet. The danger lies always in cardiac failure, with rapid irregular action, or in asphyxia from œdema of the lungs; and these conditions sometimes arise quite suddenly and carry off the patient. Sometimes fatal exhaustion follows vomiting.

The mortality of beri-beri has varied in different epidemics from 2 per cent. to 70 per cent. Vedder in 1913 states it is estimated to have an average mortality of 5 per cent.

**Diagnosis.**—The early symptoms are weakness of the legs, shortness of breath, malaise and sensory disturbances in the lower extremities. The above-mentioned squatting test should be applied. The diagnosis would be confirmed by a consideration of the dietetic conditions. Other forms of neuritis, such as those caused by arsenic and by alcohol, must be excluded.

**Prevention and Treatment.**—Both for prevention and treatment it is essential to supply the missing accessory food factor. It is important that the germ and bran of wheat should be included in the manufacture of bread and biscuits to be eaten by people with a restricted food supply. This applies specially to soldiers on active service who eat tinned foods in which the accessory substance has been completely destroyed. There are two other sources of food which are rich in this particular vitamin, viz., dried yeast, or *marmite*, which can be made into soup, and dried eggs. These can easily be taken on any expedition. Other symptoms, such as cardiac failure, must be treated as they arise.

## SCURVY

(*Scorbutus*)

Scurvy is characterised by a profound change in the blood, resulting in hæmorrhages under the skin and in other parts of the body, a spongy condition of the gums, anæmia, and prostration. It is due to the absence of an accessory food factor from the diet.

**Ætiology.**—It may occur in either sex and at any age; it has arisen over and over again in circumstances entailing a restriction of the dietary in respect of vegetables and fresh meat. Thus it has been in past times the scourge of sailors on long voyages, so that it is frequently spoken of as sea scurvy, though such a term does not now distinguish it from any other form; and it has severely affected armies and other large collections of individuals, such as those in prisons, and sometimes even in hospitals. Cases occasionally happen amongst those who could get vegetables, if for any reason, such as poverty, dyspeptic troubles, or mistaken views, they have habitually abstained from eating them.

**Morbid Anatomy.**—In fatal cases of scurvy the lesions are found which have been mostly manifest during life—*e.g.* the hæmorrhages in the skin and the effusions of blood in the aponeurotic sheaths and under the periosteum, and in infants separation of the epiphyses. Occasionally hæmorrhage has occurred on the surface or in the substance of the brain. Frequently the pleural cavities and pericardium contain blood-stained serum. There may be engorgement of the lung with serum or blood, and sometimes it is even gangrenous. Hæmorrhages may also take place into the cardiac muscle, into the pericardium, or into

the mucous membrane of the stomach and intestine; these latter may cause abrasion or ulceration. The liver and spleen are often large, much congested, soft, and friable; and an acute nephritis is described as occurring in severe cases.

**Symptoms.**—The disease generally comes on insidiously, taking from four to eight months to develop. The patient loses colour, becomes weak, languid, drowsy, or apathetic, and complains of flying pains in the loins or limbs. After a time—it may be a week or more—petechiæ appear upon the skin of the lower extremities and other parts of the body, and as a rule each hæmorrhage is situated around the base of a hair. The spots are small, red or reddish brown, and not raised above the surface. Some others appear which more or less resemble bruises produced by violence, and large wheals or *vibices* may also be present. These various hæmorrhages occur all over the body; and there may be large extravasations of blood in the eyelids, or subconjunctival ecchymoses, though very often the face is spared. Associated with this purpuric condition must be mentioned the occurrence of tense, brawny, indurated swellings in different parts of the body, especially in the popliteal space, the bend of the elbow, under the angle of the jaw, and in front of the tibia; these are due to effusions of blood, or blood-stained fibrin, or simply pale yellow fibrinous material, under the fascia, or between the muscular bundles, or between the periosteum and the bone.

Another feature which is commonly regarded as constant is the condition of the gums. These become swollen, fleshy, or spongy, detached from the teeth, and projecting beyond them in loose, bluish-red masses, which are painful, and bleed on the slightest touch. The teeth become loosened, the patient is unable to chew, and the breath is fœtid. The swelling of the gums may be so great that they project from the lips, and ulceration often results. The rest of the mouth is not affected in the same way. The tongue is large and indented. Sometimes the gums are not spongy, but only pale; and in all cases the change seems to be determined by the presence of teeth, so that it is absent where there is a gap in the series, and in toothless infants and old people.

When all these changes have developed, the patient has a sallow, bloated look, is markedly breathless on exertion, though no physical signs may be detected in the lungs, is subject to fits of syncope, and is totally unfit for bodily or mental exertion. The temperature, however, is generally not raised; the pulse is variable; and the urine is usually free from albumin. Hæmorrhage from the mucous surfaces, especially epistaxis, is not uncommon; and the feet are often œdematous. The blood has the characters of a secondary anæmia, with slight poikilocytosis, polychromasia, and punctate basophilia, and there may be a slight leucocytosis. The red corpuscles fall to 4,000,000 or 3,000,000, or lower when hæmorrhages are abundant, and there is a low colour index. The coagulation time differs little from the normal.

In more serious cases there is hæmorrhage from the stomach and intestines, or from the lungs; pneumonia, gangrene of the lung, pericarditis, or pleurisy, which may be hæmorrhagic; or enlargement of the spleen and albuminuria. The skin over blood extravasations may slough from pressure or irritation, and leave fungoid and very offensive ulcers. Dysentery sometimes complicates scurvy, but is generally regarded as having an independent origin. The impairment of vision known as *hemeralopia*, or night blindness, frequently occurs; the patient can see clearly and well in the day-time, but in the dusk, or the darkness of night, becomes quite blind, and cannot see his way about. It originates in a disturbance of nutrition of the retina; the ophthalmoscope shows no change in the eye, and normal sight is restored as the scurvy is cured.

Death takes place from increasing exhaustion, with anæmia and emaciation, generally after many weeks. But it may occur more quickly from sudden syncope, from pneumonia or gangrene of the lung, from hæmorrhagic inflammation of the serous membranes, or from cerebral hæmorrhage. In cases which

recover the improvement under suitable treatment is at once manifest, and often very rapid; but it is stated that the deeper effusions may leave thickening and fibrous bands, as a result of which the limbs are partly contracted and the corresponding muscles are atrophied. Sometimes the joints are ankylosed.

**Infantile Scurvy.**—This form of scurvy, known abroad as *Barlow's disease*, is seen in children under two years old, and is frequently associated with rickets, another dietetic disease. Scurvy is liable to occur in infants who are fed on sterilised milk, or on malted and other patent foods, and who do not have enough of, or any, fresh milk. Such children do not lose flesh, but become pallid; and then the limbs, especially the lower limbs, are affected, so that they do not voluntarily move them, and cry whenever they are touched or moved, or even when the hand is brought near them. The child lies often with the thighs abducted and the knees flexed, and so may be thought to be paralysed.

The bones are tender, there may be some œdema of the feet, and there may be swellings as a result of sub-periosteal hæmorrhages; and the crepitus of a separation of the bones at the epiphyseal lines may be felt. If any teeth are through, there may be sponginess of the gums with hæmorrhages, as in adults; and sometimes a fungating mass is seen; other hæmorrhages, such as epistaxis or hæmaturia, may occur. The anæmia is of the chlorotic type, and the blood presents an increased number of the mononuclears, some myelocytes, and nucleated red corpuscles.

**Diagnosis.**—There can be little difficulty in recognising this disease when the circumstances are such as have been known to lead to it, but the diagnosis requires care in isolated cases. It is distinguished from *purpura* by the general illness accompanying it, by the spongy gums, and by the deep-seated effusions in the limbs and elsewhere. On the other hand, amongst the poorer classes of the population one may overlook mild cases, where the symptoms mainly consist of vague pains, with anæmia and ill-health, and the patients are likely to disregard a slight change in the gums or a few spots on the skin. An inquiry into the diet will soon determine the nature of the illness. In infants it may be confounded with infantile paralysis, syphilitic epiphysitis, or periostitis.

**Prevention and Treatment.**—It is essential to give food containing the antiscorbutic vitamin. Fresh lemon juice has long been known as a very efficacious preventive and remedy, and it retains its properties to a considerable degree when it is preserved and even when dried (Bassett-Smith). Fresh limejuice is not so efficacious, and preserved limejuice is quite useless. Lime-juice obtained its popular reputation because originally the term was used to describe the juice of lemons from the Mediterranean. In the middle of the last century West Indian limejuice was substituted in the navy under the same name, but experience showed its uselessness. For soldiers in the field it is probable that dried pulses allowed to germinate just before being used may be the best means of supply of the vitamin; they are convenient owing to their portability. Cabbage is the best vegetable to use, but it must be cooked for as short a time as possible in water without soda. Scurvy has broken out in a camp where the cabbages were cooked with the meat for some hours in a stew. Fresh meat is also of some value. As regards the diet of infants, it is to be noted that cows' milk is one of the less valuable antiscorbutic foods, and such vitamin as is present is diminished by sterilising or pasteurising the milk. It is probable that the vitamin is completely absent in dried milks. In any case, when sterilised or dried milk is used some of the vitamin should be added to the food. The simplest plan is to give the infant a teaspoonful of orange juice; but swedes, which are plentiful and cheap, may also be employed. Raw swede juice is obtained by grating the cut vegetable and squeezing the pulp in muslin. For curative purposes large amounts of lemon juice can be taken without causing digestive disturbance if the free citric acid is removed by treatment with calcium carbonate.

# RICKETS

(*Rachitis*)

Rickets is a disease involving the nutrition and general health of infants and young children, and showing itself (1) in a perversion of the process of ossification, so that the bones are softer than normal, and become bent and deformed by the pressure which they must undergo in everyday use, and (2) in a liability to respiratory and gastro-intestinal infections and to functional nervous disorders.

**Ætiology.**—It is essentially a disease of children, and affects the sexes about equally. The earliest bony changes are noticeable between the ages of six and twelve months. By eighteen months the clinical features are well marked. Occasionally it begins later; a few cases of *late* or *adolescent* rickets have begun at any time between six and eighteen years. The children of the poor are much more severely affected with it than those of the richer classes, though the latter not uncommonly have it to a slight degree. Its maximum incidence is in winter and spring; it tends to become cured by the summer sunlight.

**Pathology.**—Many hypotheses have been put forward as to the cause of rickets: that the disease is due to incorrect diet, overcrowding, lack of fresh air and exercise; that it is due to some infection or intoxication, lack of calcium or phosphorus, or, finally, to hypothyroidism or other endocrine disturbance. The disease has just received intensive study in this country and elsewhere. An experimental investigation on the cause of rickets artificially produced in dogs (E. Mellanby), which has been confirmed by experiments on rats (Korenchevsky), leaves no doubt that the disease is primarily due to a dietetic deficiency, and particularly to an absence of the right kind of fat. Further, the fats that prevent the occurrence of rickets are those which contain the *fat-soluble A* vitamin, whereas the fats that contain little of this are less efficacious in preventing the disease; both the antirachitic substance ("antirachitic X") and the fat-soluble A vitamin are very resistant to heat; but it is probable that the two are not identical, though they commonly occur together in nature (**1**). While the absence of this substance is the main cause of rickets, there are several other factors which have been shown experimentally to play a part. Lack of calcium by itself does not produce typical rickets, although there soon develops a deficiency of calcium in the bones, which show *osteoporosis* with narrow layers of *osteoid* tissue; there is little change in the zone of proliferating cartilage. However, a lack of calcium undoubtedly increases the severity of rickets if caused primarily by a deficiency of antirachitic X in the diet. The absence of meat and close confinement of the animal in a cage aggravate the disease. On the other hand, if the diet contains plenty of calcium, phosphorus and meat, and if the animal is allowed plenty of exercise, much less of the antirachitic substance is required to prevent the onset of rickets. The importance of exercise has been emphasised by Findlay and Paton. There are three more interesting facts: (1) that an excess of bread, and probably other cereals, definitely predisposes to the disease. It is possible that this is connected with the well-established clinical observation that rickets is chiefly seen in the growing child and not so much in the wasted and undersized one. This fact is of importance in this country, as, if milk is deficient, the child usually makes up for it with bread. Experimentally, it has been shown in animals that growth is necessary for rickets to be produced. Now the fat-soluble A vitamin is necessary for growth, and in its absence there is no growth and no rickets. (2) The administration of a commercial preparation of caseinogen prepared from milk, and free from calcium, aggravates the disease in dogs. This is an argument against altering the proportion of the protein constituents of milk until more is known about the subject. (3) Observations on babies in the Children's Hospital at Vienna showed that exposure of the skin to sunlight or to ultra-violet light from some source such as the mercury vapour quartz lamp

played the same part as the antirachitic X in preventing and healing rickets. The action of light facilitates the use of such stores of antirachitic X as the body possesses, so that less of the substance in the diet is necessary. Light alone will not permanently prevent the occurrence of rickets. It is possible that calcification of bone may be interfered with if infection is present; but it must not be forgotten that infection is often secondary to rickets, because the resistance of the child is lowered.

The complicated nature of the metabolic disturbance in acute rickets is shown by the fact that there is a lowering of the plasma bicarbonate (*see* p. 547), which is not due to ketosis (3).

**Morbid Anatomy.**—The changes in rickets are best seen at the ends of the long bones, or of the ribs. If the swollen portion at the junction of the rib and its cartilage be divided longitudinally, it will be seen that the line between the two structures is wide and remarkably irregular, instead of being narrow and straight, as it is in healthy bones. Normally between the hyaline cartilage and the bone already developed several layers can be seen microscopically: (*a*) proliferating cartilage cells without orderly arrangement; (*b*) cartilage cells arranged in columns; (*c*) calcification; (*d*) ossification. In rickets the proliferating and columnar zones are thickened, reddened by new vessels, and have thrown out processes irregularly into the cartilage and bone on either side. The proliferation of cartilage cells, preparatory to ossification, has taken place with great freedom, but with no uniformity, as it does in health; calcification has begun unduly early in some cartilage cells, whereas it is deficient in others. The processes of proliferation of cartilage cells, of deposition of calcium salts, and of formation of medullary spaces, take place not in a uniform, regular, or progressive way, but in a most disorderly manner, and with varying degrees of rapidity at different spots. Analogous changes are seen on the surface of the bone where it is formed from periosteum. The whole bone is unusually vascular, and the contents of the medullary cavities are redder than normal. Bony trabeculæ, looking apparently normal, have a much-diminished calcium content, while abnormal *osteoid* tissue is seen in varying amounts.

The muscles are flabby and wasted. The blood may show a secondary anæmia.

**Symptoms.**—Early in the complaint two symptoms occur which are very constant. One is that the child is restless at night, kicks off its clothes, and lies with its legs and arms exposed; the other is that when it goes to sleep it perspires profusely about the head and neck, so that the pillow is saturated. But the rest of the body is often dry, and the temperature is normal. The first evidence of osseous changes is the *enlargement* of the ends of the long bones. This is well marked at the wrists, where the ends of the radius and ulna are thickened, and at the ankles and at the knees; but it is perhaps most unmistakable at the junctions of the ribs with the costal cartilages, where a series of nodules are formed, reaching on either side from the first rib near the sternum downwards and then outwards to the twelfth rib in the flank. This has been called *beading* of the ribs, or the *rickety rosary*. The defects of ossification are seen also in the skull, where the fontanelles are large, and may not close until long after the usual time, which may be put at about eighteen months. Another symptom is the *delay in the eruption of the teeth*, the first of which may not appear until the eleventh or twelfth month instead of the sixth or seventh; and the order of their appearance may present many irregularities. The permanent teeth also suffer; the enamel is defective and soft, and the calcium content is lowered. Defects in the teeth have been shown to occur in experimental rickets produced in puppies (May Mellanby) (*see also* p. 686).

Accompanying the enlargement of the ends of the bones there is an abnormal softness, in consequence of which the bones yield to the traction of the muscles or the weight of the child's body, and become bent so as to produce characteristic deformities of the limbs, the chest, and the pelvis; the head also acquires a

peculiar shape, though it is less easy to see how this happens. If the disease comes on after the child has begun to stand or walk, these accomplishments are given up, and the child "is taken off its feet," as the mothers are apt to explain. If the disease begins earlier, then the art of walking may not be attained until the eighteenth or twenty-fourth month. In either case the child tries to walk before the bones are completely consolidated, and the weight of the body causes the tibiæ and femora to be bent or "bowed," generally with a convexity outwards and forwards. Sometimes there is a convexity inwards at the lower part of the tibiæ, the feet being thus widely separated, and this is to be attributed to the child getting about the floor in a sprawling position, using the feet like the hind fins of the sea lion. If, while still unable to stand, the child crawls much about the floor, the weight of the body falls upon the arms, and the radius, ulna, and humerus get correspondingly bent. In the chest, deformity is produced by the action of the diaphragm, which sucks in the ribs at the softest part; a wide groove on either side of the sternum is thus produced. The sternum is prominent, and the upper part of the chest has a somewhat square shape; the lower ribs, however, are often expanded over the abdominal viscera, forming the upper arch of a protuberant abdomen, which contrasts strikingly with the narrow chest above, and is separated from it by a transverse depression (Harrison's sulcus). The latter is roughly at the level of the dome of the diaphragm, where the chest tends to fall in. The pelvis does not usually show any deformity in infancy, but in extreme cases of rickets the pelvic aperture is considerably misshapen, being mostly of an hour-glass type, and it may afterwards in females offer very serious obstruction to parturition.

The head, besides presenting large fontanelles and often lines of depression corresponding to the coronal and sagittal sutures (*hot-cross bun* type of skull), acquires a somewhat square shape, the vertex being flattened, and the frontal and lateral regions being rather prominent. In pronounced cases the cranium looks very large in proportion to the face, and the circumference of the skull is often increased, but it does not therefore follow that the contents are greater than normal, since the volume of a cube is less than that of a sphere with the same surface. *Craniotabes* is the name given to soft areas on the skull due to deficient ossification, which occur chiefly along the margins of the sutures.

In extreme cases there is a considerable stunting of all the bones, as well as the shortening by curvature; and children of eight or ten may be no taller than those of three years old. The bones are also more fragile than normal, and *green-stick fractures* are apt to occur. There is decided laxity of the ligaments and hypotonicity of the muscles, so that there is extra mobility of the joints (*acrobatic rickets*). The liver and spleen are easily felt because they are pushed down by the contracted chest, and are ill supported by the abdominal muscles, and there is abnormal prominence of the abdomen (*pot-belly*). The appetite may be very good, and many rickety children show a perfect or even excessive development of fat (*fat rickets*), but in severe cases there is anæmia; if any disturbance of the stomach or bowels is present it is to be attributed to diet which may have caused the rickets, and not to the rickets itself. On the other hand, the nervous system is seriously involved; rachitic children are very liable to infantile convulsions, including the special forms known as *laryngismus stridulus* (*see* p. 211) and *tetany*. Associated with laryngismus is often *facial irritability*, or Chvostek's sign, in which a gentle tapping over the superficial branches of the motor nerves of the face causes contraction of the corresponding muscles. In rickety children also, rather more frequently than in others, occurs the disorder known as *head-nodding* or *head-shaking* (*spasmus nutans*), which is often associated with *nystagmus*. These occur in babies of from four months to one year, and may be due to imperfectly lighted rooms in the winter-time, thus being comparable to *miners' nystagmus*.

Rickets is essentially a recoverable disease in the sense that it does not directly

cause death, and that the process of bone-softening ceases after a time, although it may have produced deformities which are permanent. If the disease is but slight it may leave no traces in after-life, and this is probably the case with the majority of patients: the bones become hardened, and the limbs ultimately become perfectly straight. But in other cases the effects of former rickets may be seen in the big square head with prominent forehead, the curved femora and tibiæ, and the pigeon-breast of the adult. Rickets in childhood is often associated with infection of the respiratory tract, which takes the form of bronchitic attacks. These are the more serious owing to the soft state of the ribs. By their want of rigidity the act of coughing especially is rendered imperfect, and the secretions accumulate to the imminent danger of the child. Death is often brought about thus; and in other cases the frequent occurrence of bronchitis, by the collapse of lung which it produces, helps in the formation of the pigeon-breast.

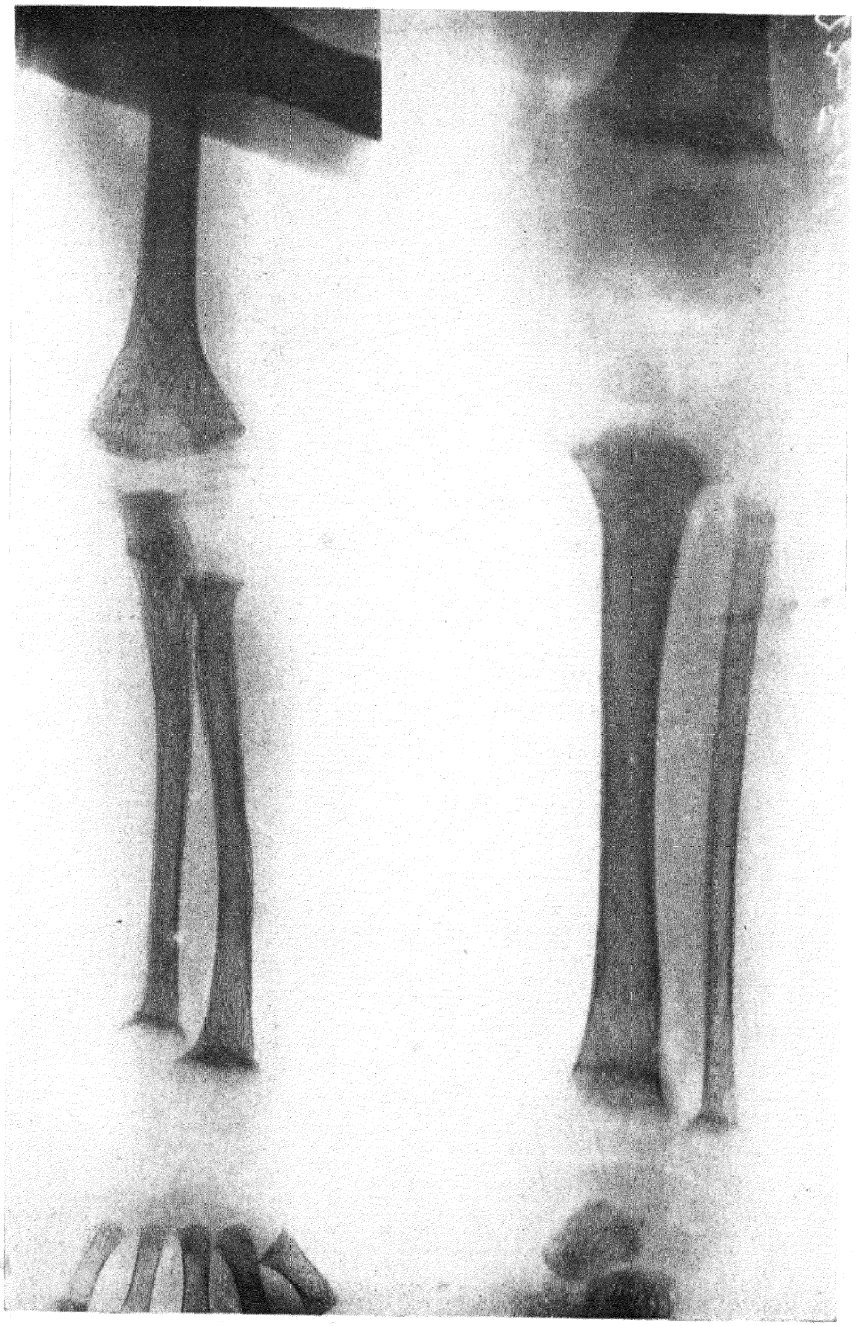
Rickety children may also suffer from symptoms suggestive of scurvy, such as tenderness of the bones. This is not to be wondered at, since the diet may be deficient in vitamins generally. Mention may here be made of a disease in children ascribed by Czerny to a diet containing excessive amounts of carbohydrates. It has been called *hydræmic atrophy*. The child becomes œdematous, resembling a case of Bright's disease; but there is no albuminuria.

**Diagnosis.**—This rarely presents difficulties. The important early signs are the beading of the ribs, and the thickening of the wrists, and the apparent enlargement and abnormal shape of the head, which are often accompanied by restlessness, sweating of the head, and the dislike to being covered at night. Sometimes the inability to walk may lead to a suspicion of infantile paralysis (anterior poliomyelitis), but the limbs can at least be moved, and the deformities of the bones should give the right clue. Syphilitic epiphysitis and scurvy cause great pain and tenderness in the bones affected.

X-rays provide a valuable means of diagnosing the extent of the disease and the progress of cure (**2**). In severe rickets (Plate XXXIII.) decalcification of the finer trabeculæ produces a coarsening of the structure of the cancellous bone, and later as the process becomes further advanced the bones can hardly be differentiated from the soft tissues. The sharp edge of the metaphysis becomes lost; the decalcification occurs more rapidly at the centre, so that the end of the shaft becomes cup-shaped. At the beginning of healing (Plate XXXIV.) a new primary calcification zone appears between the epiphysis and the abnormal transparent tissue at the end of the metaphysis. Gradually the intervening space gets filled up by calcification, and for a long time after healing has occurred a marked contrast may be seen between the dense over-calcified metaphysis and the rarefied cancellous tissue of the shaft in its neighbourhood (Plate XXXV., *cf.* Plate XXXVI., normal).

**Prognosis.**—Recovery is the rule, the bones ultimately becoming quite firm and solid; but the deformities, if considerable, will be perpetuated. The risk to life is from the complications, especially bronchitis, with collapse of lung, convulsions, and laryngismus stridulus.

**Prevention.**—Proper infant-feeding is the all-important point. Breast-feeding is the natural method, and provided there is an adequate supply of milk and the mother takes a varied diet containing all the accessory food factors, there is no fear that the child will develop rickets or other deficiency diseases. When other food is taken towards the end of the first year, the supply of fat-soluble A should be kept up. Egg yolk may be given, and later on butter, beef dripping, green vegetables, fish and meat. In this way any tendency to rickets from bread and other cereals in the diet will be counteracted. In such a diet the supply of calcium and phosphorus will be adequate. A child properly fed will spontaneously take enough exercise by movements of its limbs and body. It should live in well-ventilated rooms, and should be taken out in the fresh air



Severe Rickets in a Child 11¾ months old. Note coarse structure in diaphysis of humerus, radius and ulna, the ill-defined metaphyses of femur, tibia and fibula, the cup-shaped appearance of lower metaphysis of ulna and the almost complete transparency of epiphyses of femur and tibia. (Photograph taken by Dr. Wimberger in the Röntgen Laboratorium of the Kinderklinik at Vienna (Vorst. Prof. Pirquet), reproduced by kind permission of Miss H. Chick and of the Medical Research Council.)

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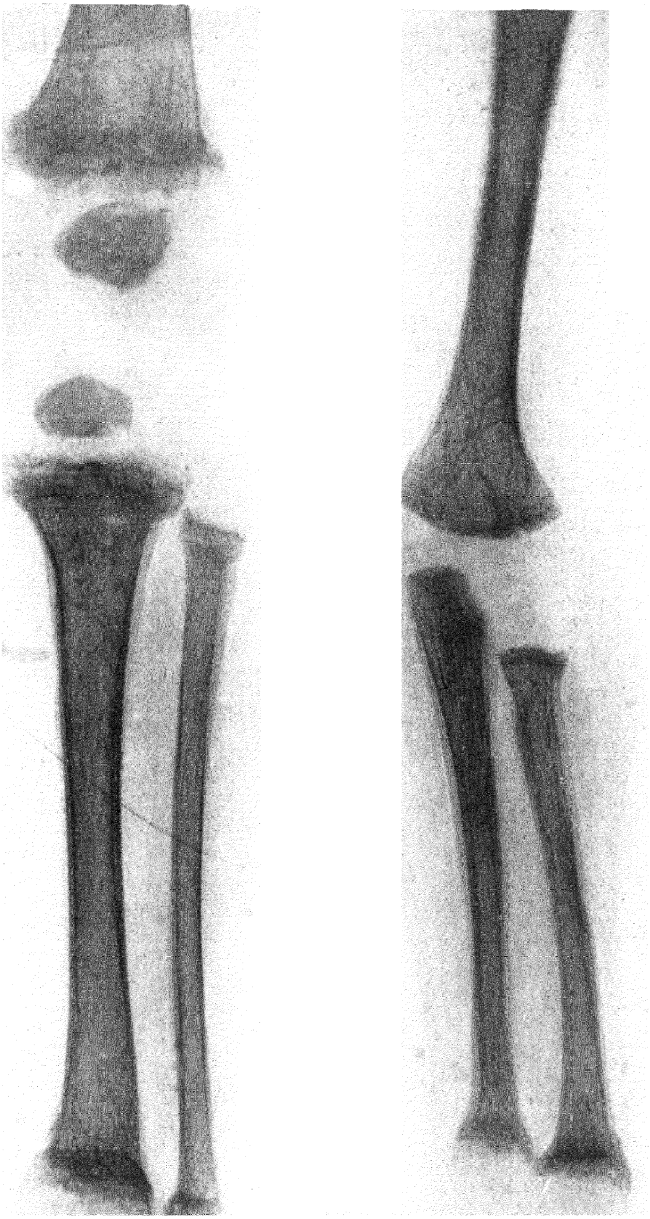

Same child one month later, showing beginning of healing. Note new zones of calcification in distal parts of metaphyses, and greater opacity of the epiphyses. (Photograph taken by Dr. Wimberger in the Röntgen Laboratorium of the Kinderklinik at Vienna (Vorst. Prof. Pirquet), reproduced by kind permission of Miss H. Chick and of the Medical Research Council.)

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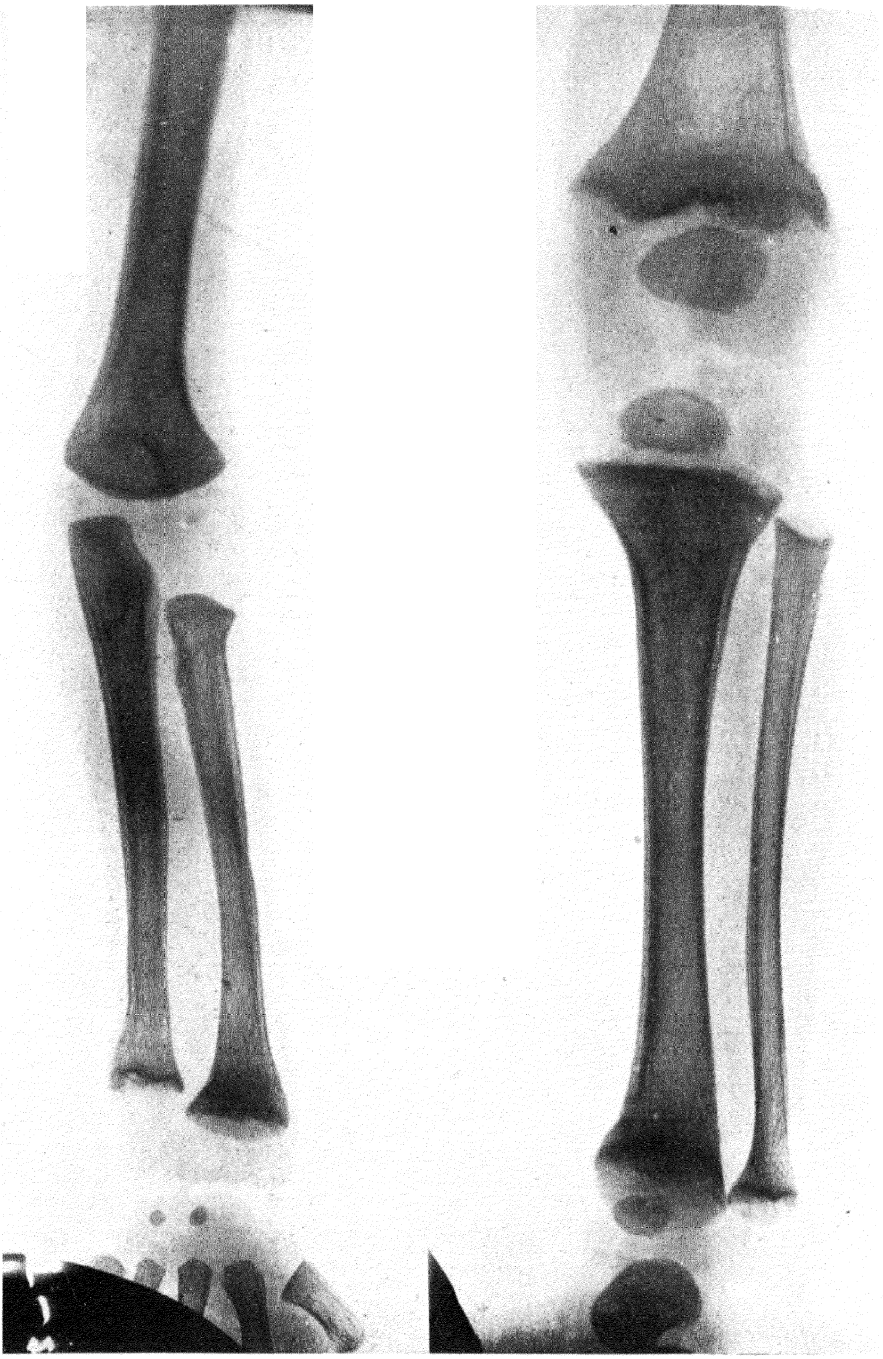

Same child, Twenty-four days later still. Note extra opacity of metaphyses contrasted with the diaphyses, shown particularly in the case of the femur and lower end of ulna. (Photograph taken by Dr. Wimberger in the Röntgen Laboratorium of the Kinderklinik at Vienna (Vorst. Prof. Pirquet), reproduced by kind permission of Miss H. Chick and of the Medical Research Council.)

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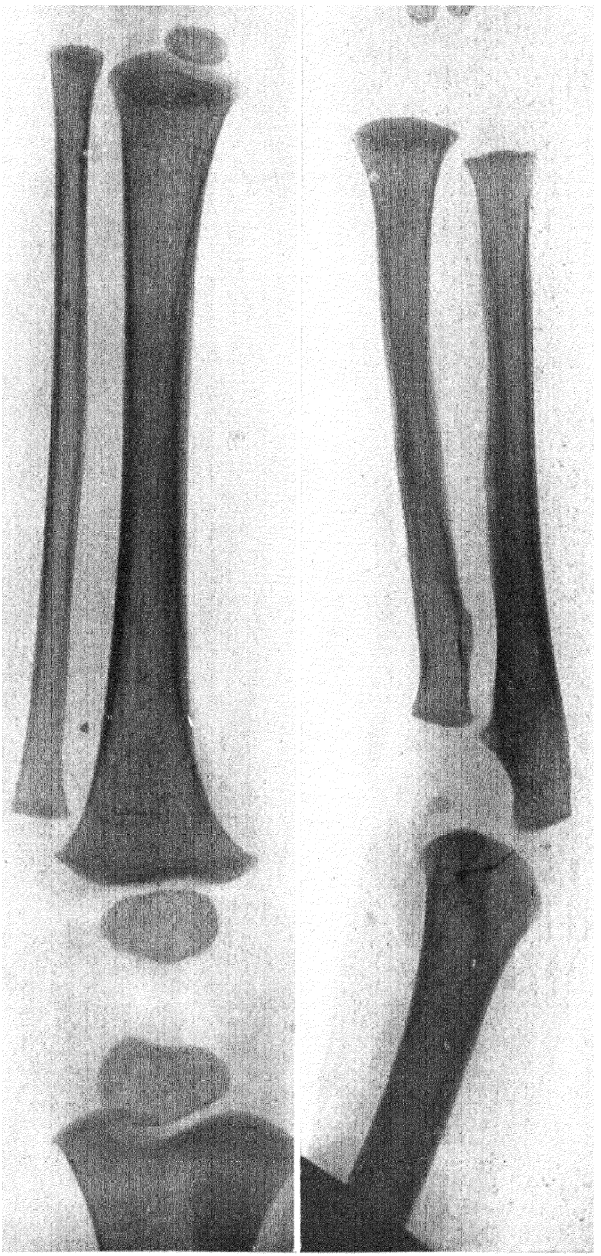

Arm and Leg of a Normal Child, aged Fourteen Months. Note structure of diaphyses, their sharply defined ends (metaphyses) and the appearance of bone formation in some of the epiphyses. (Photograph taken by Dr. Wimberger in the Röntgen Laboratorium of the Kinderklinik at Vienna (Vorst. Prof. Pirquet), reproduced by kind permission of Miss H. Chick and of the Medical Research Council.)

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regularly and get all the sunlight possible. Where artificial feeding is used, cows' milk is the best food. This may be diluted if required and extra cream and sugar added; lime water or sodium citrate (2 grains to the ounce) may also be added. Further alteration in the protein content of the milk is best avoided. Dried milks may be used if they contain all the cream, but it must be remembered that the antiscorbutic vitamin is destroyed in the process. Condensed milks are best avoided, owing to their high content of carbohydrate. Proprietary foods containing starch or dextrins are often very poor in fats, and should also be avoided in the case of infants.

**Treatment.**—The dietetic measures already enumerated should be carried out. Cod-liver oil is the most valuable drug. This may be given three times a day after meals. The dose is 15 drops for an infant six months old, 20 drops up to twelve months, ½ drachm up to eighteen months or two years, and 1 drachm for older children. Fats devoid of the calcifying vitamin must be avoided. This warning is necessary because these substances are cheap, and formerly cotton-seed, linseed, and olive oils were used for rickets. Margarine, especially the cheaper brands, must be avoided. If there is no sunlight, exposure to an artificial source of ultra-violet light, such as the mercury vapour quartz lamp, the tungsten arc lamp, etc., should be carried out. Iron is often employed, as syrup of the phosphate; and preparations of calcium, such as the lacto-phosphate, are recommended. When the child is under treatment, and the bones are soft, it is desirable to prevent their being bent by the weight of the child's body. Walking should be forbidden, and it may be conveniently prevented by fixing to each leg a flat splint from the knee downwards and projecting 3 or 4 inches beyond the foot; these may be removed at night. At the same time the child should be encouraged to move his legs about, although he may not walk, because it is generally admitted that exercise is beneficial. Plenty of fresh air should be allowed. The deformities of the limbs which remain after rickets is cured may, if extreme, be treated surgically.

## HUNGER OSTEOMALACIA

This was observed in Vienna in 1918. The symptoms and appearances of the bones are exactly the same as those of osteomalacia of pregnancy (*see* p. 673), but there is some doubt as to whether the ætiology of the two is the same. The spine may be affected, producing X-ray changes similar to spondylitis deformans or osteomalacia with severe pain on movement and tenderness (**5**). The cause of hunger osteomalacia is fairly clear. The patients had subsisted on a diet of bread and vegetables with small amounts of flour and sugar, and a little lard occasionally. Thus there was a deficiency of fat in the diet. It is noteworthy that rickets and late rickets among young adults were also unusually common in Vienna at this time. The identity of rickets and osteomalacia has been urged on histological grounds by several authors, and is also supported by the fact that tetany occurs in both (**6**).

Cod-liver oil was found to be by far the most potent agent in the cure of hunger osteomalacia, although some success was obtained with large amounts of rape oil containing 0·01 per cent. of phosphorus. These facts point to the disease as being due to a lack of fat-soluble A in the diet of elderly people.

## PELLAGRA

Pellagra (*pelle*, skin, and *agro*, rough; or *pelle* and *agra*, as in podagra), first described in Italy (1700), in Spain (1735), and France (1818), is a disease of which the chief features are an eruption on the skin leading to pigmentation, low blood pressure, diarrhœa, and mental depression ending in dementia. Thus

there is some resemblance to Addison's disease. The cause of the disease is not yet certain. It occurs in people who eat maize which is deficient in the amino-acid tryptophane, and the disease is probably due to the lack of this substance, which is necessary to life. Indicanuria is a feature of the disease, and this indicates an excessive destruction of tryptophane. Only a certain proportion of individuals eating maize are attacked, and it may be supposed that the disease occurs in those who, in addition to a diminished supply of tryptophane in the food, suffer from an excessive destruction of this substance in the alimentary canal.

**Ætiology.**—The disease has been known chiefly in Spain, Northern Italy, France, and Rumania, and more recently in Hungary, Corfu, Egypt, the Southern United States, Mexico, Brazil, Argentina, and the West Indies; and it has now been shown to have occurred in the British Isles. It occurs at all ages, and attacks both sexes. It is probably commoner in children than is generally supposed, as the symptoms may be slight and be overlooked until later life. The subjects are mostly peasants of the poorer class, dwellers in the country, and in Egypt the fellaheen mostly, as well as masons' labourers, brickmakers, potters, pedlars, readers of the Koran, and beggars (Sandwith). The disease begins commonly in the spring of the year, that is, in Europe about March, April, or May, but in Egypt its first appearance is generally in January or February.

**Morbid Anatomy.**—The most marked changes are found in the spinal cord, consisting of sclerosis or degeneration of the posterior columns and of the lateral columns. The former are most affected in the cervical and upper dorsal regions, the latter in the middle or lower dorsal regions. There is round-cell infiltration of the perivascular spaces, with pigmentation and degeneration of the nerve elements and diffuse leptomeningitis. The brain is wasted generally. There is pigmentation of the solid viscera, and of the skin; while fatty degeneration of the heart, liver, and kidneys and fibrosis of the liver, kidneys, and spleen are common. There is chromatolysis of the cells of the sympathetic ganglia.

**Symptoms.**—The first symptom is an erythema, like a severe sunburn, occupying the face, the front of the chest, the hands, forearms and elbows, the feet, insteps, legs and knees. In general the parts of the skin exposed to the sun or subject to external pressure are chiefly affected. The extensor surfaces of the limbs are more affected than the flexor. With the redness there are itching and burning sensations, and petechiæ and bullæ may occur. After about a fortnight the redness subsides, and the epidermis is shed in grey or brown flakes, leaving a surface thickened and often striated. The patient may also complain of lassitude and slight attacks of vertigo. With the advent of the autumn the symptoms disappear, and the patient may regain apparently perfect health. The symptoms, however, often recur with the next spring, and may continue to do so with increasing severity for many years. After repeated attacks the skin remains pigmented, wrinkled, dry, and atrophic. The blood pressure is low.

The disturbances of the *digestive system* may be pronounced in the form of epigastric pain and tenderness, flatulence, and thirst. The tongue is bared of epithelium, and in late stages diarrhœa almost always sets in.

The third feature in this disease is the occurrence of *spinal* and *mental* symptoms. The former consist of pains in the back, with tenderness of the dorsal or lumbar spines, increase of the knee jerk in 75 per cent. of the cases, weakness and stiffness of gait or tremor. In quite late stages there may be definite paralysis, with loss of knee jerks, and of vesical and rectal control. On the mental side are observed from the first depression, gloom, or stupor, loss of memory, insomnia, restlessness, irritability, and delusions and hallucinations; and with repeated recurrence of the attacks for three or four years the patient becomes melancholic, with occasional attacks of mania, and ultimately lapses into a hopeless state of dementia. Pellagrins contribute a large proportion of lunatics to the asylums of

the countries in which this disease occurs; and, as bearing on its ætiology, Sandwith observes that such chronic lunatics may develop a new erythema every spring in spite of the fact that maize is never given to them as food.

**Diagnosis.**—This is generally easy in the countries where the disease is endemic. Some confusion with other nervous diseases, such as tabes, may occur if the eruption is absent, as it sometimes is. In Egypt the disease is very frequently complicated by ankylostomiasis or by bilharziosis.

**Prognosis.**—This is essentially bad; but early cases seem to have been improved, or even cured.

**Treatment.**—The first essential is the removal of the patient from the pellagrous district in which he has been living to an area where the disease is not endemic. He should be given a good liberal diet. No drugs other than general tonics have any influence.

## HUNGER ŒDEMA

*(War Œdema)*

This was observed extensively in the Central Empires during the War. It is probably identical with "epidemic dropsy," first observed in India in 1877–78, which was thought to be due to a one-sided dietary owing to the high price of food. Other similar outbreaks have been described from time to time. It is probably a food deficiency disease, although it is uncertain whether it is due to the lack of fat or of a special vitamin, such as fat-soluble A. It is not due to cardiac or renal insufficiency. It comes on acutely, usually involving the feet and legs first of all, less often the face. It may become generalised, affecting all the serous membranes. The blood is hydræmic and anæmic. When the patient is put to bed a sudden diuresis often takes place, and the œdema completely disappears. All the organs are wasted; and intercurrent infections are common, particularly those of the intestine, producing diarrhœa. The pulse is slow. This is significant, as the pulse is also slow in complete starvation; but in this condition starvation œdema is not noticed. It is possible that hunger œdema is due to a combination of semi-starvation and hard muscular work, and Jaquet states that it always arises when the calorie value of the diet falls below 1,400 in the adult. The diet of heavy bodily workers usually contains 3,900 to 5,000 calories a day.

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# DISEASES OF THE NERVOUS SYSTEM

UNDER this heading we have to deal with disorders of the brain, spinal cord, and nerves—disorders which manifest themselves through the functions of motion, sensation, the special senses, and the intellect and emotions.

Although the nervous system is affected by the same pathological processes as the rest of the body, there are certain peculiarities of structure and function which distinguish the effects of disease of this part from all others.

The first point of importance is the extreme specialisation of function which reaches its height in the higher parts of the central nervous system. Thus it happens that a minute lesion for instance in the mid-brain may produce symptoms and signs which we can attribute with certainty to the situation of the disease. Such a group of symptoms and signs is said then to have a *localising value.*

A further consequence of specialisation of structure is the tendency of certain poisons—bacteriological or chemical—constantly to pick out certain groups of nerve cells or fibres. This *selective incidence* must depend upon peculiarities of a chemical or physical nature resident in the nerve cell which render it especially liable to some poisons, resistant to others. Thus it happens that a particular sign comes to be very frequently associated with the same pathological process. But we must always remember that the *sign is the result of the localisation of the lesion and not of its pathology.* The spirochætal poison of syphilis, for instance, has a selective incidence upon minute centres in the mid-brain which control the reaction of the pupils to light. Loss of the pupillary reaction to light—the Argyll Robertson pupil—is therefore one of the commonest signs of nervous syphilis. But we are not entitled to conclude that the Argyll Robertson pupil is *always* due to syphilis. Any other cause operating in the same situation *may* produce the same sign—for instance a tumour, or the virus of encephalitis lethargica.

A second distinctive feature of nervous diseases is due to the relatively enormous length of the nerve cell. When the axon of a nerve cell is injured or diseased the distal part undergoes degeneration down to its peripheral twigs. Thus a lesion of the first thoracic nerve root within the vertebral column will result in degeneration of nerve fibres supplying the small muscles of the hand, and consequent paralysis and atrophy of these muscles. And a lesion of the cells in the upper part of the motor cortex will cause degeneration of the pyramidal fibres coursing down the spinal cord as far as their termination in the lumbar enlargement, with paralysis of the lower limb.

A third striking feature of the nervous system is the way in which its parts are enclosed, and, as it were, insulated from other tissues—the central nervous system by means of the meninges and the peripheral nerves by their neurolemmal sheaths. Associated with this is the observation that primary tumours of the nervous system never erode the meninges—however malignant they may be within these bounds—or invade other tissues. Nor, on the other hand, do malignant growths elsewhere ever directly invade nervous tissue (although, of course, they may produce damage by mechanical compression).

Another peculiarity of the nerve substance is the absence within the brain and spinal cord of any fibrous (meso-blastic) supporting tissue, its place being taken by

the much more delicate neuroglia. Hence the tendency of suppuration to spread deeply instead of becoming limited by a fibrous barrier. There is a similar liability in hæmorrhage, whether accidental or the result of vascular disease. The pressure of the effusion easily suffices to tear through the fine neuroglia fibrils and excavate the substance of brain or cord. In conditions of chronic inflammation, however, the neuroglia cells proliferate and form an excess of fibrils which leads to an increased density. Moreover, the degeneration products of nerve fibres undergoing " secondary degeneration " (*e.g.*, a degeneration of pyramidal fibres in the cord secondary to a lesion of the internal capsule) appear to provoke a similar reaction on the part of the neuroglia cells, so that, after the decayed nerve fibres have been absorbed by scavenging cells their former course is marked by an increased density of neuroglia, which is often both visible and palpable. Such appearances led, in the early days of neuro-pathology, to the assumption that the overgrowth of neuroglia was the primary disease process, the destruction of nerve cells being a secondary result, and the pathological process at work was named " *sclerosis.*" We now believe that this conception was inaccurate, and that the primary effect of injury or disease is degeneration of the nerve cells with secondary neuroglial overgrowth.

A further point of importance is the fact that the brain and cord are enclosed in a tough fibrous membrane within a bony cavity. There is little room, therefore, for expansion, and a bruise such as elsewhere in the body would result in local swelling and pain, or a benign tumour not invading the nervous tissues may readily cause serious damage by compression.

Apart from the spread of organisms by direct continuity there are two main channels through which bacteria or toxins may gain entrance to the central nervous system. The main pathway is through the blood stream. But in at least two diseases—tetanus and diphtheria—the toxin travels centrally from the site of infection by way of the perineural lympathic sheaths. This possibility has also been investigated experimentally by Orr and Rows, who found that, if celloid in capsules containing bacterial cultures were placed, for instance, in contact with the sciatic nerve in a rabbit, degenerative changes subsequently developed in the corresponding posterior nerve roots at their point of entry into the spinal cord.

Before proceeding to the systematic description of the diseases of the nervous system, something must be said of its general anatomy and physiology, and of the clinical methods of investigating the symptoms which its diseases produce.

## ANATOMY AND PHYSIOLOGY

**Introduction.**—The nervous system, comprising the brain, brain stem, spinal cord and peripheral nerves, is the mechanism whereby the various motor and sensory functions are co-ordinated, and the individual is enabled to react to the environment as a unit. This integrative function is subserved by a complex system of neurons, whose full development is later than that of any other bodily system, both in the evolution of the species and the growth of the human embryo and child.

Each neuron consists of a cell body ; an elongated cylindrical strand, often of great length, known as the nerve fibre or axon, which gives off fine collateral branches ; and numerous other processes, shorter and finer than the axon, called dendrites. The cell body contains a nucleus and nucleolus, and after fixation with alcohol a granular substance, which when freshly stained with toluidin blue is arranged in characteristic aggregations known as Nissl's granules.

Each neuron is anatomically a complete unit, and the axon may be considered as a living branch of the protoplasm of the cell body. When cut off from the cell the peripheral part degenerates, and growth takes place from the central cut end only.

Functionally each neuron is in continuity with a large number of others by means both of its dendrites and its collaterals, which arborise around other nerve cells. Within the spinal cord and brain the neurons are supported and held together by a scaffolding of connective tissue peculiar to the nervous system known as neuroglia.

The axon when it leaves the nerve cell is surrounded for the whole of its course by a fat-like substance known as the myelin sheath. When an axon leaves the brain or cord to enter a peripheral nerve it acquires in addition a thin nucleated membranous sheath called the neurilemma. If an axon is severed, the neurilemma of the peripheral part performs an essential function in determining the down-growth of a new fibre from the central cut end, from which follows the most important fact that within the brain and spinal cord, where the axons have no neurilemma, once their anatomical continuity is severed there can be no hope of repair, whereas in the peripheral nerves complete anatomical restoration may be looked for.

The function of nerve fibres is to conduct the nervous impulse, and in the intact nervous system each nerve fibre normally carries impulses in one direction only.

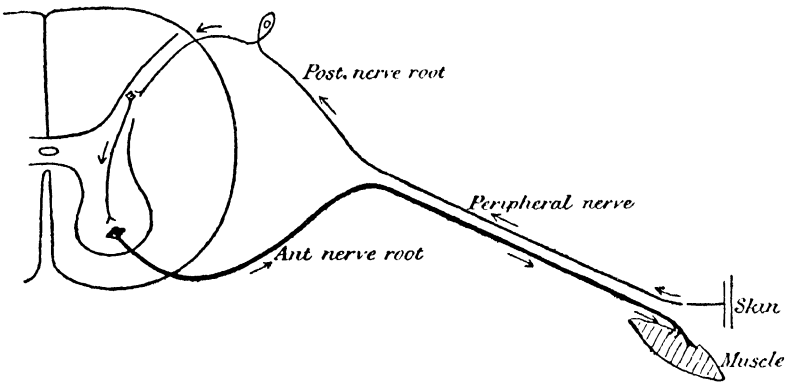


Fig. 60.—Diagram of a Simple Spinal Reflex. (Sir James Purves Stewart.)

It is therefore possible to distinguish between the fibres which conduct from the periphery toward the cord and brain, and are called *afferent* or centripetal, and those which conduct away from the higher centres toward the periphery, and are called *efferent* or centrifugal. There is also a third large group of fibres, whose function is to connect higher centres one with another; these may be efferent or afferent and are called association fibres.

The most peripheral *efferent* neuron is the *lower motor neuron*, with its cell body in the spinal cord or brain stem, giving rise to a nerve fibre which runs a more or less lengthy course in one of the peripheral nerves and ends in contact with a voluntary muscle in the motor end plate. The peripheral *afferent* neuron has its cell body in one of the posterior root ganglia, and is unique in possessing two elongated nerve fibres, one which conveys the impulse from the periphery to the ganglion cell and may be considered a specialised dendrite, the other, carrying on the impulse into and up the spinal cord, being the axon proper. At the periphery of these neurons there are special receptor end organs, which show considerable diversity of structure in accordance with their different selective functions. The function of a receptor organ is to lower the threshold of the afferent path for one kind of stimulus and to raise it for all others. The retina, for instance, reacts to light waves, but is protected by its anatomical position from all other stimuli; there are similar highly specialised organs for reception of sound waves and the differentiation of chemical stimuli in smell and taste, and also special receptors

throughout the body for pain, temperature, touch, and sensations of movement and posture. Of these most are situated on the external surface of the body, and are stimulated from without—the exteroceptors; while those adapted for receiving impulses from the muscles and joints are known as proprioceptors.

The selective function of the receptor end organ does not apply only to centripetal impulses destined to reach consciousness, but is shown in reflex mechanisms.

A reflex action may be defined in its simplest form as an exhibition of activity in an effector end organ, muscle or gland cell, in response to stimulation of a receptor end organ, the stimulus being conducted from receptor to effector through the central nervous system. The act is not voluntarily performed, though the individual may be conscious of it. The simplest reflex arc must consist therefore of at least two neurons, and, as a matter of fact, always includes at least one association fibre in addition (Fig. 60).

In the process of the development of the central nervous system there has been laid down a vast number of such reflex paths, beginning at the lowest level of complexity with those adapted for simple protective reactions, ranging up to complex mechanisms for the regulation of posture and gait, and at a higher level still instinctive behaviour reactions of the individual as a whole.

The selective function of the receptor in reflex action is to lower the threshold of excitability of the reflex arc for one kind of stimulus and raise it for all others. From this it follows that the type of reflex response elicited by stimulation of any one spot may be determined by the character of the stimulus employed.

In the decerebrate dog, for instance, in which reflex action has been carefully studied, if the pad of the foot be gently stimulated with the point of a pin or any other harmful stimulus the response is an immediate movement of withdrawal; if, however, firm upward pressure be made on the pad, such as occurs when the foot meets the ground in walking, the result is a vigorous extension of the limb. Here we have an example of two reflexes, the withdrawal reflex and the extensor thrust, elicited from the same area by different stimuli.

In man the simpler reflex mechanisms are normally obscured by the dominating picture of voluntary activity, but we have been able to study them in certain cases of injury and disease, especially during the late war.

Further consideration of reflex function will be delayed until the general anatomy and physiology of the brain and cord have been described.

**The Development of the Central Nervous System.**—In the segmented annelids, *e.g.* the common earthworm, from which the development of the mammalian nervous system can be traced, each segment forms a more or less complete unit with its sensory and motor neurons, the cell bodies of which are collected together in a mass of grey matter called the central ganglion. Even at this stage, however, the foremost or headward ganglia have begun to develop anatomical connections with, and functional control over, all the segments by means of longitudinal tracts, enabling the individual to react in some measure as a single unit.

In the early stages of growth of the mammalian embryo the neural tube consists of a mass of grey matter around the central canal, which is divided into a ventral lamina, containing the motor cells, and a dorsal lamina, into which grow from without the fibres from the posterior root ganglia.

In the course of further development function becomes more and more centralised at the cephalic end of the neural tube, with the result that the central core of grey matter becomes ensheathed within a mass of ascending and descending longitudinal nerve fibres, connecting the various segments of the cord with the cerebrum and cerebellum.

These latter structures are developed upon the dorsal surface of the originally segmented neural tube, and are conveniently included under the single term "supra-segmental mechanisms." In the cord the original arrangement is preserved

in so far as it is marked off into segments by the pairs of efferent and afferent roots, which correspond in number and distribution with the original segmentation of the whole body, so that there is a pair of nerves for each vertebra. In the trunk this distribution is easily made out, but is obscured in the limbs owing to the manner in which they have developed as lateral buds from the original segmented column.

**The Arrangement of Nerve Cells and Fibres in the Spinal Cord.**—The central column of grey matter is roughly H-shaped, being composed of two comma-shaped masses connected by a transverse commissure. In the anterior cornua are situated the large motor cells of the lower motor neurons.

The posterior cornua contain many smaller cells, whose axons follow one of four main courses :—

1. Some end in arborisations round the anterior horn cells either of the same or of the opposite side.
2. Others pass out into the white matter of the same or of the opposite side

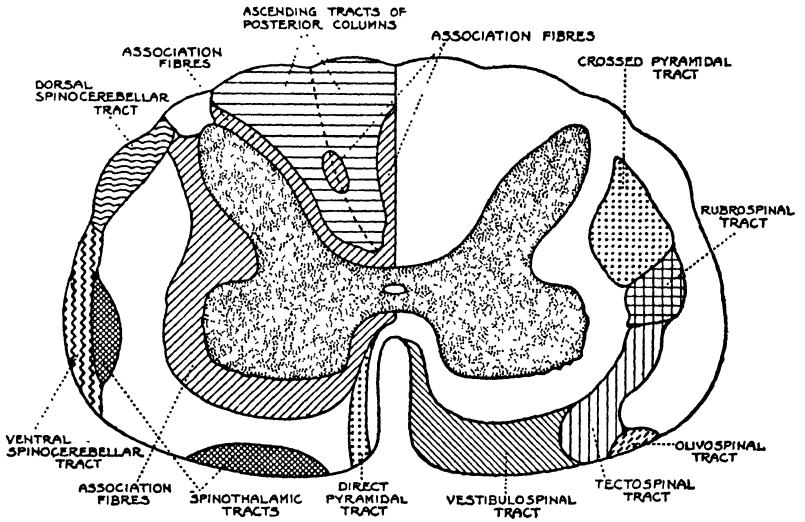


FIG. 61.—A Diagram to illustrate the Grouping of the various Tracts of Nerve Fibres in the Spinal Cord. (Modified from "Cunningham's Anatomy.")

of the cord to enter the grey matter again at a lower or higher level, thus connecting the various segments of the cord with one another.

3. Others, again, crossing in the grey matter to the opposite side of the cord, enter the white matter and are continued upward to the thalamus.
4. Others pass out into the white matter of the same side and go directly to the cerebellum.

Between the second thoracic and second lumbar segments and again at the upper cervical and lower sacral levels there is a third group of cells, known as the lateral horn, which gives rise to the pre-ganglionic fibres of the sympathetic nervous system.

The white matter of the cord is mainly composed of longitudinal bundles of myelinated nerve fibres. These may be grouped according to their direction and cell destination as follows :—

*Ascending Fibres.*—1. That part of the white matter contained between the posterior cornua is almost entirely composed of fibres arising in the posterior root ganglia at a lower level, and these fibres are known collectively as the

posterior columns. They continue upward uncrossed to the cuneate and gracile nuclei in the medulla.

2. The wedge-shaped area on either side lying between anterior and posterior cornua is called the lateral column. The ascending fibres therein are: (*a*) Those arising from cells in the posterior cornua of the same side and passing up to the cerebellum on the same side—the dorsal and ventral spino-cerebellar tracts. (*b*) Numerous fibres which, having arisen in cells of the opposite posterior horn and having crossed in the commissure of grey matter, ascend to the thalamus as the spino-thalamic tracts (Fig. 61).

*Descending Fibres.*—These have their cell bodies in the cortex cerebri or one of the important motor nuclei in the brain stem, and end in arborisations round the anterior horn cells at various levels.

1. The most important are the pyramidal fibres. Arising in the pre-central gyrus, the fibres from one side of the brain are gathered together to form a thick strand in the internal capsule, thence descend in the crus cerebri and pons, decussate almost completely in the medulla, and appear in the cord as the main crossed pyramidal tract in the lateral column and the small direct pyramidal tract to the inner side of the anterior cornua (Fig. 61).

2. The rubro-spinal tracts: Arising in the red nucleus, the fibres from one side at once decussate with those from the other, and pass downward in the lateral column ventral to the crossed pyramidal fibres.

3. The tecto-spinal fibres arise in the corpora quadrigemina, and undergo partial decussation before passing down the lateral columns in front of the rubro-spinal fibres.

4 and 5. The vestibulo-spinal fibres arise in Deiter's nucleus in the pons, and the olivo-spinal fibres in the inferior olive. Both these tracts pass down the cord uncrossed in the anterior part of the lateral column.

There is also physiological proof for the existence of fibres descending from the respiratory centre in the medulla to the motor cells innervating the diaphragm through the third, fourth and fifth cervical nerves. These are collected in the tractus solitario-spinalis.

*Association Fibres.*—These, running upwards and downwards, are scattered throughout the columns of longer fibres, and are especially aggregated in a continuous belt encircling the central grey matter, and on either side of the postero-median septum (Fig. 61).

*The Destination of the Longitudinal Fibres.*—All the descending fibres end, as has been said, in connection with the anterior horn cells. Tracing the ascending fibres upwards, we find those in the posterior columns ending in the gracile and cuneate nuclei in the medulla; thence new fibres arise and after decussating at once proceed upwards, forming a strand on either side of the mid-line dorsal to the pyramids known as the mesial fillet. These fillet fibres diverge from the mid-line as they ascend, and terminate in the nuclei of the optic thalamus; thence again a new set of fibres on either side proceeds upwards through the posterior part of the internal capsule and corona radiata to the cerebral cortex behind the Rolandic fissure.

The impulses conveyed by the spino-thalamic fibres, as their name implies, proceed up to the nuclei of the optic thalamus; but there is evidence to show that they do so by a series of relays through short association fibres rather than in continuous long tracts.

The fibres of the dorsal and ventral spino-cerebellar tracts pass to the cerebellar cortex of the same side, the former through the inferior, the latter by the superior, cerebellar peduncle.

Association fibres are still in evidence at the higher levels, one group, known as the posterior longitudinal bundle, being of great importance as connecting the nuclei of the vestibular nerve with those innervating the muscles of the eye and head and neck.

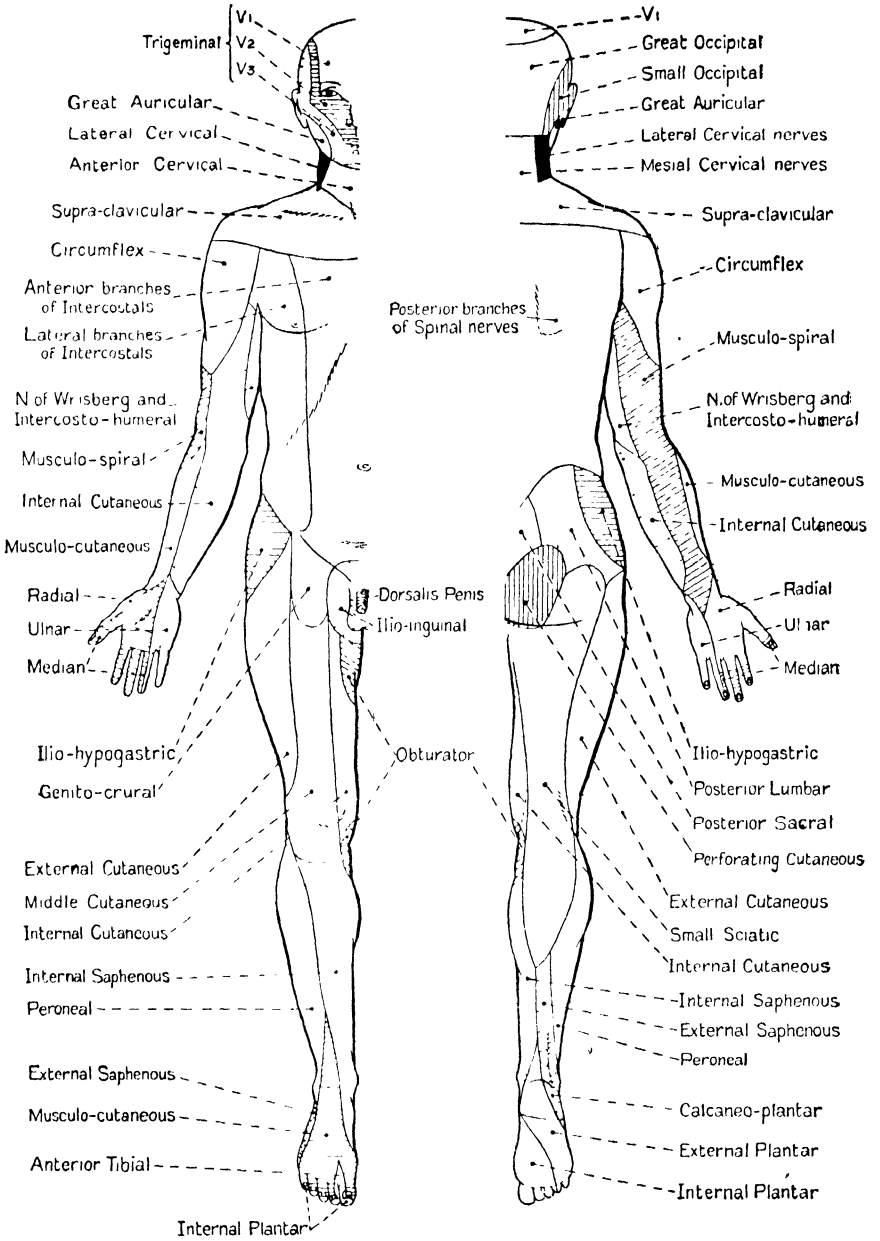


FIG. 62.—A Diagram to illustrate the Cutaneous Distribution of the Principal Nerves. (Sir James Purves Stewart.)

*Differentiation of Function among the Centripetal Fibres.*—In the peripheral nerve the various fibres are intermingled without regard to their different functions, but at a short distance from the cord the motor and sensory fibres become completely separated, when the latter pass into the posterior root ganglion and thence onwards into the cord itself in the posterior root.

On the cutaneous surface of the body it is possible to map out the sensory distribution of nerves and roots with accuracy, and it is found that on the trunk the area of skin supplied by an intercostal nerve corresponds with that innervated by the posterior root. In the limbs, however, owing to the way in which they have budded out with the formation of nerve plexuses, it is found that several nerves may contribute fibres to a single posterior root. It is therefore necessary to make out two separate charts illustrating the cutaneous distribution of peripheral nerves and posterior roots respectively, the latter being known as the segmental chart (Figs. 62 and 63).

In the posterior root all fibres, whether attached to receptors for touch, deep pressure, pain, temperature, or sense of position and movement, are gathered

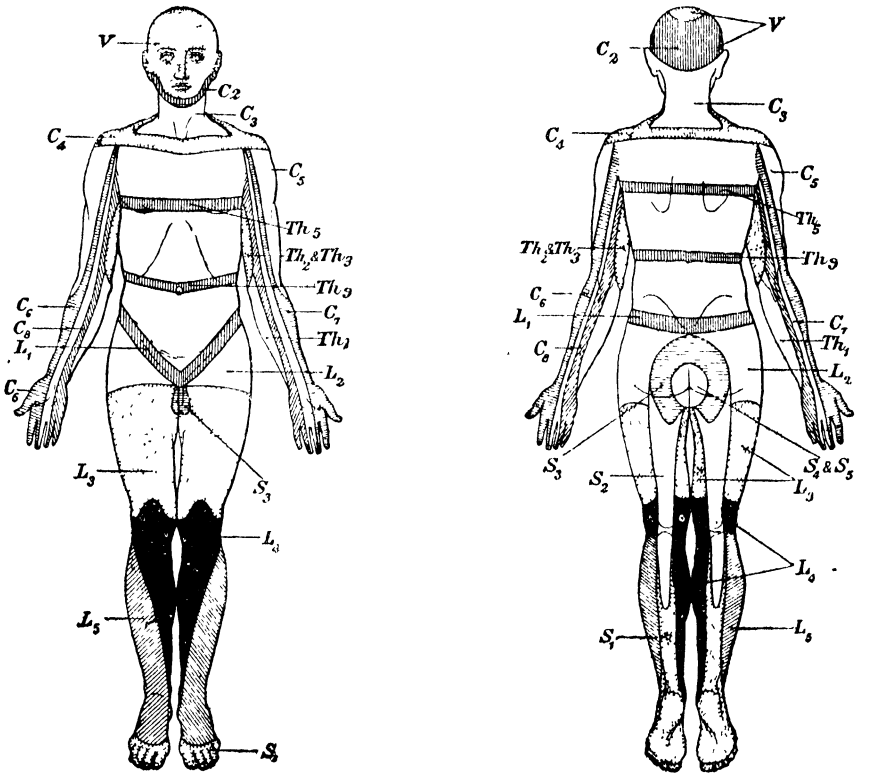


FIG. 63.—A Diagram to illustrate the Areas of Skin supplied by the Posterior Nerve Roots. (Sir James Purves Stewart.)

together. Immediately they have entered the cord a segregation of fibres takes place. Of the fibres conveying impulses of sense of position and movement, for instance, some are destined to make simple reflex connections in the spinal cord, others to convey their impulses through relays to centres in the cerebellum and mid brain for the regulation of postural tone and co-ordination, others through longer relays to carry to the cerebral cortex sensations of spatial recognition.

At this level the afferent fibres may be divided into (1) those making reflex connections through association fibres with motor cells in the cord; (2) those making similar reflex connections at the cerebellar or mid-brain level; (3) those conveying impulses destined to reach the cerebrum.

1. The function of those fibres which connect with anterior horn cells in the cord will be considered briefly under the section dealing with spinal reflexes.

2. Many root fibres end in the cells of Clarke's nucleus at the base of the posterior horn, whose axons pass into the cord of the same side to form the dorsal and ventral spino-cerebellar tracts.

The impulses carried by these are not destined to reach consciousness, but form links of the afferent chain in the important reflex arcs governing postural tone and the co-ordination of all muscular movements.

3. A large number of the fibres from each posterior root proceed uncrossed directly up the cord in the posterior columns. The fibres from the lowest segment

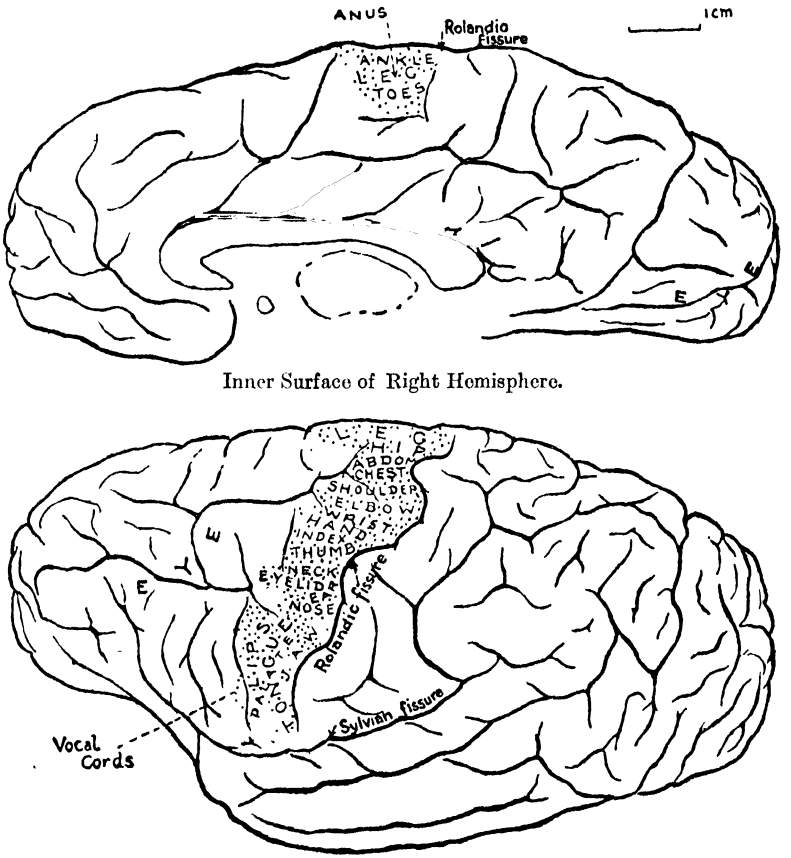


FIG. 64.—The Excitable Motor Cortex of a Gorilla as mapped out by means of Electrical Stimulation by Leyton and Sherrington (1917).

lie against the posterior median septum; those from the next form a layer external to these, and so on, so that in the cervical region the fibres from the sacral segments lie buried deeply in the posterior columns and may sometimes escape injury from compression when the function of those from a higher level is in abeyance. The posterior column fibres conduct sensations of position and passive movement, deep pressure, vibration and some tactile sensation, including tactile discrimination.

Other root fibres end around those cells in the posterior cornua which have been already considered as sending their axons across the grey commissure to form

the spino-thalamic tracts. Sensations of pain, heat and cold, and contact are carried up by these paths.

All the chains of fibres of group 3 after passing through various relay stations end around the cells of the optic thalamus on the side of the brain opposite to that on which they originally entered the cord.

Here, according to Head, there is a redistribution of sensory impulses on a new basis; fibres mediating crude sensations of contact, pain and temperature end

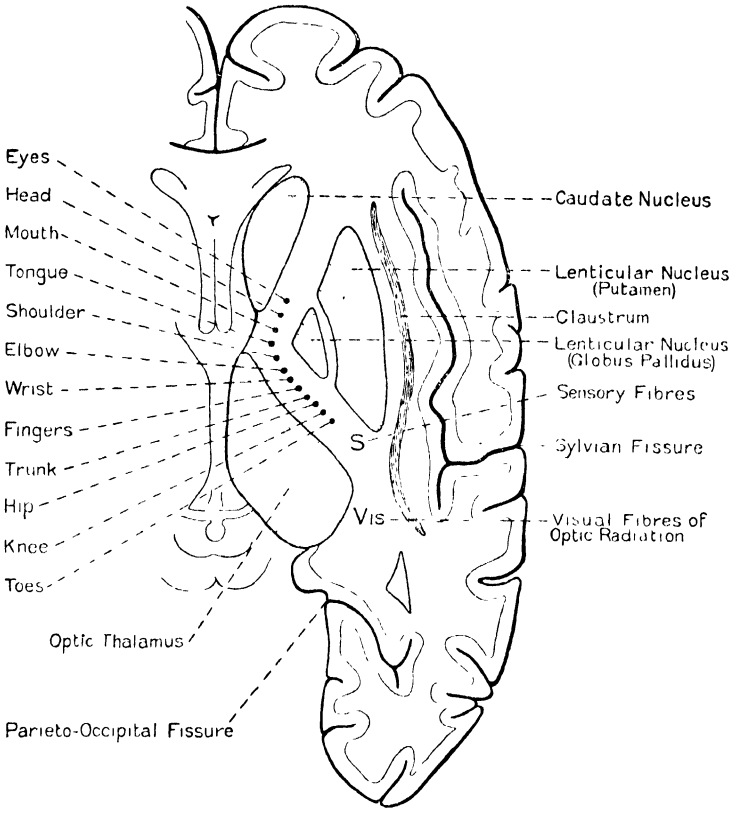


FIG. 65.—A Horizontal Section through the Right Cerebral Hemisphere showing the positions of the various Strands in the Internal Capsule. (Sir James Purves Stewart.)

in the thalamus, which is thus considered to be the seat of conscious appreciation of these forms of sensation.

Impulses of finer sensations are relayed onward to the cerebrum by way of the internal capsule and corona radiata, and spreading out, end in the cortex in the following arrangement:—

Those fibres carrying sensations related to the conscious appreciation of the position in space of the limbs and surface of the body end in the cortex of the Rolandic fissure; behind these in the post-central gyrus are collected impressions of difference and similarity in the size, shape, weight and texture of objects; and behind these again, as far back as the superior parietal and supra-marginal gyri, end fibres conveying impressions of differences in the intensity of individual stimuli, whether of touch, temperature or pain.

*Differentiation of Function among the Centrifugal Fibres.*—The *pyramidal* fibres

arise as the axons of the large pyramidal motor cells in the cortex of each precentral gyrus, and form the path whereby voluntary movements are normally executed, the right half of the brain governing the left half of the body, and *vice versâ*. Experimental stimulation and ablation has made it possible to construct a map illustrating the distribution of centres for movements of different parts of the body (Fig. 64). It is to be noted that the centre lying furthest forward is that which when stimulated results in conjugate deviation of head and eyes to the opposite side. It has been shown that the pyramidal fibres are not all motor in their function, for cortical stimulation in appropriate areas will produce inhibition of muscles already in a state of voluntary contraction.

The pyramidal tracts, whose further course has already been described, are non-medullated at birth and develop their myelin sheaths during the first two years of life; they carry all the voluntary impulses down to the anterior horn cells which set these into phasic activity. In the internal capsule they are arranged as shown in Fig. 65. The motor fibres for the leg and arm occupy the anterior two-thirds of the posterior limb, the fibres for the tongue and mouth are at the angle, and those for the face just in front. Behind the motor fibres are situated the afferent sensory fibres on their way up to the cerebral cortex, and behind these, again, the optic radiation, conveying visual impulses from the lower visual centres to the occipital lobe. Reference to the diagram will make it clear that in the case of a minute lesion in the internal capsule paralysis of the leg is more likely to be associated with sensory loss than that of the arm, and that a single lesion cannot produce paralysis and hemianopia without giving rise also to loss of sensation.

A further point in connection with the pyramidal fibres is that the anterior horn cells in the nuclei of the cranial nerves are supplied from both sides of the brain, and this supply is completely bilateral in the case of all the nuclei except those of the seventh and twelfth pairs. It follows that from a lesion of the internal capsule, even if it be severe enough to produce complete paralysis of the arm and leg, there will result no weakness in the domain of the cranial nerves except in the face and tongue on the side opposite to the lesion.

Of the other important centrifugal tracts we have little sure knowledge save of the *cerebello-rubro-spinal pathway*. Fibres taking origin in the dentate nucleus of one side of the cerebellum decussate in the superior cerebellar peduncle to reach the red nucleus of the other side; thence new fibres arise to form the rubro-spinal tract, which, decussating almost at once with its fellow, appears in the cord in front of the pyramidal tract where we have already seen it. Thus on the efferent as well as the afferent side each lobe of the cerebellum is connected with the same side of the cord. The function of the rubro-spinal tract will be discussed under Reflex Mechanisms.

From the lenticular nucleus of each side fibres descend to the red nucleus, so that there is formed a *lenticulo-rubro-spinal pathway* by means of which each lenticular nucleus is connected with the opposite half of the cord. The function of these fibres appears to be connected with the maintenance in the neuromuscular mechanism of that poise and equilibrium in the relation of antagonistic muscles to one another which is the essential basis for voluntary movement. Lesions of the lenticular nuclei are associated with "global" rigidity of agonists and antagonists together, and involuntary tremor of a peculiar type, which will be referred to again under Paralysis Agitans.

The *vestibulo-spinal* fibres form links in the efferent chain of reflex arcs concerned in the adjustment of the musculature of the whole body to the position of the head in space, and the *tecto-spinal* tracts play a similar part in the performance of reflex actions conditioned by visual and auditory stimuli.

Consideration of the course and functions of the longitudinal fibres will make it easy to understand that in the case of a complete transverse section of the spinal cord from injury or disease there results total loss of power and sensation

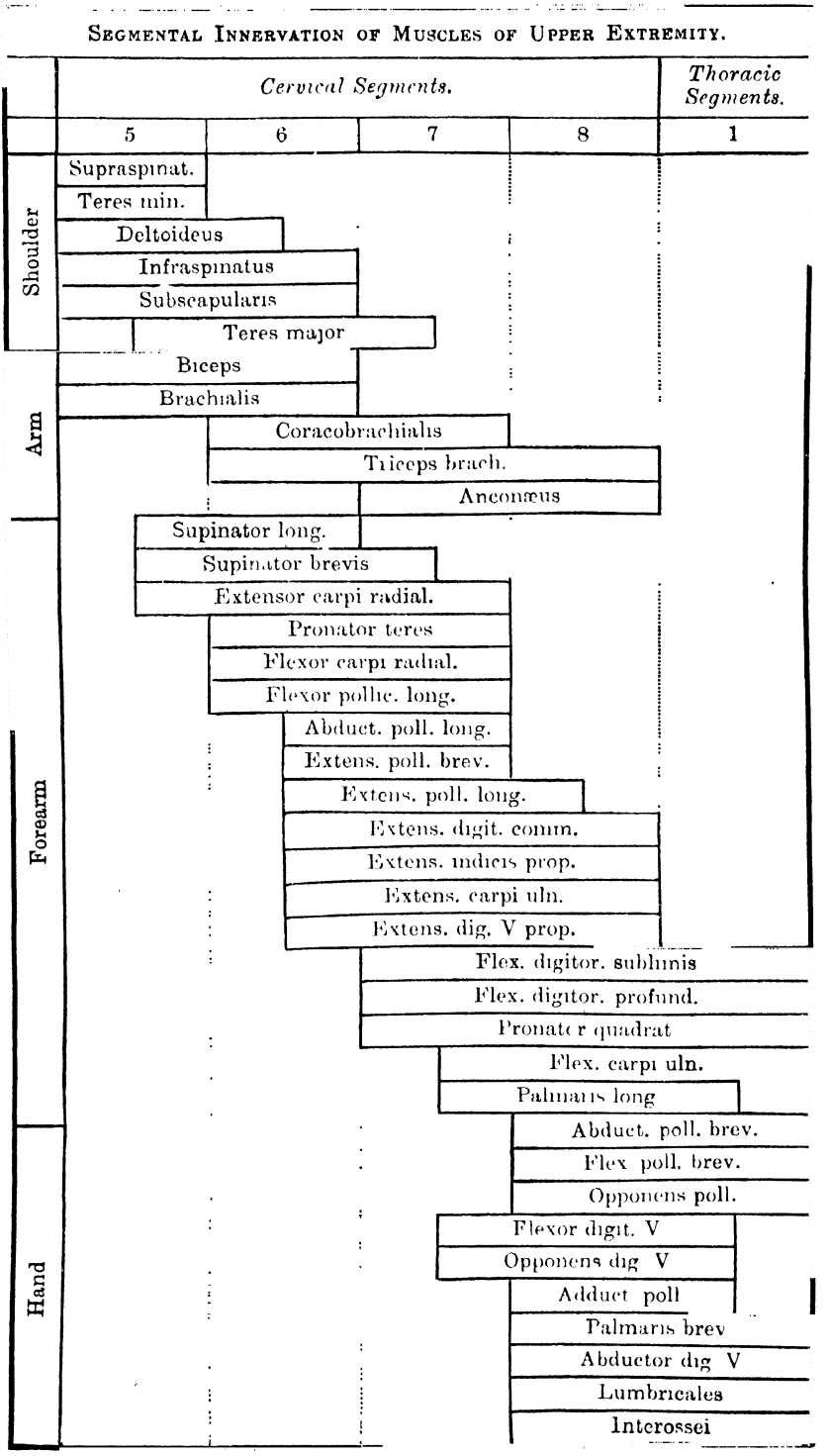


Fig. 66.—Table showing Segmental Innervation of Muscles of Upper Limb. (Bing.)

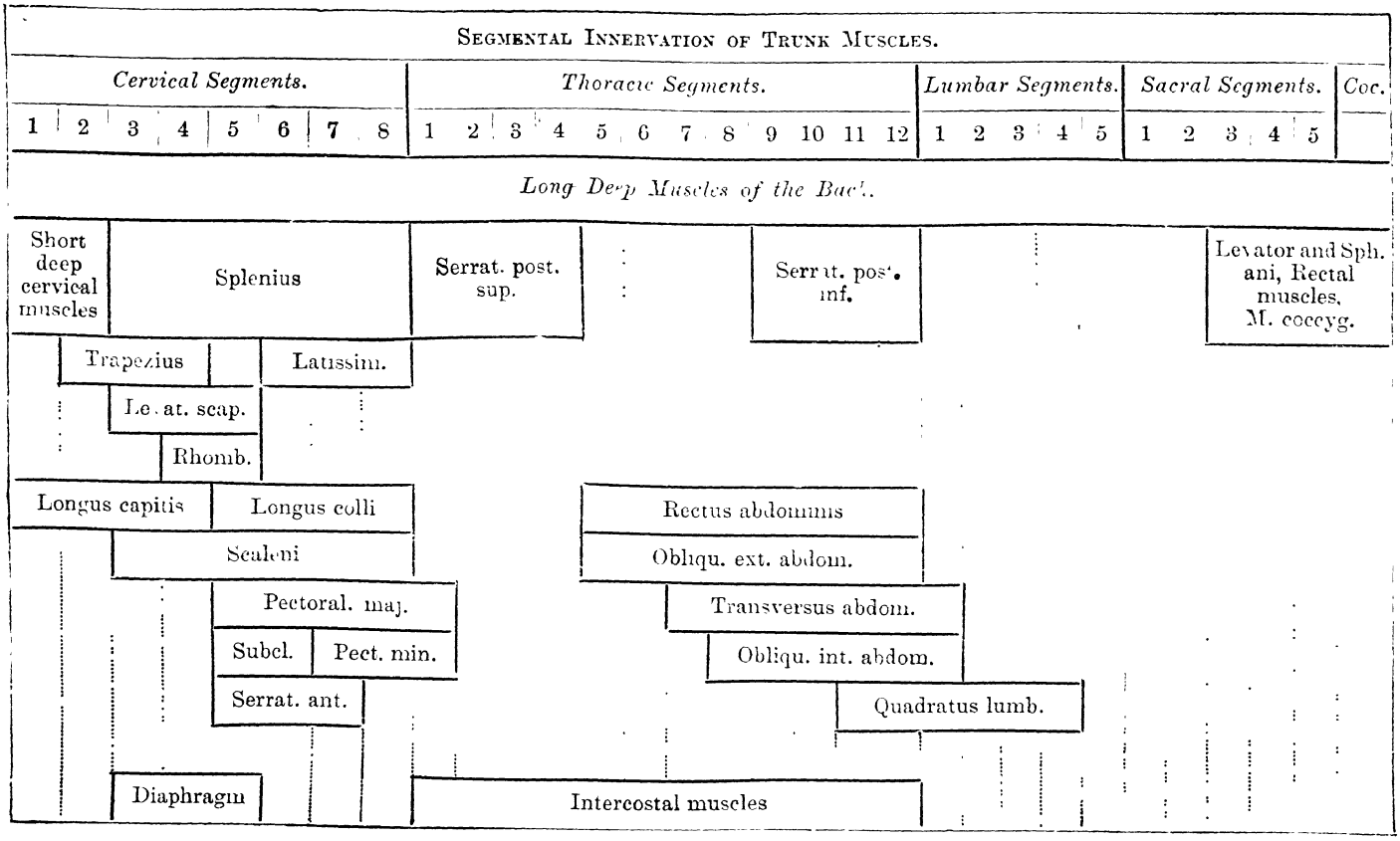


Fig. 67.—Table showing Segmental Innervation of Muscles of Trunk. (Bing.)

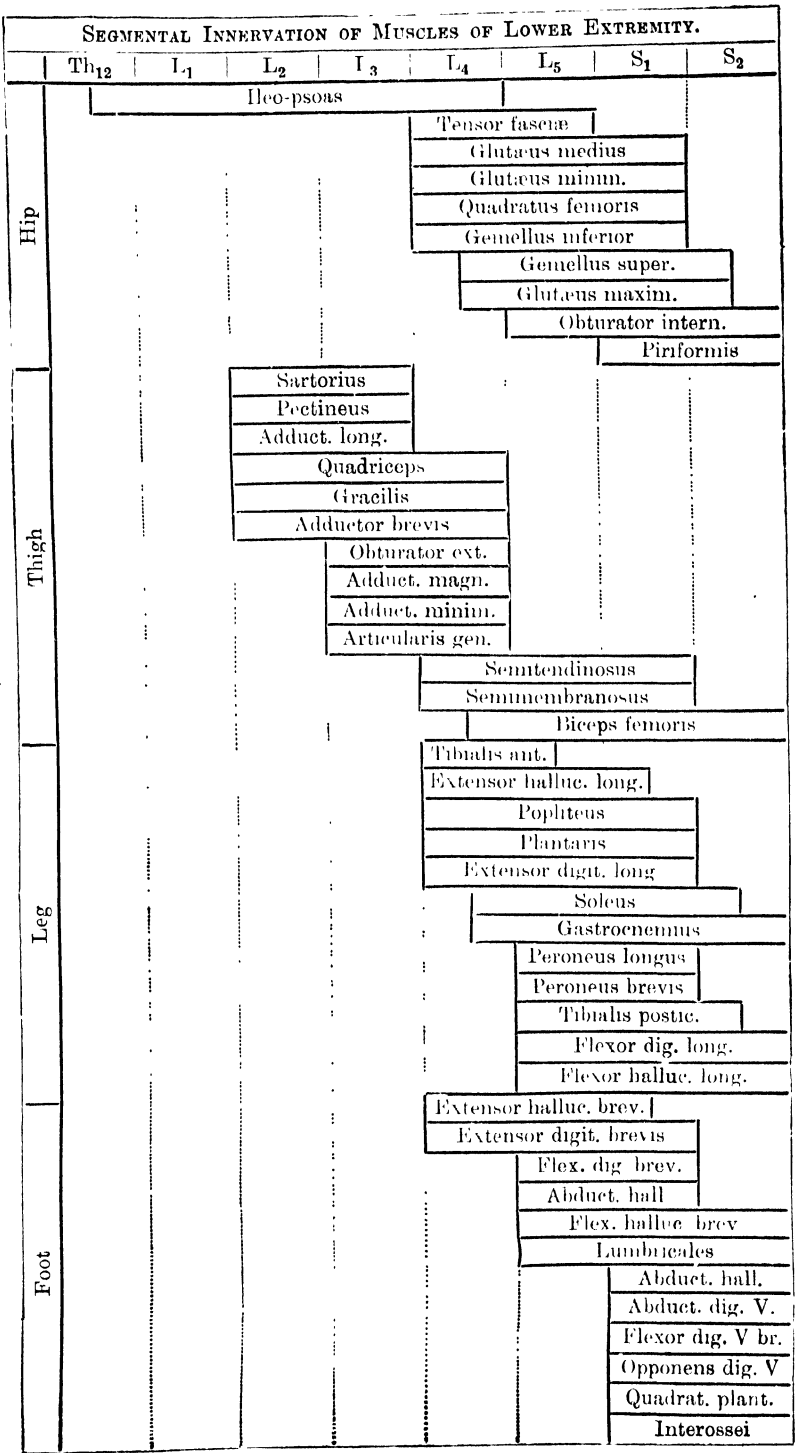


Fig. 68.—Table showing Segmental Innervation of Muscles of Lower Limb. (Bing.)

below the segmental level of the lesion. In the case of hemisection of the cord there results the following picture: On the same side as the lesion there is loss of power, sense of position and passive movement and vibration sense; cutaneous sensibility is intact except for tactile discrimination; on the opposite side there is loss of cutaneous sensibility to touch, pain and temperature. This is known as the *Brown-Séquard syndrome.*

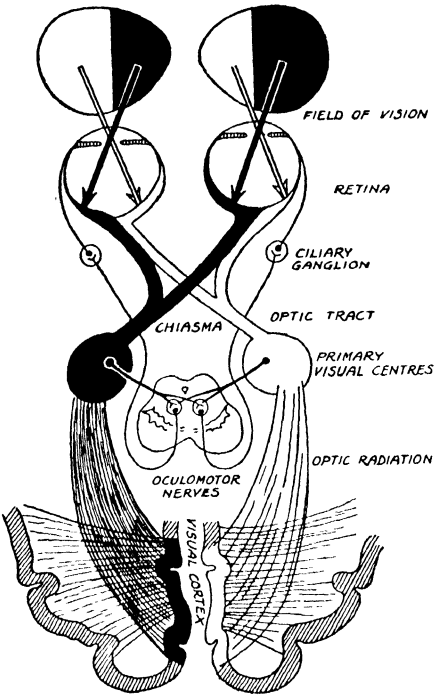


Fig. 69.—Diagram to illustrate the Course and Connections of the Visual Nerve Fibres. (*After Bing.*)

The lower motor neurons represent the final common paths for all motor and inhibitory impulses, whether in voluntary movement, tonic contraction, or phasic reflex action. The axons of the motor cells leave the cord by the anterior roots, and in the upper thoracic region the segmental distribution to the muscles is well preserved. In the limbs, however, where the segmental arrangement of the musculature is lost, with the formation of nerve plexuses there is a rearrangement of fibres in the peripheral nerves so that a single muscle is innervated from several segments of the cord. When a muscle is cut off from its motor nerve supply not only does it become paralysed for all kinds of movement, but other changes take place. Fibrillary twitchings appear in it, after a brief period it loses its excitability by the faradic current, and shows only a sluggish response to galvanism, and eventually its fibres undergo atrophy. From what has already been said concerning the muscular distribution of anterior root fibres it follows that, whereas section of a peripheral nerve will give rise to complete paralysis and atrophic changes in the group of muscles supplied by it, a lesion confined to a single segment of the cord or one anterior root will result in partial weakness and atrophy of a larger number of muscles (Figs. 66—68).

**The Special Senses.**—*Vision.*—From the retina, where they connect with the special light receptors, the rods and cones, the optic fibres pass back through the optic nerves to the optic chiasma. Here they undergo partial decussation in such wise that the fibres from the right halves of both retinæ continue backwards in the right optic tract. This means that the right optic tract conveys impulses from the left visual field, and *vice versâ.*

The fibres of each optic tract end in cells of the posterior end of the thalamus, the lateral geniculate body and the superior corpus quadrigeminum of the same side (*primary visual centres*): hence new fibres forming the optic radiations curve first a little forwards, then outwards across the posterior part of the internal capsule, finally backwards around the posterior horn of the lateral ventricle to reach the occipital cortex on either side. It will readily be seen from a reference to the diagram how a lesion of one optic nerve will cause blindness of the eye on the same side, whilst a lesion of the optic tract, optic radiation, or, if it be extensive enough, a lesion of the occipital cortex will cause loss of perception in the opposite half of the visual fields (Fig. 69).

Immediately behind the chiasma the fibres from the macula of each eye which subserve central vision form a wedge-shaped bundle on the mesial aspect of the optic tract. It will be seen then that an expanding tumour in the posterior angle of the chiasma, while eventually producing bitemporal hemianopia, may in the first place give rise to a central scotoma in one or both eyes.

From the primary visual centres of either side fibres connect with that part of each oculomotor nucleus which innervates the iris, completing the path for the light reflex. Since the primary visual centres of each side are connected with both oculomotor nuclei, it follows that both pupils will react to illumination of one eye. The reflex centres for accommodation appear also to be situated in the region of the superior corpora quadrigemina.

*Smell and Taste.*—The complex paths by which these sensations are carried to the brain from the specialised receptors in nose and tongue will not be discussed here. Of clinical importance is the fact that there are centres for these special senses in the uncinate gyrus of either side, lesions of which are associated with peculiar subjective sensations in one or other sphere.

*Hearing.*—The fibres from the cochlear apparatus on either side enter the cranial cavity in company with those from the vestibule and the fibres from the facial nerve, with which they are in close relation during their short intracranial course, so that a lesion hereabouts is likely to affect the functions of all three. Entering at the junction of pons and medulla, the cochlear fibres end in the dorsal and ventral cochlear nuclei, whence their impulses are at once conducted by relays across the mid-line, and up in the lateral fillet to the inferior corpus quadrigeminum and mesial geniculate body. Thence a new set of fibres conveys the auditory sensations to the superior, and upper part of the middle, convolutions of the temporal lobe. The inferior corpora quadrigemina are closely connected with one another, which probably results in impressions from each cochlea being carried to both temporal lobes.

**The Three Reflex Levels.**—We may now discuss briefly the functions of reflex activity at the three levels of (1) spinal cord, (2) cerebellum, mid-brain, pons and medulla, and (3) cerebrum.

1. *Reflex Activity in the Spinal Cord.*—This is much obscured in normal man owing to the domination of higher mechanisms, but a study, in cases of proved transection of the cord, of the reflexes of the limbs below the level of the lesion has brought out the following important points :—

Following such a lesion if cystitis and sepsis be avoided and the period of spinal shock be survived, after an interval varying from a few days to three weeks, while there remains permanent and complete loss of power and sensation below the level of the transection, certain reflex activities appear in the lower limbs. The most characteristic of these is a bilateral movement of both limbs comprising upward movement of the toes, dorsi-flexion at the ankle and flexion at knee and hip joints. This is at first best elicited from the sole of the foot by nocuous stimuli such as pin pricks, but later the threshold of the reflex becomes lowered, so that the least touch may produce a response, and at the same time local signature is lost, so that stimuli anywhere below the level of the lesion may be effective.

This is regarded by Head and Riddoch as a movement of withdrawal, a response with a protective purpose, of which the upgoing movement of the great toe long known as the sign of Babinski or extensor plantar response is an integral part.

Of very great clinical importance is the fact that this mass reflex, so called, facilitates the evacuation of the bladder. During the period of spinal shock immediately following the lesion there is paralysis of the bladder wall and tonic contraction of the sphincter, but as this passes off the bladder acquires an automatic activity; that is to say that when the pressure of its contents reaches a certain point its walls contract and expel the urine. Head and Riddoch have shown that the elicitation of the mass reflex facilitates and reinforces this

contraction, so that if the time be gauged when the bladder is about to empty itself automatically an appropriate stimulus will evoke at the same time as the mass reflex a contraction of the bladder powerful enough to expel its contents completely (facilitation reflex). The patient himself can learn in time the appropriate interval at which to provoke the emptying, and elicit the reflex by pinching the skin of his own thigh.

The mass reflex is a phenomenon of the activity of spinal centres entirely removed from higher control. It is a momentary reaction; after it has been elicited the limbs fall back upon the bed from their own weight, unless the threshold for the reflex is so low that the limbs are constantly retained in a position of flexion as the result of repeated stimuli, in which case contractures are apt to occur.

2. *Reflex Centres in the Cerebellum, Mid-brain, Pons and Medulla.*—Situated in the lower part of the mid-brain, the cerebellum, and the upper part of the pons is a group of motor centres whose main function appears to be the maintenance and regulation of postural tone. The functions of these centres have been mainly studied in animals, but there is evidence that similar mechanisms exist in man.

If in a cat a transection is made across the brain stem at any point below the superior quadrigemina and above the level at which the eighth nerves enter the pons, there ensues extensor rigidity in all four limbs, a condition which has been described by Sherrington as one of the features of *decerebrate rigidity*. This appears to be due to the uncontrolled activity of motor centres which have been released from higher inhibitory influences as the result of the experimental lesion. That this tonic rigidity is an expression of reflex activity is proved by its disappearance after section of the posterior nerve roots, but the further course within the central nervous system of the pathways for the reflex arc is at present unknown. Recent work, however, has shown that the red nuclei and rubro-spinal tracts do not, as was previously thought, play any part in the development or maintenance of decerebrate rigidity.

In rare instances phenomena closely resembling those of decerebrate rigidity in animals have been observed in man following injury or disease of the upper part of the mid-brain.

The condition of spastic rigidity which develops in man following paralyses due to lesions of the pyramidal fibres is in many ways akin to the tonic contractions of decerebrate rigidity, and may also be considered as an expression of the activity of lower motor centres released from cerebral control.

Thus in a man with an incomplete lesion of the spinal cord the toes point downwards, the feet are plantar-flexed, the knees and hips rigidly extended, the corresponding muscle groups being maintained in tonic contraction. Since the spinal centres are also in a measure released from control by damage to the pyramidal tracts, the flexion reflex may be elicited from either leg singly, especially by nocuous or semi-nocuous stimuli to the sole of the foot. The character of the movement when elicited is similar to that obtained from the spinal preparation, an upgoing movement of the great toe with more or less contraction of the hamstrings and other flexor groups. But, the removal of control being incomplete, there is no mass reflex; local signature is preserved, the reflex is confined to the limb stimulated, the facilitation of the bladder evacuation is not obtained, and the limb is brought back sharply to its original position of tonic extension.

A lesion of the pyramidal fibres before their decussation gives rise to tonic contraction or spasticity on the opposite side of the body, the cause presumably being the same—uncontrolled and unbalanced activity of the tonic reflex arc whose centre is in the mid-brain. In the case of the upper limb in man the tonic contraction is mainly of the flexors, and the arm is typically held adducted, with elbow semiflexed, forearm pronated and wrist and fingers flexed. A unilateral lesion of the cord below the decussation of the pyramids

will naturally produce rigidity in the limbs below the lesion on the same side of the body.

The significance of the restriction of rigidity to certain muscular groups following injury to pyramidal fibres is somewhat obscure. Sherrington considers it in decerebrate animals to be an exhibition of postural tone, the muscles innervated being those employed to counteract the forces of gravity. This explanation can hardly hold for the position of the arm in man, and the whole problem needs further elucidation.

The part played by the cerebellum in the maintenance of postural tone is somewhat uncertain. On the one hand, it has been made clear by experimental work that the cerebellum is not primarily responsible for the development of decerebrate rigidity in animals, since its ablation is not followed by any diminution of the tonic contractions of this condition. On the other hand, one of the results of a destructive lesion of the cerebellum in man is a general loss of tone in the affected limbs, which in consequence are more easily displaced from a position of rest or manipulated by the observer. Again, after the elicitation of a tendon jerk, if the limb is hanging freely, it swings to and fro like a pendulum for some little time before coming to rest (pendular knee-jerk).

The cerebellum, therefore, probably regulates postural tone without being actually responsible for its maintenance. In addition to this, it plays a dominant *rôle* in the co-ordination of muscular movements. Receiving on the afferent side proprioceptive impulses from all parts of the body, and from the labyrinths (the specialised proprioceptive sense organs), and being connected on the efferent side with motor cells at every level, including those of the pre-central cortex, it plays an essential part in the setting of every motor act.

For the smooth and orderly performance of any voluntary movement, still more in a series of such movements, many muscle groups come into play besides the prime movers or agonists. When, for instance, a man with his elbow to his side touches the tip of his nose with the forefinger, the prime movers are the flexors of the elbow, but the antagonistic triceps also plays a part in maintaining the smoothness of the movement by relaxing in perfect time with the contraction of the flexors, and, again, at the end of the movement checks it nicely by coming into balanced action at the appropriate moment. Besides the triceps the muscles of the shoulder girdle by fixation of the shoulder joint ensure that the movement of finger tip to nose shall take place in a straight line. Moreover, the biceps, which is one of the prime movers in this action, has by reason of its insertion a powerful supinator action as well as flexor, so that the pronator radii teres must exert a force just strong enough to oppose the supinating element, this being an example of what is called synergic action. The cerebellum is normally responsible for the co-ordination of all these muscular mechanisms, each lateral lobe governing its own side of the body.

So in the case of a man with a lesion of the right lobe of his cerebellum who attempts with his right arm the movement we have just described we see that the movement is jerky (intention tremor) from irregular relaxation of the antagonist; the tip of the finger describes a zigzag course owing to imperfect fixation of the shoulder, the hand is unduly supinated due to failure of synergy, and when the finger reaches the nose it strikes it with clumsy force owing to the lack of the timely checking action of the triceps.

These defects in the co-ordinating mechanism may be shown in a variety of ways in lesions of the cerebellum or its superior peduncles, and are often manifest in the muscles concerned with articulate speech. Instead of the normal smooth sequence of syllables, there is an awkward pause between each (scanning speech), and when the sound does come it is produced with explosive force. Together with inco-ordination, we find in a cerebellar lesion lack of muscular tone on the affected side, as we should expect, seeing that there is a break in one of the reflex arcs governing postural tone.

The fibres of the vestibular nerves entering the lower border of the pons convey impulses from the semicircular canals indicating changes in the position of the head in space. The vestibular apparatus thus forms the specialised proprioceptive sense organ of the head. The vestibular nerve fibres on either side end in the cells of Deiter's nucleus, which is connected with the anterior horn cells generally by means of the vestibulo-spinal tract, and especially with the nuclei innervating the muscles of the eyes and head and neck. The connections with the nuclei for the eye muscles are made through the posterior longitudinal bundle, which lies immediately ventral to these nuclei beneath the floor of the fourth ventricle and the aqueduct of Sylvius.

Under normal conditions continually incoming impulses from the labyrinths of both sides are translated in the reflex centres outlined above into impulses of tonic innervation to both sides of the body equally; the function of each labyrinth seems to be to direct its half of the body towards the middle line. When the labyrinth on one side is destroyed there ensue from the unopposed activity of the other labyrinth deviation of the eyes and inclination of the head towards the side of the lesion, with a tendency for the whole body to fall towards the same side. Supposing that it is the right labyrinth which has been destroyed, and an attempt is made by the patient to fix his eyes on an object to his left, the effort is momentarily successful, but there quickly ensues a slow deviation to the right again, due to the unbalanced action of the left labyrinth; this is almost immediately corrected by a quick voluntary movement back to the left, and if the effort is maintained, a rapid series of oscillations is kept up consisting of alternate slow movements to the right and quick movements to the left. This phenomenon is known as nystagmus, and the two movements of which it consists are called the slow component and the rapid component.

That the tendency for head, eyes and body to deviate towards the side of the lesion is due to the unbalanced action of the other labyrinth may be proved in animals by its experimental ablation when the normal poise is regained. This experiment also proves that in animals there are other mechanisms which can compensate for the loss of both labyrinths.

In patients suffering from middle ear disease it may be that one canal only of one labyrinth is destroyed, usually the external semicircular canal, which is most exposed to infection from the middle ear. This results in nystagmus of a type varying with the canal affected. A lesion of the external canal causes horizontal nystagmus, of the superior canal rotary nystagmus, and of the posterior canal vertical nystagmus. Interruption of the reflex arcs at any point between the semicircular canals and the eye muscle nuclei may cause nystagmus, and it is readily seen that lesions of the pons or mid-brain are especially liable to produce this symptom.

The vestibular nucleus of either side is also intimately connected with the corresponding lobe of the cerebellum, which plays the most important part in the co-ordination of all proprioceptive impulses for reflex action. The exact connections and the function of the cerebellum in this respect are not quite clear. A lesion of the right lobe of the cerebellum results in a tendency for the body to deviate towards the right and for the head to incline in the same direction, but causes head and eyes together to rotate towards the opposite side, so that the occiput is approximated to the shoulder of the side of the lesion, and maximum nystagmus is obtained on fixation towards the side of the lesion.

Situated in the corpora quadrigemina, there are also centres for reflex movements depending on the afferent side upon impulses carried by the optic and auditory nerves, of which the pupillary light reflex and the involuntary blinking of the eyes at a loud and unexpected noise are examples. By means of the tecto-spinal tracts these centres are connected with the general bodily musculature, providing for the establishment of more generalised reflexes of a similar order.

Finally, we have to consider here the reflex centres in the medulla. The most important are those concerned with the regulation of the cardio-vascular apparatus, the respiratory movements, and the functions of the alimentary canal. The vagus provides the pathway for both afferent and efferent limbs of these reflex arcs, and the cell stations are situated in the sensory and motor nuclei of this nerve, which lie in the floor of the fourth ventricle. Pressure upon the medulla such as is caused most commonly by cerebral tumours interferes with the functions of these visceral reflex arcs, giving rise to slowing of the pulse, Cheyne Stokes breathing, and vomiting. In such cases death usually occurs from cessation of respiration.

3. *Reflex Function in the Cerebrum.*—Although the phenomena of cerebral activity do not come strictly within the definition of reflex action given already, since they are largely exhibited in voluntary actions, the function of the cerebral centres is strictly analogous to those which we have already considered in the spinal and mid-brain regions. Those phenomena which constitute in the main the behaviour of an individual are essentially exhibitions of activity in effector end organs in response to the stimulation of receptor end organs, the stimuli being conducted from receptor to effector through the cerebrum. Just as the cerebellum and vestibular nuclei perform the function of co-ordination of proprioceptive stimuli in relation mainly to tonic motor action, the cerebrum co-ordinates stimuli received from without in relation to phasic motor activity. An individual deprived of the function of all his exteroceptive end organs would be incapable of behaviour in relation to his environment. Anatomically and physiologically the cerebrum possesses features distinguishing it from the centres at lower levels. The reflex mechanisms considered in the preceding paragraphs represent innate and invariable modes of reaction, whereas the infinitely complex association fibres of the cerebrum provide for individually acquired and variable modes of response of the order we are accustomed to associate with intelligence. As we ascend the animal scale we find, side by side with the progressive dominance of intelligent behaviour reactions, an increase in bulk of the cerebrum as compared with the lower centres. In man the most remarkable feature of cerebral activity is the development of consciousness and conscious memory.

Owing to the extreme complexity and number of association fibres, every part of the cerebral cortex is connected directly or indirectly with every other part, so that an external stimulus by spreading along paths whose resistance for it is lowered by previous experience may provoke a reaction out of all proportion to its intrinsic significance. The result of such a reaction will normally be to provoke the reception of further external stimuli, and so the whole series of reactions is initiated in which we see the behaviour of the individual as a whole in response to his environment. When we speak of paths whose resistance is lowered by previous experience we are merely using illustrative terms to express the phenomena observed in processes of learning. It is found that an animal if confronted with the solution of a problem standing between it and food, will quickly proceed to form a habitually correct mode of response. The particular combination of stimuli received in this situation initiates impulses which, travelling time after time through the same groups of association fibres, produce the same series of motor responses. Human behaviour is largely conditioned by the formation of innumerable habits acquired in this manner, and in this connection it is to be remembered that inhibition is just as important a mode of reflex response as motor action. Underlying all this manifold activity are sources of potential energy depending upon we know not what biochemical or physical causes, connected in some way still obscure with the secretions of the ductless glands; these are the instincts and emotions, and will be considered further in relation to the psychoneuroses.

The complexity of human behaviour as exhibited by cerebral activity depends

then upon the complexity of different combinations of stimuli, the previous formation of habits (which in its turn must depend to some extent on the inherited pattern of the association fibres) and the individual potency of the emotions.

**Speech.**—Reference has already been made to the localisation in the cortex of the endings of fibres conveying afferent impulses to it, and of the motor cells, from which arise voluntary motor impulses. There remains to be considered

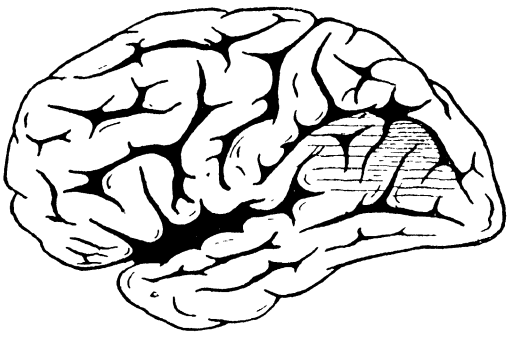

FIG. 70.—Situation of Lesion causing Word-blindness. (*After Charcot, Bouchard, and Brissaud*.)

that part of the cortex lesions of which are found to be associated especially with disorders of speech.

In right-handed persons the speech centres are situated in the left half of the brain, and *vice versâ*. The fibres conveying impressions of visual symbols (words, pictures, objects) end in the neighbourhood of the occipital cortex at the summit

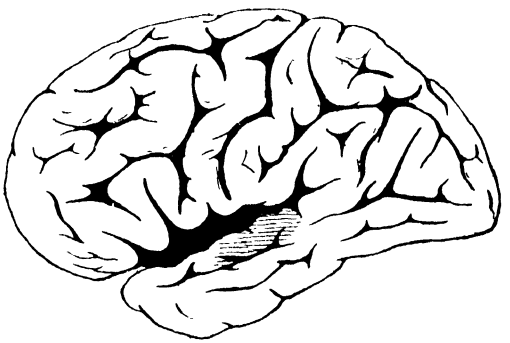

FIG. 71.—Situation of Lesion in the First Left Temporal Convolution, causing Word-deafness. (*After Charcot, Bouchard, and Brissaud.*)

of the angular gyrus: a lesion at this point small enough may produce *word-blindness* or object-blindness (Fig. 70); the fibres of the auditory radiation conveying auditory symbols (words, tunes) end in the posterior part of the superior temporal convolution, and a lesion here will produce *word-deafness* (Fig. 71).

It is only very rarely that such minute lesions occur; as a rule the intermediate region between these two areas mentioned is involved, in which case there is loss to a greater or less degree of all associative reactions in which speech and the memory of auditory and visual symbols are concerned. A widespread lesion in this area therefore reduces a man to a level rather below that of an intelligent

animal, since man has come to depend almost entirely upon the use of verbal symbols for the conduct of intelligent existence. A smaller lesion will give rise to a lesser degree of intellectual impairment together with a tendency to forget and miss the significance of spoken words if it is situated towards the temporal lobe, and a failure of perfect comprehension of written words if the situation is nearer the visual centre. All these are varieties of *sensory aphasia.*

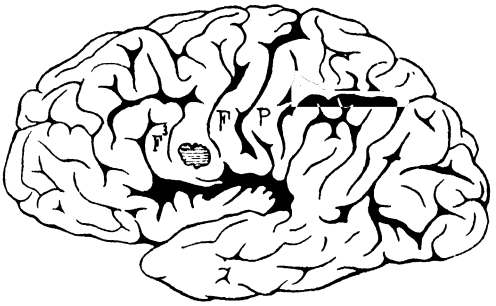


FIG. 72.—Situation of Lesion in case of Motor Aphasia. F. Ascending Frontal Convolution. $F^3$. Third Frontal Convolution. P. Ascending Parietal Convolution. (*After Charcot, Bouchard, and Brissaud*)

A lesion in Broca's area, the posterior part of the inferior frontal convolution, gives rise to what is called pure *motor aphasia* (Fig. 72). There is no failure of comprehension whether of auditory or visual symbols and no impairment of intelligence, no paralysis of any of the muscles concerned in speech production where they are used for other purposes (as the tongue for eating), but an inability

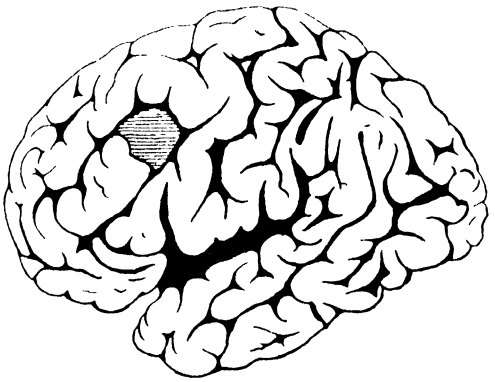

FIG. 73.—Situation of Lesion in case of Agraphia. (*After Charcot, Bouchard, and Brissaud*)

to use these muscles for the particular purpose of speech. Typically the man with a motor aphasia knows what he wants to say, but cannot say it.

Together with motor aphasia is usually associated *agraphia, i.e.*, the patient is at the same time unable to express his thoughts in writing; and in certain rare instances the existence of agraphia has been recorded apart from aphasia in association with lesions of the second frontal convolution (Fig. 73).

In relation to aphasia may be considered *motor apraxia,* a condition in which without loss of power, sensibility or co-ordination the ability is lost to perform certain purposeful movements (such as brushing the teeth, lighting a match).

This phenomenon has been observed after lesions of the upper left frontal convolution and of the corpus callosum, and is closely analogous to motor aphasia and agraphia, which may perhaps be considered as special types of apraxia.

## THE INVOLUNTARY NERVOUS SYSTEM

This is made up of a system of reflex arcs which are entirely outside voluntary control. The structures innervated are the unstriped musculature and the secretory glands of the body, which include the cardio-vascular system; the gastro-intestinal system below the pharynx, excepting the external anal sphincter; the genito-urinary system, except for the external urinary sphincter; the greater part of the respiratory system; and certain muscles in connection with the eye.

Anatomically the involuntary nervous system is characterised by the fact that the cells of the lower motor neurons are situated in ganglia outside the cord, and their axons are non-medullated. These are known as the post-ganglionic fibres, in contrast with the medullated pre-ganglionic fibres, which arise from the cells already described in the lateral horns of the grey matter of the cord. The afferent fibres of this system are medullated, and enter the cord by way of the posterior roots. On the physiological side the system may be divided into two parts, the cranio-bulbo-sacral and the sympathetic. The pre-ganglionic fibres of the former division leave the central nervous system at the level of the brain and the sacral portion of the cord; those of the latter emerge from the cord in the anterior roots from the second dorsal to the second lumbar segments. The functions of these two divisions are in direct opposition, and each is in a state of continuous tonic activity, so that in the normal state the condition of the structures innervated is the result of the balanced action of two opposing forces. This balance may be upset either by heightened activity or paralysis of one of the two divisions.

### THE CERVICAL SYMPATHETIC FIBRES

The pre-ganglionic fibres for the head and neck leave the spinal cord by way of the upper two thoracic roots on either side, whence they pass to ganglion cells in the chain of cervical sympathetic ganglia. Besides innervating the vaso-constrictor muscles and sweat glands in the area supplied, they have certain special functions in connection with the eye, supplying the muscle of Muller which bridges the spheno-maxillary fissure at the back of the orbit and pushes the eye forward, the unstriated fibres in the muscle elevating the upper lid and the dilator muscle of the pupil. These functions are co-ordinated by a centre in the medulla from which fibres descend to a lower centre—the cilio-spinal centre in the eighth cervical segment of the cord.

It will be seen that interference with these functions may be produced by a lesion of the medulla, of the cervical cord, of the upper two thoracic roots, or the cervical sympathetic chain. The clinical signs of unilateral cervical sympathetic paralysis are a slight degree of ptosis, enophthalmos, constriction of the pupil, vasodilatation and inhibition of sweating on the affected side.

### FUNCTIONAL DISTURBANCES OF THE INVOLUNTARY NERVOUS SYSTEM

Very many of the symptoms of disease owe their origin to disturbances of the balance between the cranio-bulbo-sacral and the sympathetic divisions of the involuntary nervous system, the exact mechanism whereby they are produced being in most cases still obscure. It is certain, however, that the internal secretion of the adrenal glands has a stimulating effect upon the sympathetic divi-

sion, and it is possible that other of the endocrine glands play a part in the maintenance of the normal balance. Even more important is the fact that the emotions may have a profound effect upon the functions of the involuntary nervous system and may therefore produce symptoms referred to the various organs innervated by it. It has been shown by Cannon and others that in animals under the influence of strong emotions, such as fear and rage, there is an increase in the secretion of adrenalin leading to a sudden stimulation of the whole sympathetic division; this results in inhibition of the peristaltic movements of the gastro-intestinal canal and cessation of secretion in the glands of

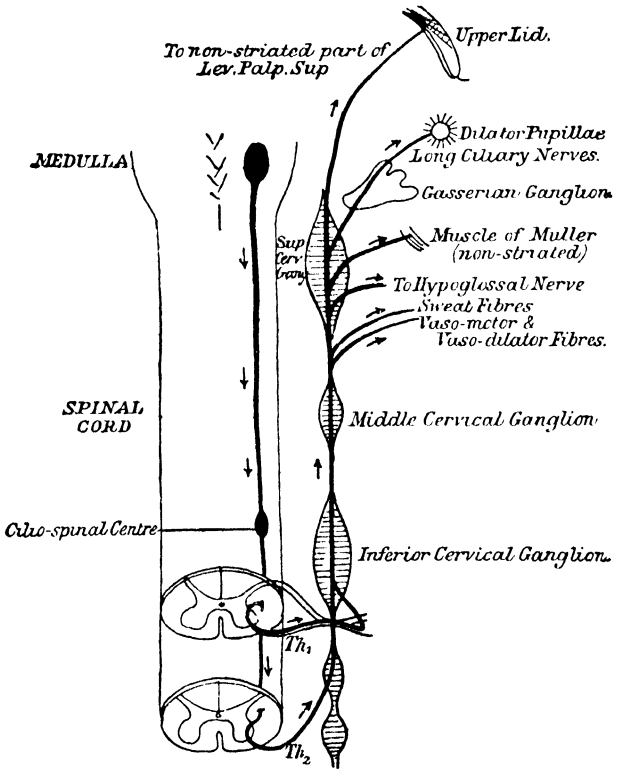


FIG. 73A.—Diagram of course of Oculo-Pupillary Fibres of Cervical Sympathetic. (*From Sir J. Purves Stewart*)

the gastric mucosa; at the same time the rate of the heart beat is increased through the accelerator nerves, and there is an increased liberation of sugar into the blood from the glycogen stored in the liver (*see* p. 589).

Similar changes occur in man, and the maintenance over long periods of a state of emotional tension is often responsible for the production of symptoms of dyspepsia or tachycardia (*see* p. 896). The blood sugar content also is raised, and this may occasionally lead in individuals with a low sugar tolerance to emotional glycosuria.

## THE INNERVATION OF THE URINARY BLADDER

The bladder, in common with the other hollow viscera, receives a nerve supply from both the sympathetic and the cranio-bulbo-sacral divisions of the involuntary nervous system. The action of the former is to inhibit the muscles of the

bladder wall and cause contraction of the urethral sphincter; the latter causes contraction of the bladder wall and inhibition of the sphincter. Under normal conditions, when the intravesical pressure reaches a certain point rhythmic contractions of the bladder wall are set up with further increase of tension. At this point in children there follows reflex emptying of the bladder, but with the acquisition of habits of control over the striated muscles around the urethra the adult is able to inhibit this mechanism; the rhythmic contractions then continue for a while with a further increase of pressure, producing a conscious sensation—the desire to urinate. This being inhibited, there ensues an active relaxation of the walls of the bladder, so that the intravesical pressure sinks below the threshold value for provocation of a reflex contraction. With the secretion of more urine the tension rises again until the threshold is reached, with a repetition of the same series of movements, until micturition is actively initiated by voluntary release of the external sphincter.

The centres for the reflex mechanisms described above are situated in the lumbo-sacral part of the spinal cord. Therefore after complete division of the cord above this level their functions remain intact, and, released from interference by higher control, assume the automatic characteristics of the bladder of infants. Actually in man after a sudden complete lesion of the spinal cord there ensues a variable period of shock, during which there is relaxation of the bladder wall and spasm of the sphincter, with consequent retention of urine, after which automatic reflex emptying appears.

## CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

In the examination of the nervous system it is convenient to proceed according to a regular plan, which enables the observer to make a rapid survey of the case. In the course of such a survey points will be noted which call for more detailed examination of particular functions, methods for which will be described in the text under the appropriate headings. The subjoined scheme is intended to serve as a practical guide to routine examination; the smaller type denotes points which are not considered essential in the absence of special indications for their use.

### SCHEME FOR ROUTINE EXAMINATION OF THE NERVOUS SYSTEM.

1. Cerebral function.
   - Intelligence.
   - Temperament.
   - Speech.
2. The special senses.
   - (*a*) Vision.
     - Acuity.
     - Fields of vision.
     - The fundus oculi.
   - (*b*) Hearing.
   - (*c*) Smell.
   - (*d*) Taste.
3. The cranial nerves.
4. Sensory functions.
   - Sensibility to cotton wool.
   - Sensibility to pin prick.
   - Sensibility to temperature.
   - Sense of position.
   - Sensibility to vibration.
   - Stereognosis.

5. The motor system.
   - Involuntary movements.
   - Atrophy or hypertrophy.
   - Tone.
   - Power.
   - Co-ordination.
   - Electrical reactions.
6. The reflexes.
7. Sphincter control (urethral and anal).
8. Trophic changes.
9. Posture and gait.
10. Cerebro-spinal fluid.

## 1. CEREBRAL FUNCTION

**Intelligence.**—A fair measure of the patient's intelligence may usually be made from the manner in which he gives the history of his illness. If the intellectual functions appear to be defective, further examination should be made. In estimating powers of memory for the remote past, it is important to compare the patient's account of his life with that given by friends or relations. Memory for the recent past may be tested by asking the patient to give an account of the past twenty-four hours, and it is important to make sure that he is correctly oriented as to date, place and persons.

**Temperament.**—Variations from the normal may appear in the form of dominating moods of depression or elation, or as emotional instability. Such variations are most commonly met with in cases of functional nervous disease, in which further questions should be directed to the investigation of special preoccupations or troubles, and inquiry made as to the presence of imaginary ideas, feelings of inadequacy, obsessions or feelings of compulsion.

**Speech.**—This may be affected by lesions at a high or a low level, the resultant disturbances of function being known as aphasia and dysarthria respectively. Aphasia means a partial or complete loss of the ability to comprehend ideas conveyed by words, *or* of the power of verbal expression. Dysarthria implies a defect in verbal articulation due to weakness or inco-ordination in the muscles involved.

The presence of aphasia will be detected in the course of obtaining the patient's history, and should be further investigated along the following lines :—

1. Can the patient understand spoken words ? Give him at first simple commands such as " Put out your tongue," proceeding to more complex directions, such as " Touch your right ear with the middle finger of your left hand."
2. Can he understand written words ? Give him similar commands in writing, first in block capitals, then in ordinary handwriting.
3. Can he spontaneously utter intelligible words ?
4. Can he write spontaneously ?

Failure in test 1 alone would indicate a pure word-deafness, in test 2 alone a pure word-blindness. These two conditions are very rarely met with as a result of minute lesions of the incoming auditory or visual paths to the speech centre. More commonly there is failure in both tests as part of general sensory aphasia.

Failure in test 3 alone indicates pure motor aphasia, in test 4 pure agraphia These two functions also are most commonly lost together.

The patient with motor aphasia from a lesion of the foot of the inferior frontal convolution can understand reading and writing, and his intelligence is therefore unimpaired He is rarely dumb, but his vocabulary is reduced, sometimes to a few very simple words, such as " Yes " and " No," whether he tries to express himself vocally or in writing. He is, of course, conscious of his own disability and its nature.

The patient with sensory aphasia, in addition to his failure of comprehension, also shows errors in the expression of speech, since his word-blindness and word-deafness render him unable to test the correctness of the words he is writing or uttering, a process which is apparently an essential adjunct to the mechanism for outgoing speech. He may talk fluently enough, but tends to mix up words and syllables, and may produce an unintelligible jargon, similar defects being shown in writing. He will naturally make mistakes in reading aloud, writing to dictation, or repeating words heard.

*Word-Forgetfulness.*—There appears to be a special tendency simply to forget the names of people and things, and to be unable to name objects seen, in lesions of the posterior part of the temporal lobe.

It is important to find out in a case of aphasia whether the patient is left-handed, and if not whether left-handedness is hereditary in his family.[1] Aphasia is found in lesions of the left side of the brain in right-handed people, and *vice versâ;* a right hemiplegia, if severe, is nearly always accompanied by aphasia, which is greater in proportion to the degree of paralysis of the arm.

(For further details as to the localisation of speech function *see* p. 718.)

*Dysarthria.*—Since the muscles involved in articulation are innervated from both hemispheres, it is only when there is a lesion of the cortico-bulbar path on each side that an *upper neuron* lesion gives rise to paralytic dysarthria. This condition occurs in multiple vascular or syphilitic lesions of the brain, being commonly accompanied by emotional instability and a severe degree of mental impairment. It is called pseudo-bulbar palsy to distinguish it from the dysarthria caused by lesions of the *lower neurons, e.g.* that met with in progressive muscular atrophy affecting the medulla. Speech in all these cases is clumsy, as if the patient had a hot potato in his mouth, and in true bulbar palsy the patient sometimes becomes completely anarthric.

Dysarthria may also result from a disturbance of the cerebellar co-ordinating mechanism, as in disseminated sclerosis and in some cases of cerebellar lesions (p. 715).

The above forms of dysarthria become apparent when the patient is giving his history, but in a suspected case of dementia paralytica in which the articulatory defect may be due partly to loss of power, partly to loss of co-ordination, and may be an early sign, it is useful to enjoin upon the patient the pronunciation of certain test phrases, such as "Methodist Episcopal," "West Register Street," etc. The defects to be looked for are slurring, elision of syllables or transposition of syllables or words.

## 2. SPECIAL SENSES

**Vision.**—*Acuity.*—In default of regulation test type, the acuity of vision may be roughly tested by getting the patient to read aloud from small print with each eye in turn; if acuity is poor, allow him to wear glasses if he has them; if these are correct, and the defect lies only in an error of refraction, his visual acuity should be normal with glasses.

*Visual Fields.*—These may be roughly tested as follows: The patient places a hand over his right eye, and fixes the gaze of the other upon the pupil of the observer's right eye; the observer himself closes his left eye, and fixes his attention upon the patient's left eye; he then takes a small white object upon a handle —the most convenient is an ordinary white-headed hat-pin—and holding it midway between himself and the patient, brings it in gradually from the periphery towards the centre of the visual field, instructing the patient to say "Now!" when he sees it. In this way the observer can compare the field of each of the

[1] Foster Kennedy has shown that in a right-handed person of strongly left-handed stock the speech centres may be situated in the *right* hemisphere, and *vice versâ*.

patient's eyes with his own and can discover defects of any considerable size. Examination of the visual fields is a most important procedure, and may lead to valuable discoveries, especially in the case of lesions affecting the optic tracts (*e.g.* pituitary tumour) or optic radiations (*e.g.* a vascular lesion at the posterior end of the internal capsule) (*see* p. 712).

*Fundus Oculi.*—For the physician the eye is the window of the brain, and the electrical ophthalmoscope renders examination of the optic disc so easy that it should be made in every case in which nervous disease is suspected. The main changes to be looked for in the disc are œdema and atrophy.

*Papillœdema* is usually the result of increased intracranial pressure, and the mechanism of its production is discussed later (*see* p. 744). In a typical early case the central vein is engorged, the physiological cup filled in with exudate, and the edges of the disc, instead of being clear-cut, are hazy; later the œdema may be such as to give the disc a "chrysanthemum-head" appearance, and there are patches of exudate and hæmorrhages in the retina surrounding the disc.

In *optic atrophy* the disc appears paler than normal, and in an advanced stage of the condition stands out a dead white against the surrounding pink retina. The minute appearances vary somewhat with the cause and are discussed later (*see* p. 744).

It is important to realise that, while optic atrophy is always associated with some defect of visual acuity, the presence of an advanced papillœdema is consistent with the preservation of normal vision, and it is not until the swelling begins to subside and the condition passes into one of secondary atrophy that acuity becomes diminished. Other evidence of value may be found in some cases of brain disease from an examination of the retinal arteries, whose condition is perhaps the best index of that of the cerebral arterioles. In cases of cerebral arterio-sclerosis the retinal arteries are often tortuous and thick-walled, so that they reflect the light like strands of "silver wire"; there may also be visible beading of the arterial walls and retinal hæmorrhages (*see* p. 367). Other discoveries of value may be disseminated choroido-retinitis of syphilitic origin, or albuminuric retinitis.

**Hearing.**—This may be roughly tested by closing the patient's right ear and bringing a watch gradually nearer to the left ear until he can hear it ticking. The process is repeated for the other ear and the results expressed in terms of the distance, roughly gauged in inches, at which the watch is heard. If auditory acuity is greatly diminished on one or both sides, Rinné's test should be performed. In this a small vibrating tuning fork is placed with its base upon one mastoid; the patient is instructed to make a signal when he ceases to appreciate the vibration, and the fork is then transferred to a point just outside the auditory meatus, when the patient should hear it again; if he does not, Rinné's test is said to be negative, and the conclusion is that the deafness is due to failure of conduction of the sound through the middle ear. If Rinné's test is positive in the presence of deafness, the trouble is in the auditory nerve itself (nerve deafness).

**Smell.**—This is tested by making the patient close each nostril in turn by pressure with his finger and then, smelling different substances, attempt to name them. The most suitable test substances are asafœtida, oil of cloves, camphor, eucalyptus, and peppermint. Unilateral anosmia may occasionally be of value as a localising sign in the case of a tumour of the frontal lobe pressing upon the olfactory tract. In this test allowance should be made for intranasal conditions such as are likely to impair sensibility.

**Taste.**—Tests of sensation of taste are seldom of clinical value, and need not be included in a routine examination. The taste fibres for the anterior two-thirds of the tongue ascend first in the lingual nerve and then in the chorda tympani, while the glosso-pharyngeal nerve supplies the posterior third.

### 3. THE CRANIAL NERVES

**The Eyes.**—The width of the palpebral apertures is first noted for the presence of ptosis, enophthalmos or exophthalmos. The pupils are next inspected. They are normally equal in size, circular, and situated centrally in the iris. The reaction to light of each pupil in turn is tested by flashing into it the light of an electric torch, and the reaction to accommodation of both pupils simultaneously by asking the patient first to gaze into the distance and then to concentrate his vision upon an object held 6 or 8 inches in front of his nose.

*Ptosis*, or drooping of the upper eyelid, unilateral or bilateral, is most commonly the result of paralysis of one or both third nerves. It may also result from cervical sympathetic paralysis, or be due to weakness of the levator muscles themselves, as in myasthenia gravis.

Inequality of the *pupils* may be caused by cervical sympathetic paralysis, resulting in miosis, or by a lesion of one of the third nerves giving rise to paralytic dilatation, on the affected side. It is, however, most commonly seen in parenchymatous syphilis of the central nervous system, together with the other abnormalities grouped under the heading of Argyll-Robertson pupils. In this condition the pupils are, as a rule, unequal (more often small than large); their outlines are irregular (often ovoid), and their position excentric. Typically they react briskly in accommodation, but not at all to light. In a later stage the reaction in accommodation may also be lost. In encephalitis lethargica a condition of the pupils is sometimes observed which is the reverse of that described by Argyll-Robertson, *i.e.* they react to light, but not in accommodation. But true Argyll-Robertson pupils may also be seen as the result of encephalitis lethargica. Lesions of the superior corpora quadrigemina may also be responsible for the production of fixed pupils.

In cases of blindness due to lesions of the optic nerve, the pupil of the affected eye is dilated, and does not react to direct illumination, but, owing to the bilateral nuclear innervation of the iris, contracts simultaneously with the pupil of the other eye when this is stimulated by light. In this condition it is said to react to consensual, but not to direct, illumination.

*The movements of the eyes* are effected by means of the third, fourth and sixth nerves. Paralysis of one or more of these nerves, if complete, may be easily detected on inspection. In third nerve paralysis there is complete ptosis, the pupil of the affected eye is dilated and does not react to light or distance, and the eye at rest is turned outwards by the unopposed action of the external rectus. The only movements left are outward movement, innervated by the sixth nerve, and a certain degree of downward movement, achieved through the superior oblique, which is innervated by the fourth nerve. In paralysis of the sixth nerve the eye is turned inwards by the unbalanced action of the internal rectus. Isolated paralysis of the fourth nerve does not give rise to a squint as long as the eyes are at rest.

The range of the ocular movements is further tested by asking the patient to follow with his gaze the observer's finger. He is thus made to look upward, downward and to either side in turn. By this means lesser degrees of weakness may be detected. The action of the fourth nerve is to turn the eye downward and outward. If, as sometimes happens, the physician wishes to test the range of ocular movements in a patient who is completely blind, he can achieve this by holding the patient's own fingers in the requisite positions and asking him to direct his gaze towards them. The finest degrees of weakness may give rise to subjective double vision or diplopia without any objective loss of parallelism of the ocular axes.

Therefore in the course of testing the ocular movements the patient should be asked to say if at any position of the eyes he is conscious of seeing two images instead of one. Then with the eyes still in this position one of them is closed,

and he is asked to state which of the images disappears, the right hand or the left.

In diplopia the false image seen by the affected eye is displaced in the direction of traction of the paralysed muscle. From this it follows that in paralysis of any one of the muscles which turn the eye inward the images seen by the two eyes will be crossed; *i.e.* if the right eye be closed the left-hand image will disappear, and *vice versâ*: while in paralysis of the muscles which move the eye outward the images will be uncrossed, *i.e.* if the right eye be closed the right-hand image will disappear. In the case of the muscles which move the eye obliquely the false image will also appear tilted at an angle to the true image depending (Fig. 74) upon the muscle involved.

The accompanying chart, devised by Bishop Harman, shows (1) the movements of the ocular muscles; (2) the position of the false image in paralysis. It should be remembered that in certain diseases, *e.g.* disseminated sclerosis and syphilis of the nervous system, ocular palsies may occur which are of very

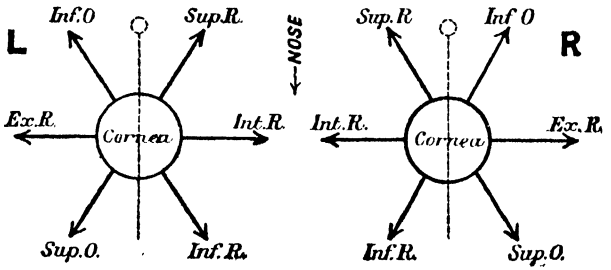


FIG. 74.—N. Bishop Harman's Chart to show (1) *Movements of Ocular Muscles*, and (2) *Positions of False Image* in Paralysis.

(1) (*a*) Rectangular movements. The arrows point to the direction in which the eye is turned by each muscle. (*b*) Rotation. Put a match, head upwards, on each of the dotted lines indicating the vertical meridians. Muscles that rotate eye inwards turn the match-head towards nose (Sup. Rectus and Sup. Oblique); those that rotate it outwards turn match in the opposite direction (Inf. Oblique and Inf. Rectus).

(2) Put matches on diagram again. The match will represent the true image. The four rays marked Sup. R., Inf. R., Sup. O., and Inf. O. will represent the relative position (in vertical and lateral displacement and tilting) of the false image produced in paralysis of each of these muscles. In paralysis of Int. or Ext. Rectus the false image will run vertically through the corresponding arrow-head. (Sir James Purves Stewart)

brief duration. It is therefore important in taking the history of a case of nervous disease to inquire for transient diplopia.

In the course of testing the ocular movements the examiner will note the presence or absence of *nystagmus*. This is a condition in which conjugate movements of the eyes are poorly sustained, and in place of a steady deviation we see a succession of jerky oscillations. These may be slow or quick, coarse or fine, lateral or rotatory.

It should be remembered that in persons weakened by old age or constitutional disease prolonged deviation of the eyes in a single direction will readily cause fatigue of the ocular muscles, and so give rise to oscillatory movements somewhat resembling those of nystagmus.

**The Fifth, or Trigeminal, Nerve.**—The distribution to the skin of the three branches of the sensory division of this nerve is shown in Fig. 62.

Sensibility to cotton wool, pin prick, and heat and cold over this area may be tested as presently to be described in relation to the other cutaneous areas. The buccal and nasal mucous membranes of the corresponding side are also supplied by each nerve. The earliest objective sign, however, in lesions of the

sensory root or its ophthalmic division, is a diminution or loss of the corneal reflex on the affected side. This sign may be sought as follows: The patient is directed to fix his vision on some object to his extreme left, while the observer, standing a little behind him on his right, brings the head of an ordinary pin into light contact with the right cornea in such a manner that the object is felt before it is seen. The process is repeated for the left cornea, and the threshold for the blinking reflex on the two sides is thus compared.

The motor branch of the fifth nerve supplies all the muscles of mastication. In paralysis of this branch, when the patient's teeth are tightly clenched the observer by placing his thumbs against the anterior borders of the two masseter muscles can detect the weakness on the affected side, and when the mouth is opened wide the point of the jaw is deflected towards the paralysed side.

Subjective sensations of numbness, tingling, or pain in one or more of the areas supplied by the sensory root may precede objective phenomena in lesions of this nerve.

**The Seventh, or Facial, Nerve.**—In complete paralysis of this nerve the affected side of the face is mask-like, immobile, and expressionless; the naso-labial fold is smoothed out; the wrinkles disappear from the forehead; the eye is wider open than on the sound side; and the corner of the mouth hangs downwards.

The patient may complain of irritation of the conjunctiva on the affected side (owing to loss of the blinking reflex from paralysis of the orbicularis palpebrarum) as well as a certain clumsiness in speaking. Lesser degrees of weakness may be detected by requesting the patient to close his eyes tightly and show his teeth at the same time, a procedure which usually unmasks any asymmetry that may be present. The patient is also made to open his mouth widely, which brings the naso-labial grooves into prominence, when they may be compared.

**The Eighth Nerve.**—The examination of the auditory functions of this nerve has already been described under the Special Senses. In cases of vertigo, or of nerve deafness, in which it is desirable to investigate the vestibular functions, a simple method is as follows: The patient is made to stand up and turn rapidly round five times, first clockwise and after a short rest counter-clockwise. Immediately after the last turn in the clockwise direction (*i.e.* from his left to his right) he is made to fix his gaze on a point to his extreme left, and after turning in the opposite direction on a point to his extreme right. In each case transient nystagmus should be observed, and if the function of both vestibules is normal, the rapidity of the movements of the eyes and their duration should be the same when the individual is rotated clockwise or counter-clockwise.

If any inequality is observed, the general rule is that there is a destructive lesion of the vestibule on the side of the lesser nystagmus. An equally simple and satisfactory test is for the patient to turn five times around a walking-stick, which he holds in the erect position with his back bent so that his forehead rests upon the handle of the stick; he then attempts to walk along a straight line marked on the floor. A normal individual diverges from the line in the same direction as that in which he has been turning, the angle being equal whether he turns to the right or left; but in severe bilateral vestibular disease he walks straight forward, or if the ears are unequally affected, the angle differs according to the direction in which he has been turning.

**The Ninth, or Glosso-pharyngeal, Nerve.**—This nerve supplies taste fibres to the posterior third of the tongue and the soft palate, and motor branches to the pharynx. Its functions cannot be adequately investigated by any simple test.

**The Tenth Nerve, or Vagus.**—The simplest objective test for the vagus is to ask the patient to say "Ah!" and watch the palate. Normally one sees the median raphé of the soft palate rise straight up, but if one side be paralysed, the healthy side alone pulls upwards, and the raphé deviates to the sound side, forming a characteristic dimple.

(For further symptoms *see* p. 752.)

**Eleventh, or Spinal Accessory, Nerve.**—In order to test the function of this pair of nerves, which are purely motor, one asks the patient to shrug his shoulders, which is effected by the trapezii, and then to flex his neck against resistance, which brings both sternomastoids into play in such a manner that the contractions of the two muscles may be easily compared.

**Twelfth, or Hypoglossal, Nerve.**—This nerve also is purely motor in function, supplying the tongue and most of the muscles attached to the hyoid bone. To investigate its function one asks the patient to protrude his tongue. In bilateral paralysis there is no movement; in cases of unilateral paralysis the tip of the tongue is pushed over towards the side paralysed by the sound muscles on the other side. If the paralysis is due to a lower motor neuron lesion (*i.e.* of the nuclei or fibres of the hypoglossal nerve), there is atrophy of the muscles on the affected side, and the superjacent mucous membrane is thrown into wrinkles.

### 4. SENSORY FUNCTIONS

In testing sensibility of any kind the observer is repaid for taking the trouble to place a bandage or handkerchief over the patient's eyes rather than ask him to keep them closed; the latter method involves an effort on the part of the patient which detracts from the attention which he is required to give to the examination.

**Sensibility to Light Touch.**—The patient is asked to say "Yes" each time he appreciates the touch of a wisp of cotton wool or a camel's-hair brush. The examiner begins with a few touches over areas of skin not suspected of being anæsthetic, and if contacts are missed in other regions, applies the stimulus again to the normal areas from time to time to ensure that the failure to respond is not due merely to lack of attention.

The sensitiveness of the limbs is first explored by means of a few touches applied to each hand and foot; the investigation should then be carried longitudinally up each limb, and finally a series of stimuli should be applied, following the circumference of the limb at one or two levels. This will ensure that a small area of segmental anæsthesia is not missed. The skin area of the trunk is investigated in a similar manner. If a zone is discovered in which sensation appears to be lost or blunted, this must be carefully tested, and the resultant area of anæsthesia outlined in blue pencil on the skin, from which it may be copied on to a chart.

The investigator should not be content with an objective examination, but should direct the patient to inform him, if at any point the stimulus appears relatively less distinct than in normal areas, and should especially inquire whether touch is appreciated equally on both sides of the body.

**Sensibility to Pain** is investigated along similar lines, the point of an ordinary pin being employed as stimulus. Here again it is most important to discover from the patient any quantitative diminution of sensation.

**Sensibility to Temperature.**—Since the paths for fibres transmitting impulses of heat and cold are closely associated with those for pain, an investigation of the latter is sufficient in an ordinary routine neurological examination. In certain cases, however (syringomyelia, tumour of the spinal cord), it is essential to test the sensibility to pain and temperature separately. For the latter two test tubes are used, one containing cold water (preferably fragments of ice), the other water at a temperature which can be tolerated by the hand. The skin areas are explored after the manner indicated above, the patient being asked to respond "Hot!" or "Cold!" to each stimulus.

In case of faulty responses being recorded, the observer should make sure that the temperatures of the two tubes still provide a reasonable contrast in sensation when applied to his own skin.

**Sense of Position** may be roughly tested for the hands in the following

manner: The observer taking the patient's right wrist, and holding it still, instructs the latter to touch the tip of his right forefinger with his left; he then alters the position in space of the patient's hand and repeats the test, which affords a measure of the patient's sense of position for his right hand (his eyes, of course, being shut). Sense of position in the other hand is investigated in similar fashion.

In the lower limbs the most delicate test is that in which the observer takes the patient's great toe between his finger and thumb and at brief intervals of time bends it up or down. The patient (whose eyes are closed) is instructed to call "Up!" or "Down!" as soon as he is conscious of any movement. The normal adult makes no mistake in this test. A further measure of this form of sensation is gained by moving the lower limbs into various positions and asking the blindfolded patient to point to his great toes.

**Sense of Vibration.**—For this examination the observer uses a large vibrating tuning fork, the handle of which is applied to the various bony prominences in turn, while the patient is asked to describe his sensations. A standard of normal sensibility may be obtained by comparable observations on one's own person. This form of sensation is early impaired or lost in the lower limbs in case of damage to the posterior column fibres, for the recognition of which the test is of considerable value.

**Stereognosis.**—This term is used to denote the power of recognising the form, size and texture of objects placed in the hands without the use of vision. Patients who have lost this power are said to have astereognosis. This condition may be due to lesions anywhere on the afferent pathways, but may also result from damage to the cerebral cortex; in the latter case astereognosis may exist without any marked impairment of the ordinary forms of sensibility, and may therefore be a sign of great value in the localisation of a tumour or abscess in the brain.

### 5. THE MOTOR SYSTEM

**Involuntary Movements** may result either from irritation of motor neurons, or from destruction of neurons which normally exercise an inhibitory influence upon lower centres.

The *convulsions*, localised and generalised, of cerebral origin, are described under the headings of Epilepsy and Tumour of the Brain.

*Tremors*, which consist of rhythmic alternating movements of groups of muscles and their antagonists, are classified according to the rate and amplitude of the movements and the muscles affected.

As an example of rapid fine tremor may be mentioned that observed in the fingers in Graves' disease, while that seen in paralysis agitans is relatively slow and coarse.

The main feature of *choreiform movements*, as seen typically in a case of so-called rheumatic chorea, is their irregularity and lack of rhythm.

The movements of *athetosis* are different from these, and occur most commonly in the spastic limbs of old-standing hemiplegias; they are usually confined to the upper limb and consist of slow, irregular writhing movements, most marked in the fingers and wrists, but in severe cases involving the arm. In a typical case from a position of rest the arm is flexed and pronated, the wrist flexed, and the fingers hyperextended and spread out irregularly one by one like the tentacles of an octopus (*see* Fig. 89, p. 819). When both arms are involved the movements also affect the face, giving rise to the most hideous grimaces. In the lower limb the foot is occasionally seized with a spasm, in which there is inversion and adduction at the ankle and hyperextension of the great toe. Athetoid movements do not occur in a completely paralysed limb; they are increased by voluntary movement, whether on the affected or the sound side of the body.

The condition known as *myoclonus* is characterised by the occurrence of

sudden shock-like contractions of various muscles, irregular in rate and rhythm, with a tendency to appear in paroxysms, each lasting for a few seconds or minutes. Involuntary movements of the myoclonic type have been frequently observed in encephalitis lethargica, and are seen as an isolated phenomenon in certain rare cases of familial disease.

*Fibrillary tremors* or twitchings are quick contractions of isolated portions or fibrillæ of muscles just visible under the skin, and only occasionally moving the joints. They occur especially in muscles supplied by anterior horn cells which are undergoing slow degeneration, as in chronic anterior poliomyelitis.

**Atrophy** of a muscle may result from a lesion of the lower motor neuron supplying it (anterior poliomyelitis, peripheral neuritis), or may be due to changes of a degenerative nature arising in the muscle fibres themselves (muscular dystrophy). Lesser degrees of local muscular wasting occur in limbs whose lower motor neurons are intact, but which have not been used for a long period,

FIG. 75.—Photograph of a child presenting Kernig's sign. Note the contraction of the hamstring muscles. (*After Turner and Stewart.*)

*e.g.*, in cases of old-standing lesions of upper motor neurons and in hysterical paralysis. Generalised muscular atrophy may be a pronounced feature of states of chronic malnutrition, such as phthisis, diabetes, or malignant disease.

Pathological hypertrophy is seen only in rare cases of muscular dystrophy (*see* p. 914 and Fig. 93).

The condition of muscular **tone** is measured by the ease with which passive movements can be performed at the various joints. In cases of extreme loss of tone, as in tabes and amyotonia congenita, the range of these movements is greatly increased, so that it is possible, for instance, to approximate the patient's feet to his shoulders by means of flexion at the hips. The increase of tone or spasticity seen as the result of pyramidal lesions is confined to the flexor muscle groups in the arm and the extensor groups in the lower limb ; it is characterised by a resistance to passive movement in the opposite direction, which is maximal at first, but subsequently relaxes, similar to that met with in opening a clasp knife.

In the rigidity of paralysis agitans antagonistic groups of muscles are affected, and the resistance to passive movements is evenly maintained.

**Kernig's Sign.**—In meningitis and some other conditions it is found that if the thigh be flexed at a right angle with the body, either by placing the patient in a sitting posture or by raising the thigh vertically while the patient is recumbent, it is impossible to extend the leg on the thigh to the same extent as usual, in consequence of the contraction of the hamstring muscles (*see* Fig. 75). The muscular spasm in this case is mainly due to a voluntary contraction whose object is to ward off the pain caused by stretching inflamed nerves or nerve roots.

**Voluntary Power** is systematically investigated by asking the patient to perform various movements against resistance, flexion and extension of the fingers, wrists and arms, pronation and supination, and so on.

In case of weakness due to a lesion of pyramidal fibres the muscles most affected are in the upper limb the extensors, with earliest loss of power in movements of the fingers and extension of the wrist, and in the lower limbs the flexors, with earliest involvement of dorsiflexion of the foot.

**Inco-ordination** of movement in the absence of weakness may be due to loss of sense of position and passive movement, or to destructive lesions of the cerebellum. Many methods have been devised for testing co-ordination, of which the simplest and best are as follows:—

*Finger-nose-finger Test.*—The observer holds up his right index finger in front of the patient and within his reach. The patient is instructed with the tip of his right index finger to touch first the end of his own nose, then the tip of the observer's finger, and to repeat these movements alternately as quickly and as neatly as possible. The observer meanwhile changes the position of his finger in relation to the patient between the contacts.

Inco-ordination is revealed by deviation from the line of movement with clumsiness and delay in arriving at the mark, and by a tendency either to stop short of it or overshoot it (dysmetria).

*Diadochokinesis Test.*—The patient holding his arms semiflexed with the elbows to his sides, and fingers clenched, performs alternate movements of pronation and supination simultaneously with both forearms as rapidly as possible.

Inco-ordination is revealed by dysdiadochokinesis; the affected limb lags behind its fellow; its action is clumsy and unbalanced, and is made to appear more so by superfluous movements at the elbow and shoulder.

*The Heel-knee Test.*—The patient in the recumbent position lifts one heel high into the air, places it gently upon the opposite knee and slides it steadily down the anterior border of the tibia as far as the ankle. This series of movements should normally be performed smoothly and evenly. Inco-ordination is revealed by unsteadiness and irregularity.

**Electrical Reactions.**—The nature of the response of a muscle to electrical stimulation is sometimes of value in distinguishing between the different causes of paralysis, and in determining the moment at which recovery begins after a lower motor neuron lesion.

The student is presumed to be familiar with the methods of physiological application of the electric current to the stimulation of muscle and nerve, which are to be found in the text-books of physiology.

The faradic current is an adequate stimulus to nerves and nerve endings, and when applied to normal muscles in health provokes a brisk and well-sustained contraction, which is especially well marked if the electrode is placed over the point at which the motor nerve enters the muscle.

In the case of a complete lower motor neuron lesion there is no response to faradic stimulation either of the nerve or muscle involved; in the case of incomplete lesions there may be a quantitative diminution of the response.

To the galvanic current muscular contraction is only obtained when the circuit is closed and opened; none is observed during the continued flow of the current. As there are two poles, four series of contractions may be observed, two on opening and closing the circuit where the stimulating electrode is the kathode and

two on opening and closing the circuit when the anode is used as the stimulating point.

These are usually represented by letters as follows :—

K.C.C. = Kathodal closing contraction.
A.C.C. = Anodal closing contraction.
A.O.C. = Anodal opening contraction.
K.O.C. = Kathodal opening contraction.

In health, galvanic stimulation is effective whether applied to the muscle or the nerve, resulting in a short sharp contraction. If the strength of the current be constant and moderate, it is found that K.C.C. is greater than A.C.C.

In the case of a lower motor neuron lesion galvanic stimulation of the nerve no longer produces a response. The muscle, however, shows certain changes in its response, which are described as the reaction of degeneration (R.D.).

Qualitatively the response to galvanism is slow and sluggish, instead of short and sharp, and quantitatively it is found that with a constant strength of current A.C.C. is equal to or greater than K.C.C.

The condition of the nerve supply to a muscle may also be investigated by determining its chronaxie.[1]

The return of faradic excitability in a muscle is usually the earliest sign of recovery from a lower motor neuron lesion.

In certain diseases, Thomsen's disease and myasthenia gravis, there may be characteristic changes in the electrical excitability, which are described under these headings.

### 6. THE REFLEXES

The reflexes are divided into superficial, deep, and visceral. Their presence depends upon the integrity of the sensory and motor end organs and the afferent and efferent paths involved in the reflex arc. They may also be modified by destructive lesions of higher centres, which have a controlling or inhibitory influence over them.

The reflexes of chief value in clinical examination are the following :—

**Superficial Reflexes.**—The pupillary, corneal and pharyngeal reflexes are described under the Cranial Nerves (p. 726). The abdominal reflexes are obtained by light stroking of the skin, with resultant contraction of the underlying musculature. There may be distinguished an epigastric mid-abdominal and lower abdominal reflex on each side. These are present and equal on the two sides in the great majority of healthy adults. They may, however, be absent when the abdominal wall is loose and pendulous, as in a multipara, in cases of intra-abdominal disease, such as typhoid, and when the patient is unduly cold.

Their absence, apart from these causes, implies an affection of pyramidal fibres ; the abdominal reflexes are lost on the opposite side of the body when the lesion is above the pyramidal decussation, on the same side if below it. Loss of these reflexes is frequently an early sign of pyramidal involvement, and a constant quantitative difference between the responses obtained on the two sides of the body may be of value in diagnosis in cases where there is no absolute loss. The cremasteric reflex is obtained by stroking the skin along the inner surface of the

[1] *Chronaxie.*—Adrian has shown that by ascertaining the chronaxie of a muscle one may frequently obtain valuable evidence as to the state of its nerve supply. When a muscle is stimulated by means of a constant current the elicitation of a response depends upon two factors: the strength and the duration of the current. The strength of current being first determined which will, if of infinite duration, provoke a minimal contraction, the duration is then gradually reduced until there is no longer any response, unless the strength is increased. The duration is again decreased, and the strength increased to obtain a contraction until the duration is reached at which the original current must be doubled in order to elicit a response. This is known as the *chronaxie*, and is for muscle with intact nerve supply 0·00016 second, and for denervated muscle 0·011 second. The results of such a test expressed graphically show characteristic curves which are different in the two cases.

thigh ; the response is drawing up of the testicle. Together with the abdominal reflexes, it is lost in the affected side in cases of pyramidal lesion.

*The Plantar Reflex.*—In conditions of health stroking the outer margin of the sole of the foot produces flexion of the small toes and of the great toe at the metatarso-phalangeal joint. This response may be complicated in ticklish persons by voluntary movements of withdrawal or rigidity due to apprehension. For the elicitation of the test it is best to have the lower limb slightly everted at the hip joint, flexed at the knee and relaxed at the ankle joint, but in some cases of disease the extensor plantar response is best obtained with the limb in the extended position. The skin should be warm and dry. The nature of the optimum stimulus depends in each individual case upon a number of variable factors. As a rule, firm pressure with the thumb nail drawn slowly from the heel towards the base of the toes along the outer border of the sole is sufficient, or a blunt-pointed instrument may be used. The reflex response is lost in lesions of the reflex arc of the first sacral segment ; in coma ; with the sensory disturbances involving the sole of the foot in tabes ; with paralysis of the toe muscles, as in acute poliomyelitis ; and when the feet are cold. In the presence of a lesion of the pyramidal fibres, in place of the normal response Babinski's sign is elicited on the corresponding side ; this consists of an upward movement of the great toe, with abduction and fanning of the others, and is accompanied by a contraction of the hamstrings. In cases of complete transverse lesion of the cord there ensues at first loss of the plantar responses, but with recovery from spinal shock there develops a mass movement of withdrawal of both limbs in response to stimulation of the sole of either foot (*see* p. 713).

**Deep Reflexes.**—These are true reflexes, the activity of which is an indication of the state of the muscular tone, while their presence depends upon the integrity of the reflex arcs and end organs concerned.

They are tested by placing the limb in such a position as to relax the muscle whose tendon jerk is to be elicited. The observer then puts the muscle under slight tension and gives the tendon a sharp tap with his finger, or better with a percussion hammer. The response is a contraction of the muscle and movement of the part. The deep reflexes of most clinical value are the following :—

*The Triceps Jerk.*—This is obtained by semiflexing the arm at the elbow and striking the tendon just above its insertion into the olecranon.

*The Biceps Jerk* is obtained by flexing the elbow joint, placing the thumb upon the biceps tendon just above its insertion and slightly stretching the muscle. The thumb is then struck, and the biceps contracts.

*The Supinator Jerk.*—To obtain this, the observer holds the patient's hand loosely with elbow semiflexed and forearm semi-pronated, and strikes the outer border of the radius just above the styloid process. The response is a contraction of the supinator longus (brachio-radialis) flexing the elbow.

*The Knee Jerk.*—When the leg is hanging freely with the knee bent at a right angle, and the ligamentum patellæ is sharply struck with the tips of the fingers, the ulnar edge of the hand, the edge of a book, or the percussion hammer, the rectus femoris contracts, and the foot is jerked sharply forward. This has been called the *knee phenomenon, patellar tendon reflex, patellar reflex,* or *knee jerk.* It is generally well obtained in the sitting position by crossing one leg over the other and striking the upper knee. Often it can be obtained by striking above the patella. When the reaction is slight, the patient should sit on a table, with the legs hanging over the edge, and the knees should be bare. If it is not elicited then, the patient's attention should be distracted, as by getting him to hold the fingers of one hand in those of the other, and to look up to the ceiling while he pulls at his hands (*Jendrassik's reinforcement*).

When the patient is in bed, the leg may be raised by placing the hand under the knee.

*The Tendo Achillis, or Ankle Jerk.*—This is obtained by putting the calf muscles

slightly on the stretch and tapping the tendo Achillis, the response being a brisk contraction of the muscles. It is sometimes difficult to demonstrate in normal persons, but may nearly always be obtained by making the patient kneel upon a chair with his weight well forward upon his knees and striking the tendons from above in this position.

*Ankle Clonus.*—*Ankle clonus*, or *foot clonus*, is a similar phenomenon, which occurs in certain spinal and other diseases, but is not, like the knee jerk, present in health, except in a modified form. To elicit it, the patient should be seated or recumbent; the leg is lifted with the left hand under the knee, so that the knee is slightly bent, and the foot, held firmly by the toes in the right hand, is sharply bent towards the knee. Immediately the calf muscles contract, but as the pressure on the foot is maintained, they relax, again contract, and so alternately contract and relax for an almost indefinite period, constituting the so-called *clonus*. The contractions occur at the rate of about seven in a second. The modification of this phenomenon which occurs in health is the series of rapid alternating movements which can be kept up continuously and without effort when, in the sitting posture, the foot rests upon the ground by the toes only.

A *knee clonus* can be sometimes obtained either as a result of percussing to get the knee jerk, or by pushing the patella down towards the tibia, while the leg is extended on a couch.

Exaggeration of the deep reflexes and ankle clonus are found together with absent abdominal reflexes and Babinski's sign as the result of pyramidal lesions. Loss of the tendon jerks may result from disease of the muscles, peripheral nerves, anterior or posterior roots, or anterior horn cells.

Quantitative alterations of the tendon jerks are of value when there is a marked difference in vigour between, for instance, the knee jerks and the ankle jerks, or between the responses obtained on the two sides of the body.

The state of the deep reflexes in general, however, may be influenced by many conditions, such as emotional excitement, fatigue, temperature and intoxication.

### 7. SPHINCTER CONTROL

**The Bladder.**—Apart from obvious retention or incontinence of urine, the patient should be questioned with regard to a history of difficulty in commencing micturition or the more common fault of being unable to delay the act once conscious of the need (precipitate micturition).

**The Rectum.**—The early symptoms of paralysis affecting this part are constipation, or more truly dyschezia (*see* p. 435), and when recourse is had to purgatives difficulty in controlling a loose motion, with occasional soiling of the garments.

### 8. TROPHIC CHANGES

The nutrition of the tissues is profoundly affected in some diseases of the nervous system, but there is no evidence of separate trophic nerves or neurons. The most marked effects are seen in lesions of nerve trunks and their centres, *i.e.*, those which injure the lower neurons, motor and sensory. Thus lesions of the anterior cornua (acute poliomyelitis) or of the nerves (injury, neuritis) are accompanied by marked wasting of muscle, which is not present in lesions of the brain or cord, involving the upper neurons only. Wasting of muscle is first shown by flabbiness, later by actual diminution in size. Its extent can be estimated by measurement, but it must be remembered that subcutaneous fat may completely mask a good deal of wasting so far as bulk is concerned. Other parts besides the muscles are often involved. The skin in some chronic cases becomes thin, red, and shiny—the "glossy skin" of Paget; erythematous, bullous, and vesicular eruptions (*e.g.*, zona), œdema, whitlows and ulceration of the skin may occur; the finger nails are pinched, from wasting of the subcutaneous tissue; the growth of

hair and nails is retarded ; and the nails are brittle. The bones may also suffer in their nutrition, becoming brittle or breaking easily ; and if paralysis occurs in early life, growth of a whole limb may be retarded, so that it is eventually 1½ to 2 inches shorter than its fellow. In acute cases the temperature of the skin is raised, the vessels dilate, vesicles or bullæ form, and bedsores occur on the slightest irritation or pressure.

### 9. POSTURE AND GAIT

If the patient is not bedridden, he should be made to walk up and down the room in his ordinary manner, and any peculiarity of his carriage or gait should be noted. The changes characteristic of various lesions are described under other headings. It should be noted here that account must be taken of skeletal deformities and disease of joints, and further that weakness of certain groups of muscles (*e.g.*, the hamstrings and glutæi) may lead not only to abnormalities in the gait, but also to the adoption of certain postures calculated to preserve the centre of gravity in the appropriate position.

### 10. CEREBRO-SPINAL FLUID

This fluid, as obtained by lumbar puncture, is normally clear and colourless, and resembles closely physiological salt solution, containing 0·73 per cent. chlorides and a trace of sugar (sufficient to reduce Fehling's solution). The normal cell content is from 0 to 5 lymphocytes per cubic millimetre. The cerebro-spinal fluid is secreted from the blood into the lateral ventricles of the brain by the cells of the choroid plexus ; thence it passes through the foramina of Monro, the third ventricle and the aqueduct of Sylvius into the fourth ventricle, and so through the foramen of Magendie into the subarachnoid space, where it surrounds the whole of the brain and spinal cord.

As the nutrient vessels enter the substance of brain and cord they invaginate the pia mater before them, and so there are formed around them perivascular spaces which are continuous with the subarachnoid space and contain cerebro-spinal fluid. There is some evidence to show that the fluid in these perivascular spaces plays the part of a medium of exchange between the blood and the nerve cells analogous to that played by the lymph in other structures of the body, and that the waste products of nervous metabolism are by this route drained into the cerebro-spinal fluid. The fluid obtained from lumbar puncture will therefore contain such waste products in addition to the pure secretion of the choroid plexus.

The path of absorption of this fluid into the blood stream is by way of minute invaginations of the arachnoid membrane into the walls of the venous sinuses of the brain (arachnoid villi), and the rate of absorption is normally parallel with that of excretion.

Examination of the cerebro-spinal fluid in disease often yields information of the greatest value in diagnosis.

**Pressure.**— Increased pressure of the fluid is most commonly due to acute inflammatory changes in the meninges, with consequent outpouring of exudates into the subarachnoid space. It may also be due, as in certain cases of hydrocephalus, to the rate of absorption in the arachnoid villi falling behind that of secretion.

**Blood** in the spinal fluid taken for examination may come from a vein accidentally wounded at the moment of lumbar puncture. It may, on the other hand, be an indication of hæmorrhage into the subarachnoid space. A useful point of distinction is that blood from an accidental source, if left to stand, will coagulate in the test tube, leaving a colourless supernatant fluid. In the case of a pre-existing subarachnoid effusion coagulation will probably have taken place already at the site of hæmorrhage, and some hæmolysis will also have occurred.

If the blood-stained fluid is allowed to stand the red cells settle to the bottom of the tube without the formation of a clot, and the supernatant fluid is coloured yellow or brown from the presence of altered blood pigment.

**Bacteriology.**—In cases of acute infective meningitis the causal organism may often be seen in smears, and may nearly always be grown from cultures. When tuberculous meningitis is suspected the bacillus should be sought for and is often found, or the fluid may be injected into a guinea-pig.

**Cell Content.**—The number of cells per cubic millimetre is of great importance. The normal value is below 5 ; any rise above this figure must be considered pathological, and is usually evidence of acute or chronic inflammatory changes in the meninges.

In cases of acute meningitis due to the meningococcus, pneumococcus, streptococcus, etc., the increase is in polymorphonuclear leucocytes, which are often sufficiently numerous to give the fluid a turbid appearance.

In tuberculous meningitis the majority of the cells are lymphocytes, usually in large numbers.

In acute poliomyelitis during the first week of the illness there is an excess of lymphocytes varying from 15 to 100 per cubic millimetre, and in encephalitis lethargica there appears to be a similar increase in the majority of cases.

In the various forms of cerebro-spinal syphilis there is an almost constant increase in the number of lymphocytes, ranging from figures just above the normal to 200 or 300 per cubic millimetre, the number depending upon the stage of activity of the disease and the degree of meningeal involvement.

An excess of lymphocytes may occasionally accompany a sarcomatous tumour of the brain or cord.

**Albumin Content.**—This may be roughly estimated on a quantitative scale by precipitation by boiling 2 c.c. of the fluid with 0·3 c.c. of 30 per cent. trichloracetic acid and comparing the density of the coagulum with that of standard tubes containing known percentages of albumin precipitated in the same way. The normal content is 0·025 per cent. This is increased in fluids from patients with acute infective meningitis as a rule to about 0·3 per cent., sometimes higher. In cerebro-spinal syphilis there is nearly always an increase, though the figure is not as a rule higher than 0·10 per cent. There is also a moderate increase in the other diseases enumerated in which raised cell counts are found.

The highest amounts of albumin, however, are found in compression of the spinal cord. When, from any cause, the subarachnoid space is completely occluded at a level above that at which lumbar puncture is performed, the fluid obtained from the lower secluded sac has certain definite characteristics. It is clear, of a golden yellow colour, contains an enormous excess of albumin—up to 4 per cent.—and fibrin, so that it undergoes massive spontaneous coagulation. This is known as *Froin's syndrome.* The presence of simple excess of albumin in quantity greater than that met with in other conditions, without the golden colour or fibrin, may precede the appearance of the Froin syndrome, or appear as a stage in its retrogression.

The occlusion of the subarachnoid space may be due to disease of the vertebræ (caries or growth) ; fibrinous or cicatricial adhesions of the meninges, as after an acute meningitis or in syphilis; or the growth of a tumour from one of the meninges or within the cord. The cerebro-spinal fluid obtained from above the level of the block shows in every case a relatively normal appearance and albumin content. The Froin syndrome may be produced experimentally in animals by artificial compression of the cord with subdural injections of paraffin wax.

The cell count in Froin's syndrome depends on the pathological nature of the compressing cause, being high in the case of syphilis, low in all other cases.

**Chlorides.**—The percentage of chlorides in normal cerebro-spinal fluid is

constantly between 0·70 and 0·76 per cent. This may be slightly lowered in acute general infections, but a figure below 0·68 per cent. as a rule indicates meningitis. The lowest percentages are found in tuberculous meningitis, in which a figure of 0·60 per cent. or less is common. The point is, therefore, of considerable value in differential diagnosis.

The estimation is made by titration with standard silver nitrate, using potassium chromate as an indicator. Two cubic centimetres of cerebro-spinal fluid are required for the examination.

**Wassermann Reaction.**—This is positive in the cerebro-spinal fluid at an early stage in every case of dementia paralytica, and in 75 per cent. of tabetics.

As regards cases which show clinical evidence of meningo-vascular syphilis the reaction is positive in a large majority of those in which the spinal cord is involved, not so constant when the disease is confined to the brain.

**The Colloidal Reactions.**—It has been found that the cerebro-spinal fluid in certain diseases contains substances (probably foreign proteins) which, when added to a colloidal suspension, cause precipitation (**1**). Experiment has shown that the constancy and degree of precipitation vary in different diseases, and this knowledge has been applied as an aid to differential diagnosis. The suspension employed by Lange, who introduced the test, was of gold chloride, but equally good results are obtained with benzoin, which is less expensive and less liable to spontaneous precipitation. The test is carried out as follows: Six small test tubes are set up in a rack: each contains the same amount of colloidal benzoin; each of the first five tubes also contains a small quantity of the cerebro-spinal fluid to be tested, the dilutions ranging from 1 in 2 in the first tube to 1 in 32 in the fifth; the sixth tube, containing no cerebro-spinal fluid, serves as control. The rack is then left at room temperature and the tubes are inspected at the end of six hours. With a normal cerebro-spinal fluid there is no precipitation, and the result of this test is expressed thus, 00000 for the series of five tubes. Partial precipitation in a tube is expressed as 1, complete precipitation as 2. In syphilitic encephalitis (general paralysis of the insane) the rule is that at the end of six hours precipitation is complete in all tubes, this being expressed 22222; on account of its constancy in this disease, and the extreme rarity of its occurrence in any other condition, this is known as the "paretic" reaction. In tabes and other forms of neuro-syphilis, as well as in some other conditions (meningitis, polyneuritis (Payne)), precipitation may be partial in some tubes of the series, complete in others. The chief clinical value of the test lies in the value of the "paretic" reaction in a suspected case of syphilitic encephalitis (*see* p. 863)

# DISEASES OF THE NERVES

## NEURITIS

**Ætiology and Pathology.**—Although the term neuritis properly implies inflammation, it is widely used in a clinical sense to describe the symptoms which result from all kinds of disease of the peripheral nerves and nerve roots, including such diverse causes as infection, intoxication and trauma.

Of these *trauma*—whether from a single severe blow, from repeated minor injuries or from stretching—will produce a local effect involving damage to the nerve sheath and more or less damage to the nerve fibres. Local injury to a nerve fibre which stops short of destruction results in temporary disturbance or loss of function with rapid recovery. Local destruction, on the other hand, leads

inevitably to secondary descending degeneration followed by the downgrowth of new fibres from above—a process which must occupy a considerable period of time, varying with the length to be traversed by the new fibres from site of injury to periphery.

*Local infection* of a nerve may occur as the result of direct spread from inflammation in neighbouring structures—bone, muscle, fascia, etc. *New growths* frequently compress, and sometimes infiltrate, nerves in their vicinity.

In all these instances of local damage the nerve is affected in its whole thickness, and, since the connective tissue elements comprising the nerve sheath bear the brunt of the injury, the process is sometimes called an *interstitial* neuritis to distinguish it from cases in which the neuro-fibrils themselves are directly injured by a selective poison. This latter variety is sometimes called *parenchymatous* neuritis.

Bacterial or chemical poisons which have a selective incidence upon the nerves, are, as a rule, conveyed thither by the blood stream. Their action, therefore is as a rule symmetrical, often multiple.

In certain cases, however, bacterial poisons emanating from an infective focus may travel up the perineural lymphatic sheaths, and gain entrance to the neural elements at the point where the posterior nerve root enters the spinal cord. The work of Orr and Rows in this connection has already been mentioned, as also the evidence for supposing that the initial neuritis of diphtheria is due to a lymphogenous intoxication of this kind. Occasionally one meets with other examples such as an ulnar or median neuritis developing after a septic finger.

**Symptoms.**—Damage to nerve fibres, whether due to injury or disease, causes disturbance of sensation, muscular power and bulk, and vasomotor control. The injury to the lower motor neuron results in wasting, weakness and flabbiness of the muscles supplied. The tendon jerks of the affected muscles are diminished or lost owing to the loss of tone, and damage to the reflex arcs concerned. The disturbance of sensation takes the form at first of uncomfortable tingling (pins and needles) in the sensory distribution of the nerve, followed by anæsthesia. There may also in the early stages be cramp-like pains in the muscles. The trophic and vasomotor changes have already been referred to.

In addition to these symptoms in the distribution of the nerve there are in cases of local damage, pain, tenderness and sometimes swelling at the site of the disease.

The pain, as in other inflammatory conditions, is usually worst at night, and it is increased by movements or positions that cause stretching of or pressure on the nerve.

In severe lesions of motor nerves the electrical reactions are altered (*see* p. 732).

The duration of neuritis is very variable; slight cases recover quickly, but others may last weeks or months.

In recovery from injury to a nerve or after reunion of a divided nerve signs of returning function should appear within three months. With the return of sensibility pin-prick and extreme degrees of heat and cold are appreciated before cotton wool touches. The return of power in the paralysed part generally precedes any decided improvement in the electrical reactions. In severe lesions with extensive atrophy of muscle, R.D. persists for some time, but after some weeks the irritability of the muscle to galvanism also diminishes, and finally becomes extinct.

**Diagnosis.**—The symptoms caused by lesions of the most important nerves and their branches are described separately. Multiple neuritis also merits separate consideration. For the rest the diagnosis of neuritis depends upon careful inquiry into the distribution of sensory disturbance and weakness, and an accurate knowledge of anatomy.

**Treatment.**—The first indications are to discover and treat the cause, if possible, and keep the affected part *at rest* in such a way as to avoid all irritation

of the nerve, and in such a position as to prevent undue stretching of the muscles paralysed (*e.g.* wrist-drop or foot-drop). For relief of pain heat should be applied locally. Hot-water bottles frequently applied provide the simplest means. Irradiation from a high candle-power electric lamp in a metal reflector is also useful. Diathermy is efficacious, but since its application involves the patient's travelling to and from the apparatus for each treatment, it is useful rather in chronic cases than in the acute stage, when rest is essential. Internally aspirin in full doses is the most effective analgesic, and may be given freely. If, as sometimes occurs, the aspirin causes dyspeptic symptoms, double doses may be given per rectum. In cases in which the cause appears to be of a gouty nature (*i.e.* defective metabolism with poisonous by-products) atophan should be tried. Guiacum resin may be given with the same purpose. Massage to the affected muscles should be prescribed except in cases where pain is a prominent symptom. In these active and passive movements of the neighbouring joints once a day will suffice until massage can be borne without pain. The electric current should be regularly employed to stimulate such muscles as cannot be exercised by the will. This is the sole indication for its use.

## MULTIPLE NEURITIS

(*Peripheral Neuritis, Polyneuritis*)

**Ætiology.**—It is in only a minority of cases that the cause can be definitely traced. The known causes may be divided into: (1) Poisons introduced into the body from without. Of these the best known are alcohol and arsenic. Carbon bisulphide, carbon monoxide and dinitro-benzol may also be responsible. Lead is often included in this category, but acts rather upon the anterior horn cells than the fibres of the peripheral nerve (p. 680). (2) Poisons arising within the body. Of these the commonest is hyperglycæmia in diabetes. Beri-beri may also be included in this class, as also certain rare instances in which polyneuritis and hæmatoporphyrinuria are associated. Probably a number of the cases of obscure origin are caused by toxins arising in the body as the result of faulty metabolism, and these are sometimes spoken of as "gouty" or "rheumatic." (3) Bacterial toxins. The common instance is diphtheria, but polyneuritis has been recorded as a rare complication of many other infective fevers. (4) There appears to be a special liability in cachectic states such as accompany malignant disease and tuberculosis.

**Morbid Anatomy.** The characteristic picture is of degenerative changes in the nerve fibres, without affection of the nerve sheaths. There is thus no abnormality visible to the naked eye, but microscopic examination by the Marchi method shows fragmentation and fatty degeneration of the myelin sheath. The axis cylinders are less affected. These degenerative changes are most marked in the peripheral nerve twigs, being slighter as one approaches the spinal cord, and the anterior roots are usually normal.

**Symptoms.**—The clinical picture of polyneuritis varies somewhat with the cause in operation. The following description applies to the majority of cases.

The onset, as a rule, is gradual, with complaint of "tingling," or "pins and needles" in the toes and fingers. These subjective sensations of discomfort gradually spread upwards, but do not usually advance higher than the knees and wrists. They are commonly accompanied by occasional "cramps" of a painful nature in the muscles of leg and forearm. Motor symptoms as a rule follow the sensory disturbance. There is weakness, especially of the dorsiflexors of the feet with consequent foot-drop, and in walking the knees are lifted high in order that the hanging toes may clear the ground ("steppage gait"). The movements of the fingers are impaired so that the grip is weakened and fine movements, such as doing up buttons, become impossible. If the weakness extends higher in the

arms the extensors of the wrists are usually next affected with the appearance of "wrist drop." The circulation in the affected extremities is often enfeebled so that they become cold and blue.

On examination it is found that cutaneous sensibility is impaired in the areas in which numbness and tingling are complained of, the degree of impairment being less as one proceeds upwards from the periphery. Deep sensibility is less affected, and the muscles are frequently unusually tender to pressure. The enfeebled muscles are flabby, and if the disease has been present for more than two or three weeks, show wasting, which is most evident in the small muscles of feet and hands, and the calves of the legs. The tendon jerks in the earliest phase of the disease are temporarily exalted. Later they are diminished or lost, the earliest to disappear as a rule being the ankle jerks; loss of knee jerks follows if the case is a severe one, and the supinator jerks also are affected if the upper limbs are extensively involved.

The symptoms are usually symmetrical, but may commence in one extremity before the others. The lower limbs are, as a rule, more severely affected than the upper.

The cranial nerves are not affected, and there is no loss of sphincter control.

In some cases, however, although the initial complaints are very much the same the clinical picture develops in a different form. Deep sensibility—including sense of position and passive movement, and the vibration sense—is extensively affected, and muscular weakness is more widely and more evenly distributed. The patient then has difficulty in estimating the extent and direction of his movements, and therefore staggers in his gait, flinging his lower limbs about in a manner reminiscent of tabes. This clinical variety is known as *ataxic polyneuritis* and is probably due to an extension of the toxic effect to the posterior columns of the spinal cord.

Polyneuritis is sometimes accompanied by a peculiar condition of mind, which is most frequent in alcoholic neuritis, but also occurs, as was first observed by Korsakow, in multiple neuritis from other toxic causes (puerperal septicæmia, typhoid, influenza). He therefore called it *psychosis polyneuritica*, and it is also known as *Korsakow's disease*. The patient suffers from loss of memory for recent events, is ignorant of his whereabouts, has false memory, and describes events which have never happened. Thus he will be unable to tell his name, age, the day of the week, or where he comes from; but, on the other hand, he may say that he has been for a walk, a ride, or has seen certain friends, in obvious antagonism to facts. There may be a stage of talkativeness, or even delirium; but in advanced cases the patients show extreme apathy, and complete indifference to surroundings.

In cases of known causation there occur variations from this description, which in some instances are characteristic. Those in the case of diphtheria and lead-poisoning are described on pp. 68, 680.

*Alcoholic Neuritis* occurs more often in women than in men, and commonly results from frequent slight excess. It is at present a rare disease in this country. Other signs of chronic alcoholism, such as dyspepsia and cirrhosis of the liver, may or may not be present. The lower limbs are more seriously affected than the upper and may be involved alone.

Complaint of painful cramp in the calves and tendencies of the same parts to pressure are prominent symptoms. Of the cranial nerves the vagus is sometimes affected, leading to persistent tachycardia, and (rarely) to laryngeal paralysis. Nystagmoid jerkings of the eyeballs are often present (due probably, to slight weakness of the motor nerves), and the facial muscles may also be involved.

*Arsenical Neuritis.*—This occurs occasionally as a result of continued full doses of arsenical preparations given medicinally. In the year 1900, in Manchester and some other towns in the north of England, a number of cases of

neuritis, at first thought to be due to alcohol, were shown to be caused by the accidental impregnation of beer with arsenic in the process of brewing.

The distribution and character of the sensory and motor symptoms are much the same as in alcoholic neuritis, but there are in the arsenical form greater cutaneous hyperæsthesia, more frequent affection of the facial muscles and lower intercostal muscles, earlier atrophy, more frequent inco-ordination, and more rapid progress (Judson Bury), and extreme sensitiveness of the muscles to pressure (Reynolds).

*Diabetic Neuritis.*—Although the complete clinical picture of polyneuritis may be met with in this disease, more commonly it is found that the ankle and knee jerks are diminished or absent without any sensory complaint or localised muscular weakness.

*Acute Febrile Polyneuritis.*—This is a somewhat rare illness, in which preliminary symptoms of constitutional disturbance with pyrexia are rapidly followed by those of polyneuritis, without any discoverable cause. The cranial nerves are frequently involved, especially the seventh, and if, as often occurs, this affection is bilateral, a remarkably expressionless facies results. Superficial sensibility is little affected, nor is pain at all a prominent feature, but there may be considerable loss of the senses of position and passive movement. The weakness is more widespread and more evenly distributed than in most cases of polyneuritis caused by known poisons. There is some risk to life from broncho-pneumonia in the early stages, if the muscles of deglutition are involved, but after two or three weeks steady improvement sets in, and complete recovery may be expected.

*Family Hypertrophic Neuritis.*—This is a rare disease occurring in the members of the same family, in which there is a great thickening of the peripheral nerves due to replacement of the nervous elements by proliferating fibrous tissue. This process may extend to the posterior nerve roots, with consequent secondary degeneration of the posterior columns. The essential clinical features are symptoms of peripheral neuritis in the form of numbness, wasting and weakness of the extremities, with loss of tendon jerks, together with palpable enlargement of the nerve trunks, which may appear double their usual diameter. In addition to this, there have been present in most of the cases recorded signs of more widespread disease of the nervous system, of which the most prominent are myosis with Argyll-Robertson pupils, lightning pains, ataxia, tremors and dysarthria.

**Associated Conditions.**—As neuritis is frequently caused by poisons from without, such as alcohol, arsenic, and lead, its symptoms are often associated with others due to the particular poison concerned: thus in alcoholic cases cirrhosis of the liver may be present; in arsenical cases, various lesions of the skin, viz. pigmentation, keratosis or hypertrophy of the epidermis, erythema and herpes zoster. Cardiac failure with œdema occurs with the neuritis of beri-beri, and may be present in arsenical, alcoholic, and diphtherial cases.

**Diagnosis.**—The early symptoms of polyneuritis bear a close resemblance to those of *subacute combined degeneration*. The peripheral numbness and weakness and loss of ankle and knee jerks are common to both. In combined degeneration, however, symptoms of spinal cord disease are usually present in the form of girdle pains, sphincter disturbance, or extensor plantar responses. Lesions of the lumbar enlargement of the spinal cord or cauda equina may also produce weakness of the lower limbs with absent tendon jerks, but here again sphincter disturbance and the segmental distribution of the anæsthesia are points of distinction. Some cases, especially those of the ataxic form of polyneuritis need to be distinguished from tabes. But in the latter disease there is usually a history of lightning pains, quite unlike the sensory complaints of neuritis, the arms are rarely affected at the same time as the legs, and the calf muscles, so far from being tender, are unusually insensitive to pressure. Other signs of tabes, especially Argyll-Robertson pupils, are also of use in the differential diagnosis.

**Prognosis.**—Alcoholic cases may be fatal either in an early stage

where the cause is not promptly removed, or after months from emaciation, or bedsores, or phthisis. In many cases the symptoms develop rapidly in the first six or eight weeks, and then the condition of the patient may remain stationary for months, or very slowly improve. After years recovery may be only partial. But in alcoholic and in other cases where the disease is not too far advanced, and the cause can be completely removed, the prognosis is more favourable, recovery taking place slowly in the course of from two to six months. In the cases of unknown origin complete recovery is the general rule, although relapses may occur.

**Treatment.**—If any external poison such as alcohol or arsenic is the cause, it must be henceforth kept from the patient. In diabetic cases the cause should be treated. In acute and severe cases complete rest is sometimes necessary, at first on account of the muscular pains. As soon as possible massage and active and passive movements should be commenced: in milder cases these measures should be employed from the beginning. In very slight cases it is not necessary that the patient should go to bed, but he should be warned against fatiguing the affected limbs. Splints are frequently necessary to prevent contractions. In severe cases these must be worn continuously. At a later stage of the illness, or in less severe cases, it will suffice that they should be worn at night only. The electric current should be used to stimulate muscles which cannot be exercised by the will.

Of drugs, aspirin is useful for the pains of onset: atophan may be given in cases suspected to be of gouty causation: and a strychnine mixture should be prescribed during convalescence.

## NEUROMA

Growths in the nerves may consist of nerve tissue (true neuroma) or of the same tissues that form tumours in other parts (false neuroma). True neuromas are very rare and are only found in connection with the sympathetic nervous system. Under the heading of false neuromas are included all tumours, whether benign or malignant in character, which originate from the connective tissue elements of the peripheral nerves.

The commonest of these are the *neurofibromas*, which are usually multiple and are often associated with von Recklinghausen's disease (*see* p. 1017). They may arise from the peripheral nerves, from the nerve roots within the spinal theca, or from the cranial nerves. Of the latter, the eighth nerve is most frequently the starting point for one of these tumours (*see* p. 856).

*Plexiform neuromas* are of the same pathological type as the neurofibromas, but are softer and more diffuse. They are commonly confined to a single area of the body, where they form a nodular mass of interlacing cords.

The malignant tumours arising from nerve sheaths are, as a rule, sarcomas, spindle or round-celled.

The term *amputation neuroma* is often used to denote the inflammatory nodule which sometimes forms upon the end of a cut nerve in an amputation stump, and is a source of much pain and tenderness.

The **Symptoms** are pain, anæsthesia, numbness, and tingling in the distribution of the nerve, and paralysis of muscles supplied by it, or more commonly reflex spasms in adjacent or even distant muscles. The tumour may be sometimes felt.

The **Treatment** is purely surgical.

## LESIONS OF CRANIAL NERVES

### OLFACTORY NERVE

A diminution or loss of the sense of smell (*anosmia*) arises from altered conditions of the nasal mucous membrane, such as excessive dryness, or coryza,

and in affections of the base of the skull involving the olfactory bulbs, such as injury, tumours, caries of the bone, and meningitis. It sometimes occurs in tabes dorsalis, and is not uncommon in hysteria, as a part of hysterical hemi-anæsthesia. It has sometimes occurred after excessive stimulation of the olfactory nerve by strong odours. It should be remembered that loss of smell may affect the power of appreciating flavours, which really requires the combined action of the sense of taste and the sense of smell through the posterior nares.

Excessive sensibility to odours (*hyperosmia*) is noticed in hysteria and insanity; and morbid subjective sensations occur in the insane, and sometimes as an aura in epilepsy.

The primary cause of these defects must be treated, if possible.

## OPTIC NERVE

The main points of anatomical importance in connection with this nerve and the methods of investigating its functions have already been referred to (pp. 724 and 712, Fig. 69).

**Affections of the Optic Nerve.**—With the ophthalmoscope it is not always possible to distinguish between *optic neuritis* and *papillœdema*, but it should be borne in mind that, despite their similarity, these two conditions are the result of quite different pathological processes.

*Papillœdema* is the term used to denote swelling of the nerve head (or papilla) due to the mechanical conditions produced by increased intracranial pressure, from whatever cause. The increased pressure, being transmitted to the sheaths of the optic nerves by way of the cerebro-spinal fluid in the subarachnoid space, gives rise to compression of the veins, with resulting transudation through the vessel walls at the point of least resistance, which is the nerve head; the same factors cause a blockage of the paths whereby this transudate might naturally be absorbed, so that the condition is a progressive one, and the papillœdema may increase until the highest point on the nerve head stands out 2 or 3 mm. in front of the general retina.

*Optic neuritis*, as the term implies, is an inflammatory condition of the nerve which may be caused by various infective or toxic agents. It is a comparatively uncommon condition. It may or may not involve the nerve head. If it does so, the appearance on ophthalmoscopic examination resembles that seen in papillœdema, and it should strictly be called *papillitis*. If it does not, the condition is spoken of as a *retrobulbar neuritis*, and at first gives rise to no changes recognisable with the ophthalmoscope, though later on the disc may show the pallor of optic atrophy from spreading degeneration of the nerve fibres and their replacement by fibrous tissue.

The causes of *papillœdema* are those which give rise to increased intracranial tension, of which cerebral tumour is the commonest. The causes of true *optic neuritis* are generalised infections or toxic affections, of which malignant endo-carditis, Bright's disease, leukæmia and lead-poisoning may be mentioned as examples. A unilateral optic neuritis may be produced by the spread of inflammatory changes in the orbit to the nerve.

*Optic Atrophy.*—This may be produced as the result of various processes, of which the most important are:—

1. Degeneration of the nerve fibres as the result of *syphilitic infection;* the disc is of a greyish pallor with clear-cut edges; this is frequently seen in tabes and dementia paralytica.

2. Degeneration of the nerve fibres secondary to a *retrobulbar neuritis;* this is most commonly seen in disseminated sclerosis, in which the disc shows a dead-white pallor, which is best marked on the temporal side.

3. Degeneration of the nerve fibres as the result of *pressure* upon the optic nerve or optic tract. In this case the disc presents an appearance of startling pallor with exceedingly clear-cut edges.

4. Degeneration of nerve fibres *secondary to papilloedema* with organisation of the exudate. The nerve head is dead white, but may still show some swelling; the edges are indistinct, the physiological cup is filled in, and there are whitish streaks of exudate along the course of the vessels.

Optic atrophy of the first three types mentioned above is often called *primary optic atrophy*, in order to distinguish it from the *secondary optic atrophy* following papilloedema. Vision is very greatly reduced or abolished in optic atrophy.

**Optic Chiasma and Optic Tracts.**—The optic chiasma may be affected by tubercle or syphilitic gumma or meningitis, by the pressure of internal hydrocephalus, or of tumours of the pituitary body, and by hæmorrhage into its substance. Since it contains the decussation of those fibres which pass from the nasal half of the right retina to the left tract, and those which pass from the nasal half of the left retina to the right tract, it follows that its lesions produce paralysis of the *nasal* half of each retina, and a corresponding blindness in the *temporal* half of each visual field; this forms a *double temporal hemianopia* (*see* Fig. 69).

If the lesion extends sufficiently to one side, or forwards in the optic nerve, or backwards in the optic tract, the direct fibres on that side are affected as well, and the vision of that eye is quite lost. Indeed, the lesion is often unsymmetrical, one eye being completely blind, the other blind only in its temporal half. In some such cases it is evident that the trouble begins with loss of vision in the temporal half of the worse eye. Clinically it may be very difficult to trace the cause of a hemianopia.

A *double nasal hemianopia*, or blindness in the inner half of each field, would result from a separate lesion on each side of the chiasma, involving the direct fibres to the outer half of the retina. It is necessarily very rare.

The optic tract contains fibres which pass to the visual centres, partly direct from the outer (temporal) half of the retina of the same side, partly across the chiasma from the inner (nasal) half of the retina of the opposite eye. A lesion of the *left* optic tract paralyses the left half of each retina, and causes blindness in the *right* half of each field; similarly a lesion of the right optic tract produces blindness in the left half of each visual field. This form of blindness is called *lateral* or *homonymous hemianopia*, in contradistinction to the above-mentioned double nasal and double temporal hemianopia, which are called *heteronymous* (*see* Fig. 69).

Lesions of the optic chiasma or optic tract lead to degeneration of the nerve fibres which extends to the optic nerve and papilla and is visible at the nerve head as optic atrophy (*see* above). The pupillary reaction to light is also affected. If a bright light be thrown upon the blind part of the visual field the pupils fail to contract.

**Optic Radiations and Occipital Cortex.**—The fibres of the optic radiation after leaving the primary visual centres (*see* Fig. 69, p. 712), spread out fanwise and in their course towards the occipital cortex in which they end, some of them proceed at first downwards, forwards and outwards, so as to pass into the temporal lobe, until they reach the inferior aspect of the descending horn of the lateral ventricle, where they run backwards to their destination. Thus a lesion of the temporal lobe may produce a defect in the opposite visual field.

The precise form of the visual defect produced by a lesion of the optic radiation or occipital cortex varies with the particular fibres affected. The fibres of the optic radiation are divided into an inferior bundle which terminate below the calcarine fissure, and a superior bundle which ends above it. Lesions of the inferior bundle of the optic radiation or lower part of the visual cortex produce defects in the upper quadrant of the opposite visual field, and *vice versâ*.

Lesions of the optic radiation or visual cortex may be produced by tumour, or may result from vascular disease or encephalitis. The transient hemianopic disturbance which sometimes occurs in migraine must be remembered in this connection.

In the case of these lesions, which are above the primary visual centres (*see* Fig. 69, p. 712) the pupillary reactions to light are not interfered with, and a bright light thrown upon the dark part of the visual field, though invisible to the patient, will still cause contraction of the pupils.

A patient with lateral hemianopia is likely to keep his head turned towards the dark half of the field, in order to see distinctly things in front of him. If the left side of the field is dark, the right half of each retina is paralysed; if then the head is turned to the left, objects in front of the patient fall upon the left, or normal, half of each retina.

### THIRD, FOURTH, AND SIXTH NERVES

The lesions causing ocular paralysis may affect—

1. The trunks of the nerves. Here the lesions are syphilitic and so-called rheumatic inflammations and toxic neuritis; the pressure of orbital or intracranial growths, or of aneurysms, and rarely tumours of the nerves themselves; direct injury; and inflammation or suppuration spreading from the middle ear. If all the muscles supplied by the third nerve are paralysed on one side, without the fourth or sixth being involved, the lesion is behind the cavernous sinus. If this condition is associated with paralysis of the opposite side of the face and body (symptom-complex of Weber), it may be due to a lesion of the inner side of the crus cerebri. If all three nerves are concerned on one side, the lesion is in the orbit, in the superior orbital fissure or in the cavernous sinus.

2. The nerve fibres in the brain connecting the nerve trunks with the nuclei, such as hæmorrhage, softening, tumours, and disseminated sclerosis.

3. The nerve nuclei.

Paralyses of ocular nerves occur in connection with some diseases without our being able to say with certainty what is the seat or the nature of the lesion. Diphtherial paralysis, admittedly due to peripheral neuritis, often begins with strabismus from paralysis of an ocular muscle, and with loss of accommodation. In alcoholic peripheral neuritis, the third nerve may be involved, or the sixth; and to neuritis of the latter the jerking movements on deviation of the eyes may be attributed (James Taylor). In tabes dorsalis, paralysis of the third, fourth, or sixth nerve occurs, which may be permanent or transient. Syphilis may act by causing meningitis or gummatous thickening, and may contribute to the formation of aneurysms, which press upon ocular nerves. Nuclear degenerations also occur with some frequency in syphilitic subjects.

Cases of *relapsing* or *recurring paralysis* have been described. They begin with pain in the eye, often with headache and vomiting. These last two or three days, and coincidently, or as they lessen, there is paralysis of several ocular muscles, internal and external. Ptosis is generally present, and in many cases the third nerve is alone affected, but in some the sixth nerve is also or alone involved. The paralysis lasts a few days or weeks, and recurs at intervals of months or a year; but in some rare cases it persists. The disease lasts from early life to middle age. In more recent times this complaint has been called by French writers *migraine ophthalmoplégique*.

**Ocular Paralysis from Disease of the Nuclei.**—Ophthalmoplegia is a common result of disease of the nuclei. The acute form is most frequently seen in encephalitis lethargica. Chronic ophthalmoplegia occurs in association with locomotor ataxy, and in syphilitic subjects, and also with progressive muscular atrophy, bulbar paralysis, and general paralysis of the insane: it is gradual in its course; first one or two muscles, then others, and finally, after some years, nearly all the muscles, are affected. Ptosis, however, may be absent, and is rarely complete. If present, it gives the patient a sleepy look; the eyeballs are fixed or staring, and sometimes they are slightly prominent. Double vision may be present in the early stages, but often disappears in

the course of time. Degenerative changes have been found in the ocular nerve nuclei. A nuclear lesion is certainly present if the internal muscles are alone paralysed in both eyes, or if the external muscles are paralysed in both eyes without the internal, or if associated muscles in the two eyes are paralysed at the same time. But if both external and internal muscles are paralysed in both eyes, coarse disease of the base of the brain may be a cause, though nuclear lesions are far more probable

A lesion of the oculo-motor nucleus at its anterior or upper part will cause an internal ophthalmoplegia; one of the lower part of the nucleus will paralyse the external muscles.

The centre in the pons for *conjugate deviation* of the eyes to one side, *e.g.* to the right, is in the neighbourhood of the sixth nerve nucleus on the right side. This innervates the right external rectus directly. It also sends a branch up through the right third nerve nucleus and across to the left third nerve nucleus and from there to the left internal rectus muscle. Consequently a lesion on one side of the pons affecting the nucleus of the sixth nerve causes *conjugate deviation* of the eyes to the opposite side (*see* Hemiplegia). A lesion of the sixth nerve nucleus is often accompanied by some facial paralysis from the close proximity of the facial nerve fibres, which wind round it.

**Ptosis.**—Besides ptosis due to lesions of the fibres and nucleus of the third nerve, there are other forms: ptosis from paralysis of those fibres of the levator palpebræ superioris which are supplied by the sympathetic; *congenital ptosis*, probably due to a central defect; ptosis in weakly adults, especially women, which is worse in the morning; ptosis in myasthenia gravis; and, finally, *hysterical ptosis*. This last affects both sides; the head is thrown back, and the frontales may be strongly contracted when the patient tries to look up. But the attempt is accompanied by a spasmodic movement of the orbicularis, which prevents the action of the levator.

**Treatment of Ocular Paralysis.**—This must depend upon the cause, if it can be ascertained. Syphilitic cases—and they form a very large proportion—should be treated by salvarsan and potassium iodide and mercury. For cases of an inflammatory nature counter-irritation by a blister behind the ear or leeches to the temple should be tried. If the paralysis has appeared to follow after exposure to cold, hot fomentations should be applied, and salicylates may be given internally. The so-called rheumatic cases, however, tend to recover without treatment. Diplopia may be relieved by the use of a prism, which should not be strong enough to fuse the images, but only to approximate them, so that muscular efforts may be encouraged. For ptosis in weakly individuals tonics, such as iron, quinine, and strychnia, are required, as well as local stimulants. In the hysterical form similar remedies, and especially stronger stimulants, such as blisters to the temples, and faradism, should be used; if one eye only is affected, the other eye should be bound up

## FIFTH NERVE

The fifth nerve may be injured in any part of its course. In the pons its origin may suffer from tumours or hæmorrhage; its trunk may be affected by tumours or meningitis at the base of the brain; in front of the Gasserian ganglion the first division is liable to pressure, from the tumours about the cavernous sinus, or aneurysms or cellulitis in the orbit; the second and third divisions may be injured by growths in the spheno-palatine fissure. Injuries to the mouth or nose may involve various branches of the second and third divisions, and neuritis of these branches may be caused by neighbouring inflammations.

**Symptoms.**—These must depend on the position of the lesion, and whether it involves the fibres of sensation, taste, or motion. If the *sensory* fibres are involved, the result is anæsthesia of the face, corresponding to the distribution of the nerve.

The loss of sensation is often preceded by tingling and numbness or neuralgic pains, and there may be tender points like those found in ordinary neuralgia; it may be accompanied at first by sensitiveness to pain, but this also is finally lost. The conjunctiva and the nasal and buccal mucous membrane are, of course, involved as well as the skin. The nose is insensitive to the stimulus of pungent vapours like ammonia, and though smells are at first perceived well, the sense becomes afterwards blunted. In consequence of the mouth being insensitive on one side, food is not chewed on that side, and a thick fur collects on the tongue for want of the cleaning operation of mastication.

Certain *trophic* changes also occur in lesions of the fifth nerve; the secretions of the mucous membranes are diminished, there may be swelling and ulceration of the gums, and the teeth become loose; if the cheek is bitten it heals slowly. Often the cornea becomes inflamed (*neuro-paralytic keratitis*); this begins on the lower side, with cloudiness, opacity, and ulceration, by which eventually the eye may become perforated and destroyed. It has been attributed to irritation of the insensitive surface by foreign bodies, but also to lesions of the nerve root, and of the Gasserian ganglion. Herpes zoster occurs especially in connection with the first division (*Herpes zoster ophthalmicus*), and appears to arise from inflammation of the Gasserian ganglion.

If the *motor* portion of the fifth nerve is involved, which is only likely to happen in lesions near the origin of the nerve, the temporal, masseter, and pterygoid muscles are paralysed, and after a time atrophy of the temporal and masseter muscles may be recognised.

Lesions of the sensory root between the ganglion and the pons usually give rise to progressive numbness and anæsthesia without pain. In the case of lesions of the ganglion itself or its branches anæsthesia is preceded or accompanied by pain. Paroxysmal trigeminal neuralgia is described on p. 885.

**Diagnosis.**—The presence of severe pain may give for a time a resemblance to neuralgia, but anæsthesia, diminution or loss of corneal reflex, or wasting of the muscles supplied by the motor root prove an organic origin. If one or other branch is alone affected the lesion is in front of the Gasserian ganglion; if all the branches, it must be near the origin. The association of other nerve paralyses, such as those of the ocular nerves, or of the motor tract, may also help to localise the lesion.

**Treatment.**—Besides dealing with the cause where this is possible, relief from pain may be obtained by injection with alcohol of the Gasserian ganglion or its branches.

### SEVENTH OR FACIAL NERVE

The pathological interest of the seventh nerve chiefly centres in its motor fibres, and its tortuous course from the pons through a bony canal to its distribution on the face renders it especially liable to inflammation and compression. Paralysis of the facial muscles, indeed, may be caused by lesions, not only of the facial nerve itself, and of the nerve nucleus, but also of the facial portion of the cortical centres, and of the fibres which connect this with the nucleus. These last, supra-nuclear, lesions cause a limited form of paralysis, which will be described with *hemiplegia*. We have here to do with the more complete nuclear and nerve trunk paralysis which goes sometimes by the name of *Bell's palsy*.

**Causes.**—The nucleus and the fibres in the pons may be involved in tumours in that part of the brain, and occasionally the nucleus is degenerated as a part of true *bulbar paralysis*. Tumours of all kinds at the base of the brain and meningitis may involve the nerve trunk between the brain and the internal auditory meatus. In the petrous bone the nerve is liable to injury from otitis, and suppuration of the mastoid cells; and rarely hæmorrhage has compressed the nerve in the aqueduct of Fallopius. On the side of the face the nerve may be injured by blows, or may be involved in cellulitis or parotid growths. The

more common cases of facial paralysis often come on after exposure of the face to a draught, as by sitting at a window, but sometimes without any recognisable cause. Such cases are attributed to neuritis, which probably affects the nerve at its emergence from the stylo-mastoid foramen. Cases of facial paralysis have been recorded after herpes zoster of the face, when it must be supposed that the neuritis first affecting the branches of the sensory fifth nerve has spread to those of the motor seventh. Facial paralysis is commonly unilateral. Double facial paralysis (*diplegia facialis*) may occur from bilateral disease of the pons, or from double otitis, or from syphilitic lesions successively affecting both nerves. Diphtheria, syphilis by its toxins in the secondary stage, and influenza appear also to cause double facial neuritis. An incomplete, yet double, facial paralysis may occur in multiple neuritis, especially in that form already described as acute febrile polyneuritis.

The **Symptoms** of facial paralysis have already been described under the Examination of the Cranial Nerves (p. 728).

**Course.** Facial paralysis runs a variable course. The so-called rheumatic form (facial neuritis) often develops quite suddenly, and is complete in a few hours. It may gradually recover in a few weeks or months; it may recover only partially; or the face may remain permanently and absolutely paralysed. A partial recovery is often followed by contracture of the paralysed muscles. These are somewhat shortened, the eye is a little closed, and the angle of the mouth is slightly drawn up by the zygomatici; and if the muscles of the sound side are at rest, the first impression that one gets is that the paralysed side is active and that the sound side is paralysed. This idea is corrected at once when the patient speaks or smiles, or tries to shut the eyes. The contracted side can contract very little more, while the sound side has a wide range of movement. In this condition also the affected muscles cannot be moved independently - in closing the eye, the angle of the mouth is raised; in smiling, the eye is partially closed. This is called *secondary over-action*. In children facial paralysis is not so obvious as in adults, because elastic tissue plays a greater part in the facial expression of children, whereas in older people the muscles are all important.

**Diagnosis.** The recognition of facial paralysis is not difficult. The important point is generally to distinguish the seat of the lesion (*see* p. 817). In facial paralysis of *cerebral* or *supra-nuclear* origin, the muscles of the lower part of the face are mostly affected, those of the upper part very little; the wrinkling of the forehead is slight, and the eye can always be closed, though not so tightly as on the opposite side, and the eyeball is not rolled up under the upper eyelid as in peripheral paralysis. A smile started by emotion is less impaired than one voluntarily attempted, the electrical reactions of the affected muscles are normal or nearly so, and reflex contractions may be obtained. In a lesion of the facial nerve nucleus, the highest lesion causing *peripheral* paralysis, the orbicularis oris escapes, as the nuclear origin of the nerve of this muscle seems to be connected with that of the tongue. A tumour of the pons not infrequently involves the sixth and eighth nerves as well as the seventh, from their close proximity to one another, and disease in the internal auditory meatus must involve the eighth. Lower down the loss of taste will localise the lesion, since the chorda tympani will be involved, and it is through this nerve that sensations of taste are conveyed from the anterior two-thirds of the tongue. Rheumatic neuritis may begin below this section of the nerve, and involve it by extension.

**Prognosis.**—This is largely dependent on the cause. In facial neuritis an opinion may be formed from the reaction to electrical currents. If this is still normal after a week or ten days, recovery is probable; rapid and complete RD is unfavourable.

**Treatment.**—When an accessible tumour is the cause its removal should be attempted. Potassium iodide, mercury, and salvarsan should be used in

syphilitic cases. In ordinary rheumatic paralysis, warmth locally applied, counter-irritation by a blister over the mastoid process, and salines with potassium iodide should be employed. Electrical treatment may usefully be employed so long as the muscles cannot be exercised by the will. Massage of the facial muscles is useful. The flaccid muscles on the paralysed side may in some measure be supported in the position of rest by means of a piece of copper wire ensheathed in a suitable length of rubber tubing, the two ends being bent so that one fits over the ear and the other hooks into the angle of the mouth. In uncured cases of long standing, where healthy fibres still persist in the facial nerve trunk, the tone and voluntary power of the muscles have been restored, and deformity has been diminished by dividing the nerve and suturing its distal end to the proximal end of the divided hypoglossal nerve. The atrophy of the lingual muscles, thus deprived of their innervation, is obviated by uniting the distal end of the hypoglossal to a portion of the spinal accessory (Ballance).

## EIGHTH NERVE

Various cerebral lesions may involve the nuclei of the nerve in the pons, or its higher connections in the brain; the nerve itself may be injured by meningitis, by thickening of the petrous bone, by aneurysms or tumours; and the expansion of the nerve in the labyrinth may be damaged by acute or chronic inflammation, by syphilitic disease, or degenerative changes.

The results of these lesions are *deafness*, *vertigo*, and various subjective sounds, especially *tinnitus*. Excessive sensibility to sounds is more often a functional disorder.

**Deafness** from the above cause is called "nerve" deafness, and has to be distinguished from loss of hearing due to interference with the conduction of sound through the middle ear and external auditory meatus (*see* p. 725).

When the conduction is found to be normal, deafness must be due to a lesion either of the nerve or of the labyrinth. Which of these is more likely to be at fault must be determined by associated symptoms. These may sometimes point to an intracranial lesion, but, as a fact, deafness is not a very common symptom in cerebral cases, unless the trunk of the auditory nerve is directly compressed by a tumour.

**Tinnitus Aurium.** This term includes the various subjective sensations of sound, generally of a hissing, whistling, ringing, or roaring kind, with which, in their slightest degrees, nearly every one is familiar. It is clearly due to irritation of the auditory nerve fibres, and may occur in almost any form of disease of the ear, whether of the external meatus, of the middle ear, of the labyrinth, or of the nerve or nerve centres. Thus it may result from cerumen in the external meatus, is not infrequently present in acute and chronic inflammations of the middle ear, and is a prominent symptom in cases of tumour involving the eighth nerve between the internal auditory meatus and the pons. The commonest cause of tinnitus, however, is disease of the nerve endings of the cochlear apparatus. These may be affected temporarily by the action of certain drugs, especially quinine and the salicylates, or as a result of defective blood supply, *e.g.* in the secondary anæmia following a severe hæmorrhage. More frequently the tinnitus is associated with progressive deafness and is due to irritation of the nerve fibres as a preliminary to destruction. The cause in such cases may be disease of the bony capsule of the cochlea (otosclerosis), degenerative changes in the arteries leading to a permanently defective blood supply, or primary degenerative changes in the nerve fibres themselves as in the common form of Ménière's syndrome. In the last instance the vestibular nerve endings are also affected with resultant attacks of vertigo.

*Treatment.*—The cause of the tinnitus must be first considered. Disease of the external or middle ear should be directly treated. In the cases of obscure

origin the symptom may be alleviated by the avoidance of fatigue, especially of the mental order, and the prescription of sedatives such as bromide and luminal in small doses taken regularly.

**Vertigo.**—This term is commonly used to denote a sensation of giddiness, from whatever cause. Thus it may occur as a symptom of cardio-vascular disease, anæmia, chronic nephritis, arterio-sclerosis, or the increased intracranial pressure of cerebral tumours, and it is commonly experienced by a person getting out of bed for the first time after an illness. In all these conditions the sensations of giddiness are probably due to functional disturbance of the centres presiding over equilibrium, the cause being a failure of the blood supply to these parts of the brain.

A more direct cause is to be found sometimes in a tumour or localised inflammatory affection of these centres themselves, that is in the cerebellum, or pons varolii. Thus a patch of disseminated sclerosis in the latter region may give rise to severe attacks of vertigo, and the symptom is quite commonly met with in cases of cerebellar tumour.

It is, however, in direct affections of the vestibular nerve and its peripheral end apparatus that vertigo may occur as the most prominent and troublesome symptom, being, as a rule, accompanied by deafness and tinnitus.

Thus vertigo may result from involvement of the eighth nerve by tumours or from inflammation of the meninges; from destructive processes affecting one or other labyrinth, such as hæmorrhage, and acute and chronic infections; or from more trivial causes such as a difference in the pressure exerted upon the two labyrinths, which may be caused by middle ear catarrh and obstruction of the Eustachian tube, or even by wax pressing upon the membrana tympani.

The exact nature of the symptoms varies with the cause, but they are usually somewhat sudden in their onset and of transient duration with a tendency to recur. They may consist merely of a subjective sensation of giddiness, or the patient may reel and stagger during an attack, and be obliged to take hold of some fixed object for support. Severe attacks are almost always accompanied by nausea, and there is often vomiting.

In its most severe form vertigo is the result of an acute labyrinthitis secondary to an infection of the middle ear, or a hæmorrhage into the labyrinth such as may occur in cases of trauma, severe anæmia, or possibly arterio-sclerosis. In such an attack the patient is suddenly seized with the most violent giddiness, so that if standing he falls to the ground, and this is accompanied by nausea and vomiting. The severe symptoms last for a few days and subside gradually, leaving nerve deafness and tinnitus. By far the commonest cause of vertigo is a chronic progressive degeneration of the vestibular nerve endings. In these cases the cochlear nerve endings are also affected with resultant tinnitus.

The diagnosis of vertigo presents little difficulty when it is associated with complaints of tinnitus and deafness. In some cases, however, the attacks may need to be distinguished from those of minor epilepsy.

In the latter condition there is loss of consciousness which does not occur with vertigo. It is also necessary to bear in mind that neurotic patients often complain of attacks of giddiness which depend upon causes acting at a psychic rather than a physical level, and are associated with other symptoms of mental abnormality.

*Treatment.*—Vertigo is to be regarded as a symptom rather than a disease, and treatment must, therefore, be directed to the underlying cause when this has been ascertained. In cases due to chronic otitis media surgical treatment is required. In some instances syphilis is the cause of a destructive labyrinthitis and should be treated by the appropriate remedies. In those cases in which the pathology is obscure, potassium bromide appears to be of value in diminishing the frequency and the severity of the attacks, and should be given in doses of from 15 to 20 grains three times a day.

**Ménière's Syndrome.**—It has already been pointed out that the commonest cause both of tinnitus and vertigo is degeneration of the cochlear and vestibular nerve endings, and that both structures are as a rule involved together. The resultant combination of progressive nerve deafness, tinnitus and attacks of vertigo is known as Ménière's Syndrome after the French physician who first described it. In one of the first cases which Ménière observed the symptoms came on acutely. The patient died, and the appearances suggested a hæmorrhage into the labyrinth. In all probability this was a case of acute streptococcal infection. Such cases are extremely rare. The common story is of a very gradual onset of tinnitus and deafness, which begins in one ear, and after a considerable interval, usually of years, affects the other, the whole period being punctuated by attacks of vertigo. The patient eventually becomes completely deaf and the tinnitus and vertigo cease. The cause in these cases is still uncertain.

In some instances the blood pressure is low, while the arteries appear calcified (senile or Monckeberg's degeneration, *see* p. 367), and these patients are benefited by a stimulating *régime*, and strychnine tonics. In explanation of other cases it has been suggested that the condition is due to an increased pressure of the endolymph. Operation with the establishment of a permanent opening from the external semi-circular canal into the surrounding tissues has given relief in certain instances. Apart from this one can but relieve the symptoms by prescribing a quiet life, as free as possible from mental excitement and fatigue, together with regular small doses of bromide and luminal.

## NINTH OR GLOSSO-PHARYNGEAL NERVE

The nerve is rarely, if ever, affected alone: the nucleus is involved in cases of true bulbar paralysis, and the trunk is likely to be affected by tumours or other lesions of the medulla, in association with the root of the pneumogastric. In dysphagia, globus hystericus, and anæsthesia of the pharynx this nerve may have a share.

## TENTH, PNEUMOGASTRIC, OR VAGUS NERVE

This nerve has both motor and sensory fibres, the latter being connected with the ganglia at the base of the skull; while many of its motor fibres are contributed by the accessory portion of the spinal accessory nerve. The vagus is extensively distributed to the pharynx, larynx, lungs, heart, stomach, intestines, and spleen.

It is liable to still more lesions than the other cranial nerves from its great extent and varied course—*e.g.* lesions of its nuclei from degeneration, softening, or hæmorrhage, generally in association with the adjacent nuclei; of its roots from meningitis, syphilitic gumma, tumours, or aneurysm; of the nerve itself from wounds, surgical operations, aneurysms, new growths, or enlarged glands. The last three are frequently causes of difficulty with the pneumogastric in the thorax and the recurrent laryngeals in any part of their course. Diphtherial, alcoholic, and other forms of multiple neuritis also involve the functions of the pneumogastric nerve.

If the *pharyngeal* branches are affected, swallowing is difficult; the food lodges in the pharynx, and small portions of liquids may pass into the larynx and cause choking. The palate is also paralysed. If the *laryngeal* branches are diseased, various forms of paralysis of the vocal cords and other parts of the larynx are produced; and if the superior laryngeal nerve is involved, there is anæsthesia of the larynx (*see* Diseases of the Larynx). *Pulmonary* branches are both afferent and efferent; the afferent fibres regulate the depth of respiration in particular; and the efferent fibres are said to supply the muscular fibres of the bronchi. But it is only in rare instances that the results of lesions of these fibres are observed clinically. The phenomenon called Cheyne-Stokes respira-

tion and the spasms of hydrophobia are probably dependent on changes in the respiratory centres, with which the vagus nucleus must be connected. The *cardiac* fibres have an inhibitory action, and are believed to be involved in cases of alcoholic neuritis, when the pulse may become excessively rapid; a similar acceleration has occurred from local disease of the nerve trunk. Some curious cases are on record of slowing of the heart from irritation of the vagus by pressure. Lesions of the *gastric* branches seem to have caused in different cases pain, vomiting, or excessive appetite; the vomiting frequently observed in cerebral disease must be due to irritation of the vagal nuclei.

**Treatment** must be conducted on the lines indicated in the case of other nerves. (*See also* Diseases of the Larynx.)

### ELEVENTH OR SPINAL ACCESSORY NERVE

The external portion of this nerve arises by a series of roots from the cervical part of the spinal cord, and is really a motor spinal nerve directly connected with the anterior cornua. It is distributed to the sterno-mastoid and trapezius muscles; it is the chief supply of the former, but the latter is largely innervated by cervical and dorsal nerves.

In addition to cerebral and intracranial lesions, like those which may involve the vagus, the spinal accessory may be injured by caries of the cervical spine, by enlarged glands or abscesses in the neck, or by blows and strains. If the lesion is in the posterior triangle, the sterno-mastoid will of course be spared. Paralysis of the sterno-mastoid is shown by the want of prominence due to contraction of this muscle, and by deficient power of rotation of the head to the opposite side. In paralysis of the trapezius, the natural slope between the neck and the shoulder is converted into a deep hollow, which is exaggerated when the shoulder is raised, as it still can be by the action of the levator anguli scapulæ. The point of the shoulder lies lower than normal, and the posterior border of the scapula is inclined from below upwards and outwards. Elevation of the hand above the head is, however, difficult or impossible, because the trapezius does not fix the scapula for the use of the deltoid, nor does it assist in that rotation for which the serratus magnus is chiefly employed. If the whole muscle is paralysed, the approximation of the shoulder-blade to the spine is incomplete; but in spinal accessory lesions it is chiefly the upper part between the occiput and the acromion which is affected. With a persisting lesion atrophy and electrical changes naturally follow.

**Treatment.**—Here we must deal, when possible, with the causative lesion and with the muscular failure by electrical stimulation, and perhaps massage.

### TWELFTH OR HYPOGLOSSAL NERVE

This nerve, like the last, has a purely motor function, supplying the tongue and most of the muscles attached to the hyoid bone. Its lesions are very similar to those of the two nerves last considered. As the two nuclei are so close to the middle line, they are often affected together, producing bilateral results. Unilateral paralysis may result from disease above the nucleus, between it and the cortical centre in the ascending frontal convolution, and below the nucleus from meningitis, simple and syphilitic growths, caries of the cervical vertebræ, and tumours, cellulitis, or injuries beneath the jaw. If it is paralysed on one side, the back of the tongue on that side is slightly raised, from loss of the tonic contraction of the hyoglossus muscle. In the mouth it cannot be moved freely to the same side, but when protruded is pushed to the affected side by the contraction of the posterior fibres of the genio-hyoglossus, and by the elongating action of the transversus muscle on the healthy side. In bilateral paralysis the tongue lies motionless in the mouth. Articulation is impaired in proportion to the loss of movement, but very slightly in unilateral disease. Mastication

also suffers at the same time. If atrophy supervenes, the tongue shrinks in bulk and feels flabby, and the mucous membrane is thrown into wrinkles. The position of the lesion is suggested by the associated symptoms. If it is above the nucleus, there may be hemiplegic weakness on the same side as the lingual paralysis, but there will not be atrophy ; if below the nucleus, atrophy may ensue, and paralysis of the limbs, if any, will be on the opposite side. If the symptoms are bilateral, as in bulbar paralysis, the lesion is at or near the nuclei ; and it is the same if the other lower cranial nerves are involved.

**Treatment.**—This must follow the causal indications.

## LESIONS OF SPINAL NERVES

The spinal nerves may be injured or diseased at the roots, or in the plexus or nerve trunks beyond them. Isolated nerve paralyses in the limbs are more likely to be caused by lesions below the plexuses, while injuries of the plexuses, or nerve roots, are more liable to be followed by grouped paralyses.

Lesions of the nerve roots arise in connection with diseases, injuries and tumours of the spinal cord or spinal column, and the symptoms may be combined with those of the central affection, as, for instance, in meningitis, caries, and the degenerative changes of anterior poliomyelitis and tabes dorsalis. Lesions of the spinal nerve trunks are mostly injuries from pressure, wounds, fractures, and dislocation in the distal parts, from new growths, aneurysms, and abscesses in the proximal portions near the spinal column. Effusion into the nerve sheaths may also occur as the result of comparatively slight trauma, or excessive stretching, especially if this is often repeated. Inflammation of the nerve sheaths sometimes results from poisons gaining access from the blood stream, and these may be either of bacterial origin or by-products of faulty metabolism. Owing probably to their more exposed position and greater liability to stretching the nerve fibres of the brachial and sciatic plexuses are those chiefly affected by these causes.

Since the spinal nerves contain both motor and sensory fibres, the symptoms of their lesions are both loss of muscular power and anæsthesia, determined by the distribution of the nerve fibres to muscles and skin respectively. If the lesion is persistent, atrophy and altered electrical reactions (*see* p. 732) of the muscles will ensue, and perhaps trophic changes in the skin.

**Treatment.**—The results of these lesions which require treatment are paralysis and muscular atrophy on the one hand, and pain or other sensory symptoms on the other. In either case a removable cause, such as pressure by abscess or tumour, should, if possible, be dealt with ; or, if a toxic neuritis is the presumed cause, all possible sources of infection or intoxication in the body should be investigated and remedied as far as may be, *e.g.* pyorrhœa alveolaris, septic tonsils, chronic infections of the alimentary tract. Atophan and aspirin may be given internally, and dietetic reform prescribed in cases suspected of a gouty origin. Massage, electrical stimulation and exercises are of use in dealing with the paralysis. For pain the local application of heat, or of the liniments of belladonna, aconite, turpentine, etc., may be valuable. *Rest* is also essential for a quick recovery ; the arm should be carried in a sling, or, if the leg is affected, the patient should be in bed, and when necessary special splints should be employed to maintain paralysed muscles in the position of rest.

Some of the more important and frequent lesions of the spinal nerves are here shortly described.

### UPPER CERVICAL NERVES

These may be involved in diseases of the upper part of the spine, especially in tuberculous disease of the atlas or axis. The posterior rami are often affected by neuritis of traumatic or toxic origin. The common "crick" of the neck is a

familiar example of minor injury, and in persons predisposed to toxic neuritis cold appears to be a deciding factor in producing an attack (*e.g.* sitting in a draught). If the trouble is confined to the branches of the second cervical nerve the pain is felt in the occipital and sub-occipital regions and is frequently complained of as headache. Tenderness over the scalp and the points of emergence of the great occipital nerves are present during the paroxysms of pain and serve to distinguish these cases from those of intracranial disease.

**Treatment,** as in other cases of neuritis, should be directed towards the cause. Rest, warmth and aspirin are useful for relief of symptoms.

### PHRENIC NERVE

The fibres of the phrenic nerve are involved in disease of the cervical portion of the spinal cord, such as acute myelitis; occasionally the nerve is injured by wounds in the neck, and it may be pressed upon by tumours in the neck and thorax. It is not infrequently involved in the paralysis of diphtheria, alcoholism, and beri-beri, and in some other cases of multiple neuritis; and it may be affected in lead-poisoning. The characteristic symptom of a bilateral lesion is paralysis of the diaphragm. The breathing is effected solely by the action of the intercostal muscles, and accessory muscles of inspiration; the abdominal wall, instead of advancing during inspiration, is retracted, and it is driven out during expiration. Dyspnœa may be slight when the patient is tranquil, but movement increases the difficulty, and then the over-action of the thoracic walls becomes especially striking. In a less marked stage of paralysis the diaphragm seems to remain in a semi-inspiratory position, not contracting upon the contents of the abdomen, but it resists being drawn up into the chest, so that the abdominal wall is more stationary, during respiration. In a bad case a full respiration is impossible, coughing becomes difficult, husky, or noiseless, from inability to fill the chest, and the voice is almost lost. Two results are likely to follow. One is extensive collapse (*massive collapse,* Pasteur) of one or both lower lobes, more often the left, with dulness and absent or faint bronchial breathing. The other is impeded circulation in the lower lobes, so that mucus or œdema fluid accumulates which the patient is unable to expel. The danger thus arising is, of course, increased by any bronchitis. Diaphragmatic paralysis, though it sometimes gives the *coup de grâce* in alcoholic and diphtherial paralysis, is not necessarily fatal – it may last some days or weeks, and then gradually clear up.

### POSTERIOR THORACIC NERVE

This nerve is sometimes injured, as it lies in the posterior triangle of the neck, by loads carried on the shoulder. The lesion is thus common in porters, etc., and is nine times more frequent in men than in women; and may also be seen in anterior poliomyelitis. The paralysis of the serratus magnus which results is distinguished by the position of the scapula. The inferior angle approaches the spine from the unopposed action of the rhomboidei and the levator anguli scapulæ. The arm is with difficulty raised above the horizontal, since complete elevation is largely effected in health by the serratus magnus rotating the lower angle of the scapula forwards. When the arm is moved forwards in the horizontal position, the angle of the scapula projects from the chest, so that the fingers can be placed underneath it; and it approaches the spine at the same time (*alar scapula*). Cutaneous anæsthesia is, as a rule, absent, but the onset may be accompanied by neuralgic pains. The digitations of the muscle below the axilla may be obviously wasted or inactive as compared with those of the other side.

### CIRCUMFLEX NERVE

Dislocations of the shoulder, falls or blows on the shoulder, and the pressure of a crutch are the special causes of a lesion of this nerve. In lesions of the brachial

plexus, in lead paralysis, and in spinal lesions, it may also be involved. It is rarely affected by cold. The chief symptom is paralysis of the deltoid muscle, so that the arm cannot be raised to the horizontal position ; any attempt results in elevation of the shoulder by the trapezius and serratus, while the arm hangs vertically. In old cases atrophy and reaction of degeneration supervene. Cutaneous anæsthesia is often absent, and paralysis of the teres minor, also supplied by the circumflex, cannot generally be recognised. It must be remembered that ankylosis of the shoulder joint fixes the arm in the same position, and leads to atrophy of the muscle. Passive movement will distinguish between them.

## MUSCULO-SPIRAL NERVE

This nerve, from its exposed position as it winds round the humerus, is especially liable to injury from prolonged pressure, from the use of a crutch, or from hanging the arm over the back of a chair during sleep, or from sleeping with the whole weight of the body upon one arm. It may also be injured by fractures and dislocations, and rarely by violent action of the triceps. The muscles paralysed by a lesion high up are the extensors of the elbows and wrist, the long extensors of the finger and thumb, and the supinators ; but in the more common lesions a little above the elbow one or more muscles escape, especially the triceps and the supinator longus. With paralysis of the triceps there is inability to extend the forearm on the arm. This must be tested with the arm raised, so as to avoid the action of gravity in extending the forearm. The extensor paralysis of the wrist and fingers is shown by the " wrist-drop " or " dropped hand." If the forearm is extended in pronation, the hand hangs vertically, and cannot be raised, nor can the fingers or thumb be lifted from their pendent position. If the hand is raised and the first phalanges are supported, the middle and terminal phalanges can be extended by the action of the interossei and lumbricales. The supinator paralysis prevents any movement from the position of complete pronation, but if the forearm be flexed supination will be effected by the biceps. Flexion in semi-pronation is weakened by the absence of the supinator longus, and the characteristic prominence of this muscle in movements of flexion is absent. Flexion of the fingers is considerably weakened by the passive approximation of the ends of the muscles, and a prominence forms on the back of the wrist, which is due either to the thickening of the tendon sheaths or to the synovial sacs and carpal bones projecting backwards when unsupported by the extensor tendons. If there is cutaneous anæsthesia, it affects the outer side of the back of the hand, the back of the thumb, and the back of the first phalanges of the fore and middle fingers (*see* Fig. 70).

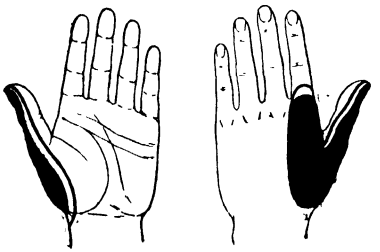

FIG. 76.—Loss of Sensation following a Lesion of the Radial and External Cutaneous Branches of the Musculo-spiral Nerve. The areas of complete analgesia are coloured black, and those of insensibility to light touch are enclosed by continuous lines. (*After Head.*)

## ULNAR NERVE

This is exposed to wounds and injuries in the arm, and near the wrist, and to injury by dislocations of the shoulder and elbow, and by fractures of the forearm. Symptoms of neuritis may develop long after an injury to the elbow as the result of cicatricial changes, with a resultant fusiform swelling or " neuroma " at the point of damage. Such a neuroma is most commonly found in the groove at the

back of the elbow where the nerve is most exposed to trauma, and may result from the accumulated effect of repeated minor injuries. Constant friction of the nerve upon its bony support in this region—as in a professional violinist or blacksmith—may produce a similar result. The movements affected are flexion of the wrist towards the ulnar side, flexion of the fingers, especially of the first phalanges, with extension of the second and third, adduction of the thumb and the lateral movements of the fingers by the interossei. In old cases the unopposed action of the extensor muscles leads to over-extension of the first phalanges, and flexion of the second and third, producing the claw-like hand (*main en griffe*). Anæsthesia is variable: its limits are the ulnar part of the hand corresponding to one and a half fingers in front and one (or two) and a half on the back (*see* Figs. 77, 79).

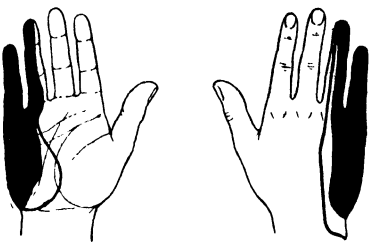

FIG. 77.—Loss of Sensation following a Lesion of the Ulnar Nerve. For explanation see Fig. 76. (*After Head.*)

In the cases of traumatic neuroma, particularly those in which the bony groove is unduly shallow and the nerve ill-protected, surgical intervention provides the surest remedy. The nerve is freed from adhesions and transferred to the front of the elbow. In slighter cases rest may suffice.

## MEDIAN NERVE

This is mostly affected by injuries, occasionally by neuritis. When it is paralysed the forearm cannot be pronated more than half-way, flexion of the wrist takes place towards the ulnar side, the thumb cannot be flexed or abducted, the second phalanges of the fingers cannot be flexed on the first, nor the third phalanges on the second except in the case of the third and fourth fingers, in which this is effected by the ulnar half of the flexor profundus. Anæsthesia, if it occurs, affects the radial half of the palm of the hand, the anterior aspect of the thumb, forefinger, middle finger, and radial side of the ring finger, and the dorsal surfaces of the thumb and the same three fingers (or fore and middle only in some persons) beyond the first joint (*see* Figs. 78, 79).

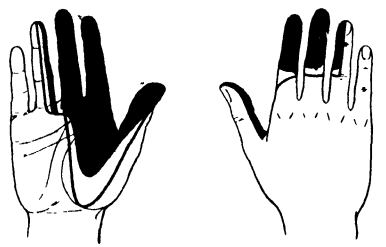

FIG. 78.—Loss of Sensation following a Lesion of the Median Nerve. For explanation see Fig. 76. (*After Head.*)

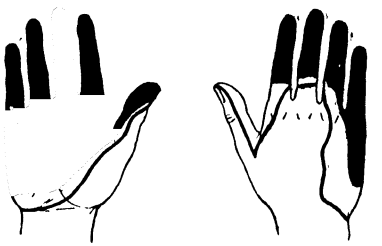

FIG. 79.—Loss of Sensation following a Lesion of the Ulnar and Median Nerves. For explanation see Fig. 76. (*After Head.*)

## BRACHIAL PLEXUS

The nerves forming this plexus may be more or less completely involved as a result of injury, growths, or severe inflammation. This produces paralysis of all

the muscles of the hand, arm, and shoulder, as well as anæsthesia of the hand, forearm, and outer side of the upper arm, the inner side being spared because it is in part innervated by intercostal nerves. It is most commonly seen in one of the two forms described below, but there are minor variations in the muscles affected, due probably to differences in the way in which the nerve roots combine to form the plexus.

*Upper arm type*, or *Erb's paralysis*.—The following muscles are paralysed: deltoid, biceps, brachialis anticus, supinator longus and perhaps the supinator brevis, supra-spinatus, and infra-spinatus, and radial extensors of the wrist. Elevation of the arm and flexion and supination of the forearm are impossible. Sensory symptoms in the area of the circumflex and musculo-cutaneous nerves are variable.

*Lower arm type*, or *Klumpke's paralysis*.—This appears to be due to a lesion of the eighth cervical and first dorsal nerves. The small muscles of the hands and the flexors of the fingers are chiefly concerned, and there is anæsthesia of the inner side of the arm up to the elbow. In addition there are *oculo-pupillary* symptoms, viz. contraction of the pupil on the same side and diminution of the palpebral fissure, due to involvement of the cervical sympathetic fibres as they pass out from the cord in the first dorsal root.

*Brachial Neuritis*.—This may result from the direct spread of a local suppurative process, but is more commonly due to the combined effects of minor injury and blood-borne toxins—bacterial or chemical. Exposure to cold also appears to play a part in its causation. The complaint is chiefly of pain which is of an aching or burning nature, and is more or less constant with occasional severe paroxysms. The spinal roots of the plexus are frequently involved so that pain is experienced behind the scapula from affection of the posterior rami as well as down the length of the arm and into the fingers. Although subjective numbness and tingling may be present in the distribution of certain nerve roots it is very rare to find any definite band of anæsthesia. There are often points of extreme tenderness to pressure at the points of emergence of the posterior rami between the spine and the scapula, and the cervical roots are tender when pressed upon in the supraclavicular fossa. Localised muscular wasting is not common, but the whole musculature of the limb becomes wasted from disuse if the illness is prolonged.

The course is variable. As a rule the trouble persists for several weeks, and is apt to recur as the result of stretching or injury.

The treatment consists in dealing with the cause if it can be found. Septic foci in teeth or tonsils if they are undoubtedly present should be eradicated. In other cases dietetic reform may be indicated and atophan or aspirin prescribed. In the acute stages rest is essential. The arm should be carried in a sling by day and supported on a pillow at night: gentle passive movements should be performed daily to prevent adhesions at the joints. Heat locally applied is useful in the form of hot-water bottles or the rays from a high-power electric lamp in a metallic reflector. Diathermy is valuable in chronic cases. Of drugs, aspirin is most useful. With the gradual cessation of pain massage should be prescribed to restore the weakened muscles, and active movement can gradually be resumed.

*Cervical ribs*.—The occasional development of the anterior transverse process of the seventh cervical vertebra to form a rib is liable to produce symptoms referable to pressure upon the eighth cervical or first dorsal roots. These may appear at almost any time of life, but are most commonly seen either at puberty or in middle age. In a large number of cases their onset is determined by occupational factors; thus they may be first noticed by the patient on taking to the violin, playing golf, or riding a horse, or in married women as the result of carrying their babies. The patient usually complains of weakness of the hand, together with pain, tingling and numbness chiefly along the ulnar border of the forearm. The weakness is accompanied by wasting of the small muscles of the

hand, which may conform to either of two types. In the first and most characteristic the wasting is confined to the two chief muscles of the thenar eminence: the abductor and the opponens pollicis. The radial border of the adductor pollicis is thus thrown into prominence, and on palpation of the outer part of the thenar mass the metacarpal bone is easily felt. The movements affected are abduction of the thumb (which takes place in a plane at right angles to the palm) and opposition. In the second type all the muscles of the hand are equally affected, so that the condition has to be differentiated from progressive muscular atrophy, syringomyelia or an ulnar nerve palsy. There may be some diminution of sensibility along the ulnar border of the forearm, but this is inconstant. Vascular symptoms also occur in 35 per cent. of the cases. These may take the form of diminution or obliteration of the radial pulse, due to pressure upon the subclavian artery, or may be due to involvement of sympathetic fibres and appear as vasomotor changes, such as flushing, heat and swelling of the hand. In a

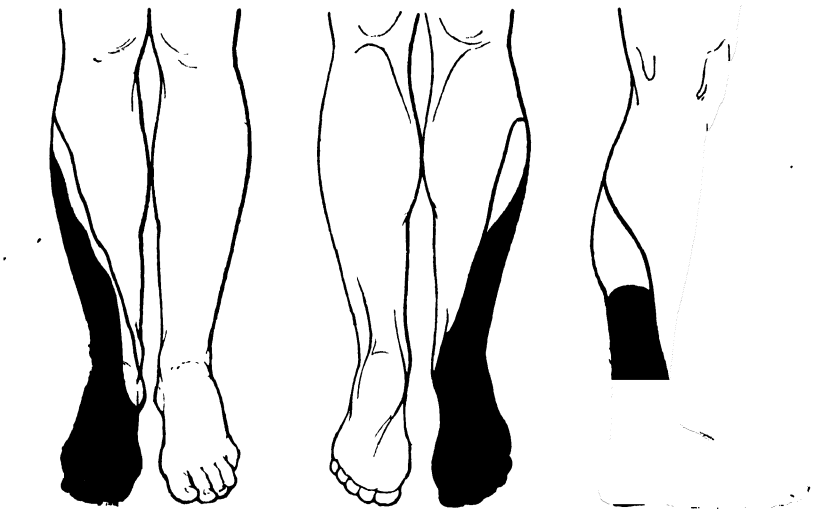

FIG. 80. Loss of Sensation following a Lesion of the Sciatic Nerve. The areas of complete analgesia are coloured black, and those of insensibility to light touch are enclosed by continuous lines. (*After Head and Sherren.*)

number of cases thrombosis of the radial artery has been recorded, spreading up the limb as far as the subclavian. In 5 per cent. of the cases vascular changes have been recorded without symptoms of paralysis or anæsthesia. Oculo-pupillary symptoms may also be produced by pressure upon the fibres of the cervical sympathetic.

If large the abnormal rib is palpable, and is usually revealed by the X-ray, which frequently shows the condition to be bilateral, even though the symptoms be confined to one side. It is important to recognise that the size of the rib, as revealed by the X-ray, bears no relation to the severity of the symptoms, which may be due to pressure exerted by a fibrous band passing from an enlarged anterior tubercle on the seventh cervical vertebra to be attached to the first rib. Occasionally the subclavian artery is lifted so high in the neck as to produce a pulsating swelling above the clavicle which may be mistaken for an aneurysm.

Radiographic experience has shown that cervical ribs are not uncommon in persons without symptoms referable to their presence, and care must therefore be taken to exclude other possibilities in the diagnosis.

The **Treatment** is removal of the rib, or rest and change of occupation.

### SCIATIC NERVE

The most common paralysis in the lower extremity is that due to disease of the sciatic nerve, wholly or in part. It may be from tumours or diseased bone in the pelvis, from dislocations of the hip, from wounds, tumours, or neuroma in the thigh. Neuritis is relatively common, and to this many, if not all, cases of *sciatica* are to be attributed. In a lesion of the sciatic trunk above the upper third of the thigh, the flexors of the leg upon the thigh are involved, in addition to those affected through the two popliteal branches.

### EXTERNAL POPLITEAL (PERONEAL) NERVE

This nerve occupies an exposed position near the knee, like that of the ulnar at the elbow. Lesions cause paralysis of the tibialis anticus, the peronei, the long extensors of the toes, and the short extensor on the dorsum of the foot. The foot hangs down when raised from the ground (foot-drop), and lies extended

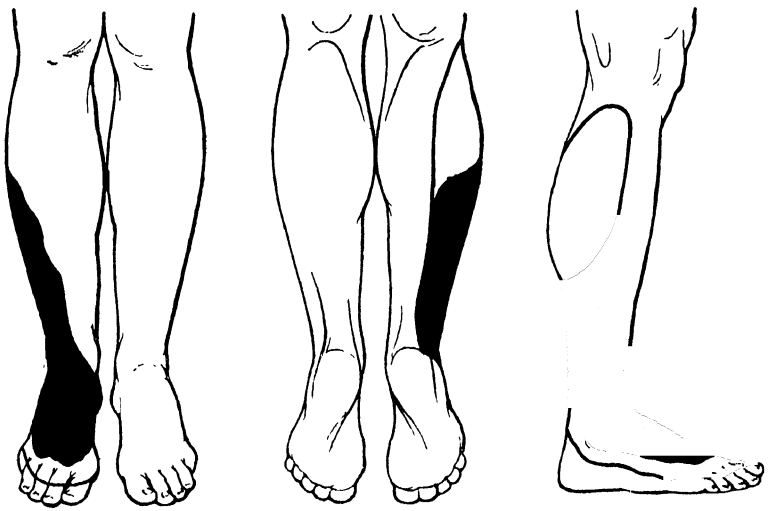

FIG. 81.—Loss of Sensation following a Lesion of the External Popliteal Nerve. For explanation see Fig. 70. (*After Head.*)

when the patient is in bed; dorsal flexion of the foot and of the toes and abduction of the foot and elevation of its outer border are deficient or impossible. Wasting of the anterior tibial muscles and of the extensor brevis can be recognised by comparison with the other leg; in old cases permanent extension of the foot (talipes) is produced mainly by the action of gravity, especially when the patient lies in bed. Anæsthesia affects the outer half of the leg and the dorsum of the foot (*see* Fig. 81).

### INTERNAL POPLITEAL NERVE

Extension (plantar flexion) of the foot and flexion of the toes cannot be effected. The patient cannot raise himself on his toes, nor can he adduct the foot. In old cases talipes calcaneus may develop, and a kind of claw-foot from over-extension of the proximal phalanges and flexion of the second and third. Anæsthesia corresponds to the sole of the foot and its outer border up to the external malleolus (*see* Fig. 82).

## SCIATIC NEURITIS

(*Sciatica*)

**Ætiology.**—The sciatic nerve is more frequently affected by subacute inflammatory changes in its sheath than any other. Although in a number of cases no definite origin can be traced, the causes are probably the same as those of similar affections elsewhere (*e.g.* Brachial Neuritis), and are (1) trauma, either from a single violent stretching movement, or from repeated friction against the bony parts, as in stooping, or from prolonged pressure upon the hard edge of a chair; (2) local infection, as from a cellulitis of the pelvis; (3) poisons circulating in the blood-stream, which may be either bacterial from some septic focus, or chemical as the result of faulty metabolism.

The disease is much more common in men than in women, and is more frequent, on the whole, among those whose occupations involve much stooping (*e.g.*

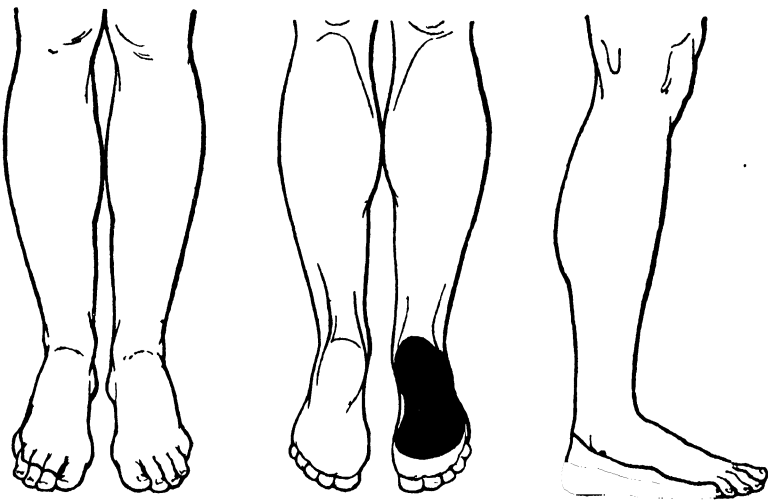

FIG. 82.—Loss of Sensation following a Lesion of the Posterior Tibial Nerve. For explanation see Fig 70. (*After Head.*)

gardeners, charwomen). It is almost confined to the latter half of life, seldom occurring in younger persons except as the direct result of trauma.

**Symptoms.**—The chief symptom is pain, which is felt in the nerve trunk or its branches; it comes on either gradually or suddenly, and is aggravated by movement or the attempt to walk, or by anything which causes the nerve to be stretched or pressed upon. To avoid this, the patient holds the leg fixed at the knee when walking, and when lying down the most comfortable position is one of flexion. When standing he throws his weight upon the unaffected limb. The pain is most often in the back of the thigh, but may extend down the back of the calf, along the outer side of the leg, and to the sole of the foot; and is often most intense at certain spots—namely, near the posterior iliac spine, at the sciatic notch, about the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the dorsum of the foot. The nerve, too, is tender to pressure, especially at the sciatic notch, along the back of the thigh, and in the external popliteal branch behind the head of the fibula.

The pain is burning or gnawing, more or less continuous, but intensified by movement or manipulation. In severe cases, other disturbances of nerve function occur. These are tingling, numbness, and anæsthesia, in connection with

sensory fibres, and atrophy of muscles, muscular weakness, and sometimes fibrillary tremors, from implication of motor fibres. Other conditions accompanying sciatic neuritis are abolition of the gluteal fold, which may occur without obvious wasting of the gluteal muscles; Lasègue's sign, that is, pain on flexing the hip joint with the leg extended at the knee; and absence of the ankle jerk. Lasègue's sign may be accompanied by momentary dilatation of the pupil, raised blood pressure, and quickened pulse. The electrical reactions are not markedly altered except in severe cases, when reaction of degeneration may occur.

**Diagnosis.** This has to be made from other diseases involving the sciatic nerve, by a process of exclusion. Disease of the hip joint, sacro-iliac disease, new growth or chronic osteomyelitis in the upper end of the femur, and malignant disease within the pelvis compressing the sciatic nerve must all be considered. Rectal examination should be performed in every case, and an X-ray photograph may be necessary where the diagnosis is doubtful.

A tumour of the cauda equina may at first produce symptoms resembling those of a sciatic lesion, but in the former the pain sooner or later becomes bi-lateral and the sphincter control is impaired.

**Prognosis.** This is, on the whole, favourable, but the duration is very variable. Slight cases may recover quickly; severer cases last months or years; and after subsidence of the pain the muscular wasting, fibrillary contractions, and a tendency to cramps may persist for some time. There is a considerable tendency to recurrence.

**Treatment.** Complete rest is essential from the outset. The patient should be put to bed, with the limb supported upon pillows in the position that most eases pain. To ensure adequate rest it may be necessary at first to splint the limb either by means of a modified long Liston's splint or with the aid of a plaster of Paris bandage around the hip joint. In addition, to relieve the pain all the ordinary forms of counter-irritation can be tried, and of these heat often proves the most beneficial, being most conveniently applied in the form of hot water bottles along the course of the nerves. A high candle-power electric lamp contained in a metal reflector also provides a simple and effective method of heat irradiation. Of drugs for relief of pain the most useful are aspirin, phenacetin and veramon. An occasional dose of opium is justifiable for the relief of severe paroxysms, and it is then preferably given as the tincture. Massage and manipulation should be avoided in the acute stages, but may be usefully employed when the pain has subsided. The bowels should be kept well open to prevent pressure upon the nerve from fæcal accumulations.

An attempt should be made to discover and eliminate any possible causes of the condition in the shape of focal septic infections. In cases which are suspected to be of gouty origin the diet should be altered, and atophan prescribed (*see* p. 667).

### MERALGIA PARÆSTHETICA

This is an affection, probably inflammatory, of the external cutaneous nerve of the lumbar plexus. The symptoms are *pain*, sometimes brought on by walking, at others when the patient is lying still or sitting, and abnormal sensations, such as numbness, pins and needles, cold feeling, burning sensation of tightness, felt on the front and outer part of the thigh within the distribution of the above nerve. Over the same area there is some modification of sensibility, either hyperæsthesia, or more often anæsthesia, or changes in the appreciation of pain, heat or cold. In most cases there is some tenderness on pressure below the anterior superior iliac spine, where the nerve comes through the fascia lata. It is more frequent in men than in women, and has been attributed in different cases to injury and to toxic and infective agents; in women, a badly fitting corset may be the cause. Rest, warm baths, and counter-irritants should be tried for its treatment.

## LOCALISED MUSCULAR SPASM

A number of local disorders are recognised in which clonic or tonic spasm of muscles supplied by a particular nerve is the main feature. They are sometimes functional, at others dependent on organic disease. Some of them are described in other parts of the volume, namely, *conjugate deviation* of the eyes and head (*see* Hemiplegia), laryngeal spasm, and spasmodic wryneck. Other disorders of muscular action, less obviously of nervous origin, are described in the section on Diseases of the Muscles.

### SPASM OF OCULAR MUSCLES

This occurs in association with various diseases of the eye, in the conjugate deviation of cerebral disease, in hysteria, and other conditions. The clonic spasm termed *nystagmus* consists of oscillating movements of the eyeball, generally in a lateral direction, sometimes vertically. Sometimes they are constant; more often they are brought out by movements of the eye to the extreme limit in one or other direction. They are often jerky; that is, there is a quick movement in one direction, and a slower return in the other (*see* p. 716). Nystagmus may result from lesions of the semi-circular canals of the vestibular nerve and its central connections in the pons and mid-brain, or of the cerebellum (*see* p. 716). Among diseases of the central nervous system it occurs with greatest constancy in disseminated sclerosis and Friedreich's ataxy, but also frequently in tumours of the cerebellum. It results also from ocular defects, errors of refraction, especially extreme myopia, hypermetropia with astigmatism, corneal opacities, choroidal atrophy, strabismus, albinism and other conditions impairing the retinal images, or the transmission of retinal impressions to the brain. It sometimes runs in families; that is, it is hereditary. It may be associated with other muscular spasms, such as spasms of the eyelids, or of the head (head-nodding, or *spasmus nutans*. *See* Rickets). It is well known to occur in miners, especially in those who work in the recumbent position.

### SPASM OF THE JAW

Trismus, or spasm of the muscle closing the jaw, is one of the early symptoms in tetanus. The jaw is fixed by tonic contraction of the masseter or temporal muscles, so that the teeth cannot be separated more than a very little. A similar spasm may be due to irritation of the teeth, or to stomatitis, or, on the other hand, to central disease, such as disease of the pons in the neighbourhood of the fifth nerve nucleus. It must be distinguished from tumours or rheumatic arthritis fixing the jaw joint. *Clonic* spasm of the jaw occurs in rigor, in convulsions, and in hysteria, rarely as an isolated phenomenon. Jaw clonus may sometimes be obtained in upper neuron lesions (*see* p 735).

### FACIAL SPASM

Irregular contractions of the facial muscles take place in chorea, and a tonic contraction is a late stage of facial paralysis. Boys and girls often acquire a habit of twitching certain muscles of the face, neck, or other part of the body, and this habit may last into adult life, or it may begin in adult life (*see* Tic).

More serious cases of facial spasm occur in people over twenty years of age, and mostly between thirty and sixty. In some of these there is actual irritation of the facial nerve by tumours in the pons, or of the facial cortical centre on the opposite side of the brain; in most cases the causation is obscure. The spasm chiefly affects the orbicularis palpebrarum (*blepharospasm*) and the zygomatici, so that the eye is half closed, and the angle of the mouth is drawn up. Other facial muscles, including the platysma myoides, are also contracted, but the orbicularis oris and frontalis muscle, as a rule, escape. The contractions are

momentary, and frequently repeated; or the spasm is of longer duration, and recurs at longer intervals; but it causes no pain. The spasm is at first entirely on one side, and only in severe or prolonged cases affects the other side. The electrical reactions are usually normal.

The **Prognosis** in a well-established case is unsatisfactory; the disease will last months, or years, and even to the end of life.

The **Treatment** consists in the removal of causes of irritation, if they can be recognised. Sedatives such as bromide may be of value. The most effective form of treatment, however, is injection of alcohol into the branches of the facial nerve as they diverge from the stylo-mastoid foramen.

Cases occur in which the eyelids are moved involuntarily whenever the jaw is moved by the action of the will (*jaw-winking*). This is often associated with congenital or acquired ptosis, and, it may be, with nystagmus.

### HICCOUGH

This is a repeated convulsive contraction of the diaphragm induced reflexly, as a rule, by irritation in the abdominal cavity, as by a full meal, pepper or spices taken into the stomach, or by peritonitis. It is also the result sometimes of excessive laughing. Persistent hiccough as a rule is due to peritonitis or other abdominal lesions, or is a part of hysteria, but may be a prominent symptom in certain cases of organic nervous disease in which the spinal cord is affected in the cervical region (*e.g.*, encephalitis lethargica). Hiccough persisting for several days may also occur as an isolated symptom of what appears to be an extremely mild epidemic disease, possibly related to encephalitic lethargica. Hiccough may be stopped by holding the breath, by taking spiritus ætheris nitrosi, or other diffusible stimulant; in protracted cases inhalations of chloroform, or nitrite of amyl, or injections of morphia may be necessary. Chloretone in doses of 5 to 10 grains may also be effective. Sometimes firm traction on the tongue is successful.

# DISEASES OF THE SPINAL CORD

For the proper understanding of this group of diseases an acquaintance with the principles of physiology and anatomy summarised on pp. 702 *et seq.* is essential.

**Primary Disease of the Neurons.**—The several groups of neurons in the spinal cord may be separately affected by degeneration or disease. Such degeneration is the result of toxins, or is due to congenital want of vitality, or remains entirely unaccounted for. The lesions have been known as *tract diseases* or *system diseases*, and the following are instances: tabes dorsalis, in which the posterior columns are diseased; primary lateral sclerosis—pyramidal tracts; subacute combined degeneration—posterior columns and pyramidal tracts; Friedreich's disease—posterior columns, pyramidal tracts and spino-cerebellar tracts; progressive muscular atrophy—anterior horn cells; amyotrophic lateral sclerosis—anterior horn cells and pyramidal tracts.

**Transverse Lesions.**—The spinal cord, having an elongated form, is naturally liable to lesions affecting its whole thickness, such as may occur from trauma, localised external pressure, the indiscriminate growth of tumour, the diffuse spread of inflammation, or the anæmia of vascular obstruction. In such cases grey and white matters are equally affected. The results of such a transverse lesion show themselves mainly as an interruption of the *conducting* power of the cord; but if the lesion is at all extensive vertically, its effects upon the *nerve-cells* must also be considered—that is, both upper and lower neurons are affected. The results also vary, according as the lesion is *bilateral* or *unilateral*,

in consequence of differences between the motor and sensory fibres in the process of decussation.

*Results of a Unilateral Transverse Lesion as Affecting Conduction (Brown-Séquard Syndrome)*

| On the same side as the lesion and below it. | On the opposite side below. |
|---|---|
| Paralysis of muscles. | Loss of senses of touch, pain, heat, and cold. |
| Hyperalgesia. | Muscular power and nutrition, the muscular sense, reflex action, and temperature are normal. |
| Loss of appreciation of posture of limbs, of passive movements, of separation of points applied to the skin, of size, shape, weight, and consistence of objects, and of vibrations of tuning-fork. | |
| Tendon jerks at first lessened, then increased. Plantar reflex extensor, abdominal reflexes absent (if lesion high enough to affect them). | |
| Vasomotor paralysis and elevation of temperature. | |
| The sense of touch is retained, as well as sense of pain, heat and cold, the nutrition of muscles and their electrical reactions. | |

*Results of a Bilateral Transverse Lesion as Affecting Conduction.*

Anæsthesia below the level of the lesion.

Paralysis of all muscles below the level of the lesion ; there is no wasting ; the electrical reactions are not altered. Abdominal reflexes absent ; tendon jerks increased ; plantar responses extensor.

Loss of sphincter control.

These symptoms vary with certain conditions as follows : *if the lesion is of acute onset* there is at first, during the period of spinal shock, complete flaccidity of the paralysed muscles with abolition of tendon jerks and plantar responses. After a few weeks, *if the lesion is incomplete,* the paralysed limbs become spastic in extension with increased tendon jerks and extensor plantars. *If the lesion is complete* the paralysed muscles remain flaccid, but the lower limbs are subject to involuntary flexor spasms which may result in contracture ; the tendon jerks return : the plantar responses are extensor.

To these symptoms in each case may be added those caused by damage to the nerve cells of the particular segment affected, together with the motor and sensory fibres directly connected with it by the anterior and posterior nerve roots. These will naturally vary with the *level of the lesion.*

*A bilateral lesion of the upper cervical region* will produce spastic paralysis of all four limbs with sensory loss below the level affected. The local destruction of anterior horn cells will add to this wasting and weakness of the sternomastoids and trapezii. Only patients with partial lesions at this level survive. A complete lesion causes paralysis of the diaphragm.

*A lesion of the cervical enlargement* will produce wasting and weakness of the muscles supplied from the segment affected, *e.g.,* in the case of the fifth cervical segment the deltoids, spinati, and biceps, with loss of the biceps tendon jerk (*see* Diagram, p. 709). Below the level of the lesion spastic paralysis and sensory loss.

In addition, all lesions above and including the first thoracic segment will produce paralysis of the cervical sympathetic fibres either in their downward course from the medulla, or at the point of their emergence in the first thoracic root (*see* Fig. 73A, p. 721).

*Lesions of the Thoracic Region* produce little in the way of segmental signs. Nevertheless palpation may sometimes determine the presence of wasting and weakness of the intercostal muscles supplied by the affected segment. When the lesion is below the level of the eighth thoracic segment the upper halves of the recti abdominis are spared. Therefore the upper abdominal reflexes are preserved. And when the patient attempts to raise his head the umbilicus is pulled upwards by the unopposed action of the upper halves of the recti. As before, there will be sensory loss and spastic paralysis below the level of the lesion.

*Lesions of the Lumbar Enlargement* may be clinically divided according to their level as follows :

Third and fourth lumbar segments : flexion of hip preserved ; apart from this lower limbs paralysed ; atrophy of quadriceps and adductors ; absent knee jerks ; brisk ankle jerks and extensor plantar responses.

First and second sacral segments : flexion of hip, adduction of thigh, extension of knee and dorsiflexion of foot preserved ; atrophy of calf muscles and intrinsic muscles of foot. Weakness of flexion of knee and extension, abduction and external rotation of hip. Knee jerks present ; ankle jerks and plantar responses absent.

Third and fourth sacral segments : paralysis of large bowel and bladder ; anal reflex lost.

Reference to the segmental sensory charts on p. 705 will indicate the extent of the sensory loss in each case.

*Lesions of the Cauda Equina*, when complete, may involve all the nerve roots, both motor and sensory, from as high as the first lumbar roots downwards, with resultant paralysis and atrophy of all the muscles supplied, sensory loss, and abolition of all reflexes. The differential diagnosis between a lesion of the lumbar enlargement and a lesion of the cauda equina may be extremely difficult. In the case of an acute lesion during the period of spinal shock there may be no means of distinction. Later the universal muscular wasting and permanent abolition of reflexes serves to distinguish the cauda equina lesion.

### *The Relation of Spinal Segments to the Spinous Processes of the Vertebræ*

The spinal cord ends below at the level of the lower border of the first lumbar vertebra. This corresponds to a line drawn across the back immediately above the first lumbar spine. The sacral segments lie between this line below and the upper level of the twelfth thoracic spine. The lumbar segments lie between this point and a horizontal line immediately above the tenth thoracic spine.

As regards the relation of higher segments to spinous processes the distance of any segment above the spine of corresponding number is roughly equivalent for the lower six thoracic segments to a depth of four vertebræ, for the upper six thoracic three, the lower cervical two, and the upper cervical one.

## COMPRESSION OF THE SPINAL CORD

The spinal cord, on account of its soft consistency, is subject to compression as the result of disease or injury of neighbouring parts. The pathological changes which take place under such circumstances are the same whatever the actual cause. The symptoms, which are those of a transverse lesion, naturally vary with the level of the lesion. Other variations may be due to the rapidity with which the compressing force acts, and to the actual nature of the compressing cause.

## PLATE XL.

**Sections of the Spinal Cord stained by the Marchi Method to show Degeneration of Fibre Tracts above and below a complete Transverse Lesion of the Second and Third Thoracic Segments, at which level the cord was compressed by secondary deposits in the vertebræ from a carcinoma of the breast.** ***The degenerated fibres are stained black.***

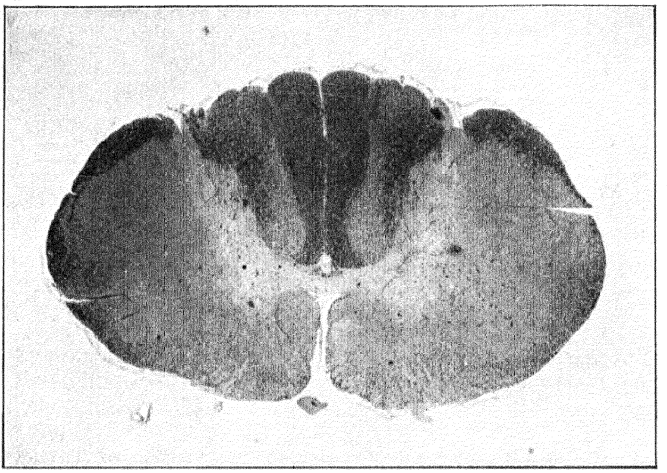

FIG. 1.—Seventh Cervical Segment: above the Lesion. The fibres of the ascending tracts are degenerated. The pale areas in the posterior columns represent the spaces occupied by association fibres. The spino-thalamic tracts are not clearly defined.

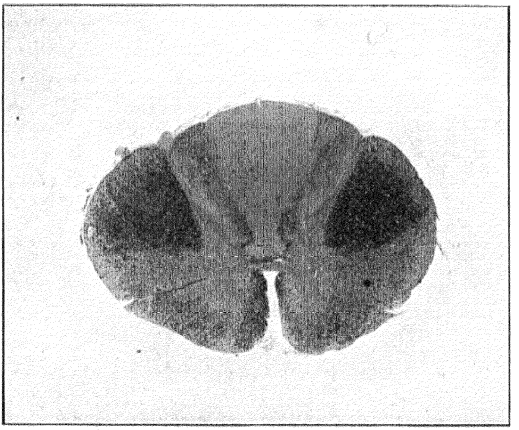

FIG. 2.—Fifth Thoracic Segment; below the Lesion. The fibres of the descending tracts are degenerated. The stippled areas in the posterior columns represent the degeneration of association fibres descending from above the level of the lesion.

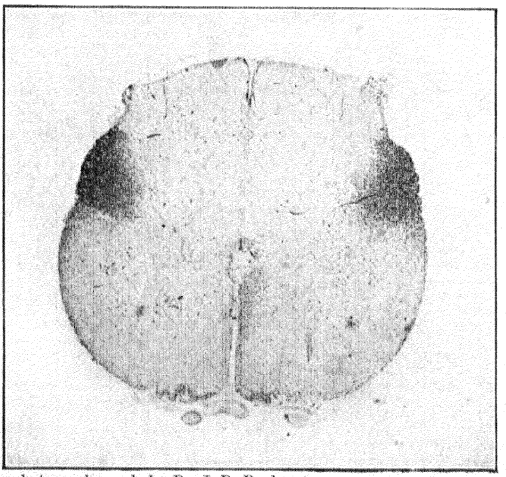

FIG. 3.—Lumbo-sacral Enlargement. The fibres of the crossed pyramidal tracts are degenerated. Those of the other descending tracts have at this level come to an end.

(From photographs made by Dr. J. R. Perdrau.)

[*To face p.* 767.

**Ætiology.** The causes of spinal compression may be divided into: (*a*) disease of the vertebral column, (*b*) disease of the extra-dural space and meninges.

(*a*) *Disease of the Vertebral Column.* The spinal cord is frequently compressed suddenly as the result of *injury* with fracture dislocation. Of causes acting more slowly the commonest are *tuberculous caries* of the vertebræ or *new growths.*

(*b*) *Disease of the Extra-dural Space and Meninges.* *Injury* may occasionally result in hæmorrhage into the meninges, with subsequent cicatrisation and compression. Commoner causes of cicatricial constriction are a localised *syphilitic meningitis*; or a preceding *suppurative meningitis.*

*New growths* of various kinds may arise in the extra-dural space, in the meninges, or upon the nerve roots.

**Morbid Anatomy.** Sudden compression of the spinal cord, such as occurs from a fracture dislocation or the sudden collapse of a vertebra infiltrated by growth, results in acute necrosis of the part compressed from cutting off of the blood supply. With this are associated hæmorrhages from ruptured veins and capillaries. The dead tissues rapidly undergo fatty degeneration with resultant softening, and this softening may spread in an upward or downward direction some distance beyond the level of the lesion.

Following the transverse lesion there is secondary degeneration of the nerve fibres which are cut off from their cells of origin (*see* Plate XLI.)

When the compression is more gradual the effect at first appears to be mechanical; the function of the compressed fibres is impaired but rapidly returns if the pressure is removed. Later there is circulatory stagnation and œdema from compression of the veins, and finally obstruction of the arteries leads to necrosis and softening.

Compression of the spinal cord, whatever its cause, is likely also to produce obstruction of the sub-arachnoid space. This results in stagnation of the cerebro-spinal fluid below the blockage, with accumulation of protein, and to this is sometimes added a yellow pigment probably derived from extravasated blood.

*Tuberculous caries* of the vertebræ may cause compression of the cord in two different ways. The commoner mode is that in which the infective process makes its way through the posterior wall of the vertebra and forms a caseous abscess separated from the meninges by the extra-dural tissues. The formation of such an abscess may occupy only a few weeks, and is quite commonly preceded by a history of injury to the diseased spine.

In the other form of tuberculous compression the infective process spreads directly to the meninges, causing a chronic localised meningitis, which in time constricts the cord. This process is very much slower than the other.

*New growths in the vertebræ* are usually of secondary origin, the commonest sources in the female being carcinoma of breast or uterus, and in the male the gastro-intestinal tract and prostate. The growth arises in the cancellous portion of the vertebra, which it erodes, until it finally bursts through the compact shell. The softening of the bone eventually leads to collapse of the walls of the vertebral canal with pressure upon the spinal cord. Often the vertebral column is much more extensively involved than may appear from the symptoms observed during life. Sarcoma may be primary or secondary in the vertebral column or the structures surrounding it, and results in changes similar to the effect of carcinoma. Myelomas in the vertebræ may also give rise to compression of the cord.

The space between the vertebræ and the dura mater may occasionally be the site of tumours, usually of a sarcomatous nature, and may be a resting place for hydatid cysts. It is also sometimes invaded by Hodgkin's disease or lympho-sarcoma originating in the mediastinum.

*Chronic syphilitic meningitis* may result in very great thickening of the meninges in the midst of which the spinal cord is compressed. This is frequently associated with syphilitic disease of the arteries, which accelerates the process of necrosis.

Of *new growths of the meninges* the majority are small encapsuled tumours of benign type arising from the inner surface of the dura, the arachnoid, or the nerve roots. According to their microscopic appearance they are classified as endothelioma, psammoma, fibroma, fibro-myxoma and neurofibroma. This is the group of tumours which lend themselves readily to surgical removal.

The other type of growth met with in the meninges is of a diffuse sarcomatous nature, with a tendency to spread upwards and downwards, of malignant type and irremovable.

*Meningitis serosa circumscripta* is a title given to a condition in which the meninges are found matted together with inclusion of cyst-like spaces containing clear fluid. This may follow injury (often after a long latent interval) or may occur as a late result of suppurative meningitis, or it may be found without apparent cause.

**Symptoms.** The symptoms of spinal compression are those already described of a transverse lesion of the cord, varying with the level affected (*see* p. 765). To these must be added the symptoms due to compression of nerve roots when these are also involved.

When the compression occurs suddenly as from fracture dislocation, or the collapse of a diseased vertebra, there is in addition to the loss of sensibility, power, and sphincter control, an initial period of spinal shock during which all reflexes are abolished below the level of the lesion. With this there is often fever, and the clinical picture at first may resemble that of an acute myelitis.

When compression occurs gradually the first symptoms are often due to pressure upon posterior nerve roots. These take the form of pain, which is usually of a severe aching character, and is referred to the segmental distribution of the roots affected. As a rule it begins by being unilateral, but may later spread to the other side. Hyperæsthesia of the skin may sometimes be found in the area supplied by the irritated roots. Pressure upon anterior roots may also give rise to muscular wasting and weakness, which may be an important localising sign in compression of the cervical or lumbar enlargements.

It must be remembered, however, that in the thoracic region the vertical length of the segment is such that the cord may quite well be compressed without involvement of nerve roots, and that in any case the compressing cause may act upon the anterior surface of the cord. In these cases the characteristic symptom of "root pain" is lacking from the history.

Of the structures within the cord the pyramidal fibres are usually the first to suffer, with resultant weakness and spasticity, increased tendon jerks, diminished or lost abdominal reflexes, and extensor plantar responses. Sensory disturbance is not as a rule evident for some time after weakness has been noticed. Careful examination is at first required for its detection. A level will be found below which pinprick is less painful, cotton wool touches are less constantly appreciated, and the difference between hot and cold tubes is less certainly distinguished.

Examination of the senses of positive and passive movement will reveal diminished accuracy, and the vibrating tuning-fork may be less keenly appreciated below a certain level.

As the disease progresses the sensory loss becomes more and more pronounced, until as a rule a definite border line may be distinguished between the healthy and the affected parts. This line, although it is not usually sharply defined, indicates fairly accurately the level of the lesion. It will occasionally be found that with a lesion in the upper region of the cord some of the fibres from the lowest segmental areas—and especially those conveying impression of deep sensibility—may escape, so that sensation to deep pressure is preserved over the sacrally supplied areas.

As the paralysis of the lower limbs increases control of the sphincters is impaired, with at first precipitate micturition and dyschezia.

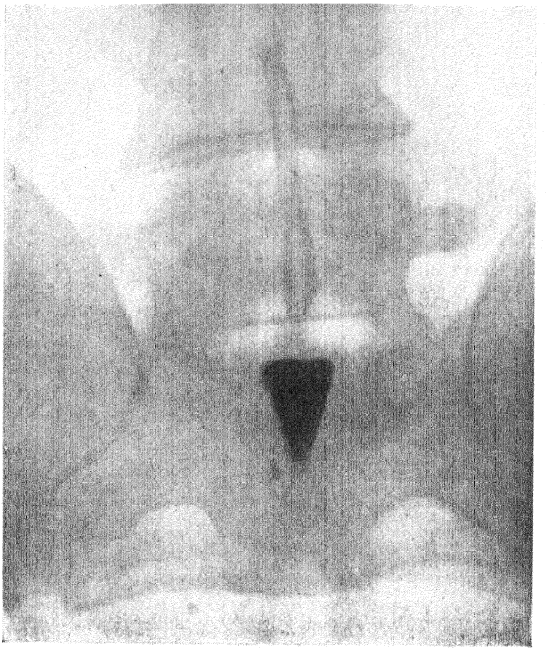

FIG. 1.

Radiogram half an hour after injection of lipiodol into cisterna magna. Patient in vertical position. The lipiodol has collected at the bottom of the thecal sac, showing that there is no obstruction in the subarachnoid space.

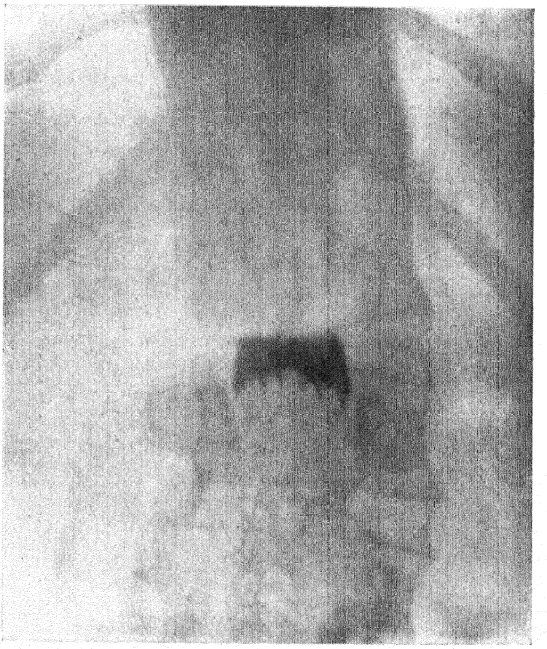

FIG 2.

Radiogram taken after Lipiodol Injection; conditions as in Fig. 1. The lipiodol is held up at the upper border of the second lumbar vertebra. Subsequent operation revealed a fibro-sarcoma of the meninges comprising the cauda equina at this level, and obliterating the subarachnoid space. (From X-ray plates taken by Dr. Lindsay Locke and Mr. Magnus Redding)

[*To face p.* 769.

Finally the compression, if not relieved, will lead to complete motor paralysis and sensory loss with abolition of sphincter control.

The details of the clinical picture will necessarily vary with the level of the lesion (*see* p. 765).

Additional evidence of spinal compression may be obtained by (1) examination of the cerebro-spinal fluid; (2) X-ray examination after the intrathecal injection of lipiodol.

(1) The cerebro-spinal fluid obtained from below the level of compression as a rule contains an excess of protein. This varies from a slight increase above the normal (0·25 per cent.) to amounts as high as 3 per cent., according to the degree and duration of the compression. When the protein content is extremely high the fluid often contains fibrinogen, and therefore may coagulate in the test tube. In addition, it sometimes exhibits a golden yellow tinge, due probably to the hæmolysis of blood transuded through the walls of obstructed veins.

(2) The practice of injecting lipiodol recently introduced by Sicard is of the greatest value in confirming the clinical diagnosis of spinal compression and determining the upper level of the lesion. Lipiodol is a compound of iodine and fat, which is opaque to the X-rays, of a specific gravity higher than that of the cerebro-spinal fluid, and without injurious effects upon the tissues.

One or 2 c.c. of this substance are injected into the cisterna magna with the patient in the sitting posture; after an interval of half an hour, during which this posture is retained, he is X-rayed in the erect position. By this time, if the subarachnoid space is unobstructed, the lipiodol should have fallen to the bottom of the thecal sac, where it is easily visible upon the plate at the lower border of the first sacral vertebra (*see* Plate XLI., Fig. 1). If the subarachnoid cavity is blocked, as must occur in an advanced case of spinal compression, the lipiodol is held up. In the illustration shown (Plate XLI., Fig. 2) the block is at the upper border of the second lumbar vertebra. The cauda equina below this level was compressed by a large meningeal tumour of a fibromatous nature.

These special methods of investigation are of great value, but it is not to be supposed that they will yield positive results until the degree of compression is considerable. There may therefore be a period in the early stages of the disease during which the clinical history and symptoms are the only guide to the nature of the case.

To the symptoms described above, which are common to all cases of compression, may be added those which belong to the compressing cause.

*Disease of the vertebræ* is commonly—but not always—associated with complaint of *local pain*, accompanied by rigidity and aggravated by any circumstance which tends to strain or jar the affected parts, such as stooping, jumping downstairs, or jolting over rough roads in a motor car. *Tenderness* and *deformity* of the diseased vertebræ are sometimes present. Finally the X-ray plate as a rule serves to demonstrate the presence of bony disease.

*Chronic inflammation of the meninges* as a rule involves several pairs of nerve roots. Root pain, therefore, is usually a prominent symptom and is often associated with reflex spasm of the back muscles leading to local rigidity of the spine.

*New growths of the extra-dural space or meninges* may cause no symptoms beyond those of simple compression, with or without root pains; they may, however, give rise to some tenderness of the overlying vertebra and reflex spasm of the muscles.

**Diagnosis.**—This includes (1) the differentiation of spinal compression from other cases of transverse lesion; (2) the differential diagnosis of the causes of compression.

(1) Spinal compression of acute onset, as from a malignant growth, has to be distinguished from *acute myelitis;* here the chief points in favour of the former diagnosis are the presence of malignant disease elsewhere, characteristic changes in the cerebrospinal fluid, and the results of lipiodol injection.

The more gradual cases of compression have to be distinguished from *intra-medullary tumour, disseminated sclerosis, syphilitic myelitis, amyotrophic lateral sclerosis, syringomyelia*, and *subacute combined degeneration.*

From intra-medullary tumour the differential diagnosis may be possible only at operation; in disseminated sclerosis the presence of scattered lesions as shown in signs or history usually gives the clue, but in certain cases the problem can be solved only by an exploratory laminectomy. Syphilitic myelitis will be recognised if the Wassermann reaction in blood and spinal fluid be included in the routine examination. The absence of sensory changes in amyotrophic lateral sclerosis and the presence of characteristic disorders of sensation in syringomyelia and subacute combined degeneration serve to distinguish these. Finally, the examination of the cerebro-spinal fluid and X-ray examination with lipiodol are often of differential value.

(2) The exact cause of compression can be discovered only by systematic investigation: in the absence of clinical or X-ray evidence of vertebral disease and with negative Wassermann reactions in blood and spinal fluid, exploratory laminectomy should be undertaken.

The **Prognosis** depends chiefly upon the cause of compression. In cases of injury the cord itself as a rule is so severely damaged by the impact that decompression has little effect in producing amelioration of symptoms.

In most cases of tuberculous caries the prognosis is good with adequate treatment provided there is no active tuberculous disease elsewhere. In those cases, however, in which the gradually progressive history indicates a chronic tuberculous meningitis, the outlook is less hopeful. Malignant growths of the vertebrae may continue for months or years before finally causing death from bedsores and cystitis.

The progress of syphilitic meningitis may be arrested by suitable treatment. In meningitis serosa circumscripta division of the adhesions relieves the compression, but the condition is apt to recur. Intrathecal growths of the benign type may be completely removed.

In all cases in which the compressing cause can be removed, whether by means of rest in tuberculous disease or by operation, the degree of recovery which may be expected is proportional to the severity and duration of the symptoms. Total loss of power and sensibility in the lower limbs, provided it has not lasted more than a few weeks, is compatible with complete functional recovery. Once sphincter control is lost the chances of recovery are diminished owing to the complicating factor of urinary sepsis.

**Treatment.** The object of *radical treatment* is to relieve the compression. In the case of new growths this can be achieved only by surgical intervention. Benign tumours can be completely removed, and in some cases of malignant disease decompression may relieve pain. Tuberculous caries is best treated by means of absolute rest in hygienic surroundings. The patient is laid upon a firm bed and extension maintained by traction upon the feet and chin; or he may be fitted from the first with a plaster jacket. Complete rest in the horizontal position should be maintained for at least a year. Subsequently the patient may be fixed in a plaster jacket and allowed gradually to resume the erect posture.

Improvement in the signs of spinal compression may be delayed for several months, but as a rule these cases do well eventually.

Compression from syphilitic meningitis must be treated energetically with the anti-syphilitic remedies (*see* p. 776). If after a sufficient course there is no improvement, operation may be performed in the hope of dividing a cicatricial constriction. The meningeal adhesions resulting from trauma or suppuration should be treated in similar fashion by operation.

*Palliative treatment* in cases of transverse lesion of the spinal cord, whether due to compression or other causes, is directed towards preserving the anaesthetic skin from ulceration, draining and irrigating the bladder, providing relief from

pain and discomfort, and preventing or remedying the contracture of spastic muscles.

For preservation of the skin the patient should be laid upon a water bed. The back should be washed night and morning, and whenever any soiling occurs; after washing and drying, the skin covering the bony prominences (including the heels) should receive a firm and prolonged rubbing from the palm of the hand. A dusting powder of chalk and boric acid should then be used. Care should be taken to avoid sodden or wrinkled sheets likely to produce excoriation, and the patient's position should be changed from time to time during the day in order to avoid constant pressure upon the same bony points. To move the patient two nurses are necessary if he is to be rolled, not dragged.

Should bedsores develop they should be dressed at suitable intervals; stimulant lotions and ointments may be used in succession; of the former Eusol (1 in 2,000) and Lotio Rubra are satisfactory; of the emollients equal parts of Ol. Ricini and Ung. Zinci, or of Ol. Ricini and Tinct. Benzoini Co. are the most useful. Should the bladder be paralysed and retention of urine occur cystitis almost inevitably follows. It may, however, be kept in check by the following measures:

Either a catheter should be passed at six-hourly intervals or the instrument should be tied in and changed at intervals of three or four days. Before the catheter is passed not only should the glans penis be scrupulously cleaned, but the anterior urethra should be washed out with an antiseptic solution. The bladder should also be irrigated night and morning with an antiseptic fluid. Oxycyanide of mercury (1 in 4,000) is satisfactory, or if the urine is strongly alkaline a saturated solution of boric acid with an equal part of water may be used. In such cases also urotropin and acid sodium phosphate should be given by the mouth.

During irrigation care should be taken that sufficient fluid is not injected to cause over-distension, the correct amount being not more than 6 oz. When automatic emptying develops the irrigating fluid should be introduced under the least possible pressure and the bladder allowed to expel its contents if the reflex is evoked. As the reflex becomes more powerful the facilitation reflex (*see* p. 713) should be elicited to assist its performance. It is not safe, however, to discontinue catheterisation until the bladder appears able to expel its contents completely without residual urine (**2**). Evacuation of the bowels should be managed with an enema every forty-eight hours, which minimises soiling; if necessary, a carefully timed aperient of senna pods may be given beforehand.

Reflex spasms of the limbs, which are not only uncomfortable in themselves, but tend to produce bedsores from friction, may be relieved to some extent by a cradle over the lower limbs to prevent contact with the bedclothes. The temperature within this cradle should be maintained at a constant level by means of hot-water bottles. If these measures are of no avail, sedatives, such as luminal, in doses of 1 gram thrice daily, may be employed, or a larger dose may be given at night time.

The onset of contractures should be guarded against by daily passive movements and splinting so long as there is any hope of ultimate recovery.

The electric current should be reserved for such muscles as are wasted and paralysed from involvement of their lower motor neuron supply.

In old standing cases, when spontaneous improvement can no longer be expected, spasticity of the lower limbs may be relieved by section of the posterior nerve roots (Förster's operation). Laminectomy is performed and alternate roots are divided from the second lumbar downwards. This is necessarily a severe operation, and should not be undertaken except in especially favourable circumstances. Adductor spasm of the thighs may be treated by the simpler operation of cutting the obturator nerves, which sometimes gives great relief from discomfort.

## TUMOURS OF THE SPINAL CORD

**Pathology.**—Tumours arising within the spinal cord, as compared with those which originate in the membranes or vertebral column, are relatively rare. They are more commonly found in the cervical and lumbo-sacral enlargements than at other levels. The usual type is the glioma. Sarcoma, tuberculoma and gumma may also occur.

The symptoms of an intra-medullary tumour are those of a gradually progressive transverse lesion of the cord (*see* p. 765). In contradistinction to cases of spinal compression root pains are absent, and the early symptoms, therefore, are complaint of numbness and weakness in the extremities. Other symptoms vary with the level of the lesion. When this lies in the cervical or lumbar enlargements muscular atrophy from destruction of the anterior horn cells is a prominent feature. Partial destruction of the pain conducting fibres sometimes causes complaint of painful paræsthesiæ below the level of the tumour. In the early stages the subarachnoid space remains free, the cerebro-spinal fluid shows no increase of protein and lipiodol injected into the cisterna magna is not held up. Eventually, however, the tumour may produce sufficient swelling of the cord to produce obstruction, and these tests will give positive results.

**Diagnosis.**—The differential diagnosis presents much the same problems as that of spinal compression (*see* p. 769). It may be impossible to distinguish clinically between an intra-medullary tumour and syringomyelia. An accurate diagnosis is usually achieved only as the result of exploratory laminectomy.

**Prognosis and Treatment.**—Some relief may be obtained from decompression, but the growth being as a rule of an infiltrating character is irremovable, and the ultimate prognosis bad.

Deep X-ray therapy should be tried when an accurate diagnosis has been made. Palliative treatment is the same as for other cases of transverse lesion, and has already been described under spinal compression.

## MYELITIS

This term is used to denote an inflammatory process occurring within the substance of the spinal cord and involving all the constituents—blood vessels, nerve cells and fibres, and neuroglia—of the part affected.

**Ætiology.**—It is assumed that in every instance the process of inflammation is due to bacterial agents, but in a number of the cases the causal organism remains unidentified. The spirochæte of syphilis, however, is responsible for a large majority, as proved by the history of primary infection, by the Wassermann reaction in blood and cerebro-spinal fluid, with increase of lymphocytes in the latter, and by histological examination of the spinal cords *post mortem*, which reveals appearances characteristic of syphilitic lesions in other parts of the body.

Apart from syphilis, several of the acute specific fevers appear to be causal or contributory agents in the production of myelitis, among which may be mentioned typhoid, small-pox, measles and diphtheria. Gonorrhœal infection may be another cause.

In cases of this type the infective agents are presumably carried to the spinal cord by way of the blood stream. In other instances it is possible that organisms may reach the cord from some peripheral focus of sepsis through the pathway afforded by the perineural lymphatics.

To these may be added a group of cases in which the invasion of the cord by micro-organisms is secondary to an affection of the meninges. A greater or less degree of myelitis of such an origin may occur in meningitis due to tubercle bacillus, or to various other organisms, such as the pneumococcus, streptococcus and staphylococcus. In some cases the inflammatory process may

spread directly from neighbouring structures, involving the meninges *en route*, as in the case of an ulcerating bedsore.

From the pathological standpoint it is convenient to differentiate between *acute infective myelitis* and *syphilitic myelitis*.

**Morbid Anatomy.**—In *acute infective myelitis* the cord is softened and bulges on transverse section, or may be quite diffluent; but previous to section it may seem hard, from mere tenseness of the membrane containing the swollen cord.[1] To the naked eye the section is congested, minute vessels are visible, especially in the grey matter, and this itself is darker than usual, and its outline is indistinct. Patches of grey tint may be present in the white column, or the whole surface is confused. Under the microscope, in early stages, are found perhaps capillary hæmorrhages, and leucocytes in the lymphatic sheaths and around the vessels. The nerve fibres suffer by breaking up of their myelin sheaths, and here and there occurs a fusiform enlargement of the axis cylinders, which in its thickest part is five or six times the diameter of the normal fibre; this may be due to imbibition of fluid. The nerve cells are swollen, granular, with perhaps fatty globules, and some of them undergo vacuolation. Subsequently both nerve cells and fibres degenerate, the connective tissue increases in quantity, the cells known as Deiters' cells are larger and more numerous, and granule cells are formed in abundance. Finally, in cases of long standing, or passing into a chronic condition, the granule cells disappear also, and the new connective tissue becomes firm, dense, even finely fibrous, so that a definite *sclerosis* is the result.

The lesion may be confined to a few segments (*transverse myelitis*), it may be quite diffuse, affecting a considerable length of the cord (*diffuse myelitis*), or there may be several areas of inflammatory softening, separated by areas of healthy tissue (*disseminated myelitis*).

*Syphilitic Myelitis.*—In this condition the spinal cord may be affected in different ways. On the one hand, syphilitic disease of the vessels may lead to thrombosis and so to softening of the nervous substance, while, on the other hand, gummatous thickening of the meninges may eventually cause compression of the spinal cord, with consequent degeneration of the part encircled. In most cases the condition found is a combination of these two processes, which should be spoken of more correctly as a *syphilitic meningo-myelitis*. Probably the specific toxins of the syphilitic virus also play a part in causing the degenerative changes.

The process is generally limited to a few segments, thus resulting in a transverse lesion of the cord. The blood vessels may show cellular infiltration of the adventitial sheath, together with thickening of the intima, which has sufficed to occlude the lumina. In this case there is complete disintegration and liquefaction of the area supplied by them. Or the appearance may correspond more nearly with that of an inflammatory lesion. There may be no endarteritis in this case, but the vessels are completely surrounded with cellular exudate, which may extend into the surrounding tissues, and is accompanied by degeneration of the nerve elements, apparently of a toxic nature. The leptomeninges are in almost all cases infiltrated with mononuclear cells, and in some instances the dura mater is also involved and may be greatly thickened, so that the whole cord is surrounded by a band of dense fibrous tissue, which in its turn may lead to occlusion of blood vessels by pressure, and so to central softening. Where the meninges are extensively affected the nerve roots become involved in the inflammatory mass, with resultant degeneration of their fibres.

**Symptoms.**—These depend rather upon the localisation of the lesion than upon its pathology. It is useful, however, to distinguish between an *acute* and a *chronic* form of the disease.

[1] Any cord may become soft from decomposition, or may be reduced to a pulp by clumsiness in extracting it from the spinal column.

*Acute myelitis* may be either of syphilitic or of general infective origin. The *syphilitic* variety of acute myelitis occurs more commonly in males than females, generally in young adults with a history of primary syphilis from a few months to five years previously. The symptoms are probably due in most cases to the sudden occlusion of thickened vessels with consequent softening of the spinal cord. The onset, therefore, is frequently sudden, paralysis and anæsthesia occurring without warning and without pain, unless, as sometimes happens, the vascular disease is associated with extensive meningeal involvement, in which case there will probably have been previous complaint of pain in the distribution of the posterior roots affected. There is little or no constitutional disturbance. Examination of the cerebro-spinal fluid reveals an excess of cells and albumin with a positive Wassermann reaction. The Wassermann reaction is as a rule positive also in the blood.

*Acute infective myelitis* may occur at any age without especial relation to sex, but is most often seen in young adults. It is a comparatively rare disease. At the onset there are usually signs of constitutional disturbance, such as headache, pains in the limbs, anorexia and general malaise, with pyrexia of varying degree, and occasionally rigors.

The nervous symptoms may develop suddenly with paralysis and anæsthesia below the level of the lesion, but more usually some days elapse before the acme of the disease is reached. The early symptoms are numbness and tingling in the extremities and slight degrees of motor weakness, together with impairment of sphincter control. Pain is but slight as a rule, but there may be complaint of a dull ache in the back, or a girdle sensation around the chest or abdomen. With the further development of the disease paralysis and loss of sensation generally become complete up to the level of the lesion, and this may progress in an upward direction (*acute ascending myelitis*). There is complete loss of control over the sphincters, bedsores rapidly develop, the pyrexia continues, and the illness progresses after a variable time to a fatal issue, or more rarely it may become arrested with some degree of recovery.

*Acute disseminated myelitis* is a comparatively rare and fatal variety of the infective form, characterised by the simultaneous occurrence of symptoms referable to lesions scattered throughout the cerebro-spinal axis, together with signs of severe constitutional disturbance, and in a number of cases optic neuritis

In all forms of infective myelitis examination of the cerebro-spinal fluid shows, as a rule, an excess of cells, a number of which are polymorphonuclear leucocytes. The Wassermann reaction, however, is negative.

The physical signs in a case of acute myelitis naturally vary with the level of the lesion and with its extent (*see* p. 765).

Death in cases of acute myelitis or softening takes place (1) from pulmonary complications following upon paralysis of the respiratory muscles; (2) from bedsores intensifying exhaustion, or leading to pyæmia; (3) from vesical complications; (4) from intercurrent affections, such as pneumonia or bronchitis. The bladder is extremely liable to cystitis, partly from trophic disturbance, partly from retention of urine; and the use of the catheter may be responsible for the introduction of organisms which set up septic inflammation. When cystitis occurs the urine rapidly decomposes and becomes ammoniacal, unless it is repeatedly removed by the catheter; it contains pus or muco-pus, and readily deposits crystals of ammonium-magnesium phosphate on standing. Almost at any time the septic condition of the bladder may extend up the ureters to the kidneys, and suppurative pyelitis and nephritis will then occur, and the patient may die with uræmic symptoms.

Cases which escape these dangers generally lapse into a chronic condition, which may be of indefinite duration, but sometimes recovery slowly takes place after many months. And a small number of cases get well comparatively quickly,

and these are more common among the milder cases of myelitis following infectious disorders.

Cases of syphilitic origin frequently recover under proper treatment, but there is usually a more or less serious degree of residual paralysis with imperfect control over the sphincters.

*Chronic Myelitis* is almost always of syphilitic origin. The process is one of gradual compression of the spinal cord and its vessels by gummatous thickening of the meninges, together with a certain amount of toxic degeneration of the nerve cells and fibres due to the syphilitic poison. The symptoms therefore are those of a localised spinal meningitis with involvement of nerve roots, to which are added those of compression of the spinal cord. The main feature of these cases in their early stages is the pain due to involvement of the posterior roots. This is of a shooting nature, and if, as frequently occurs, the lesion is in the mid-dorsal region, the pain, as a rule, begins in the back and radiates around the trunk on one or both sides; if the cervical region is affected, the pain will be experienced in the length of one or both arms, and will be accompanied by muscular paralysis and atrophy due to involvement of the motor nerve roots (*see* Internal Pachymeningitis). These symptoms of root irritation and destruction may exist for some months before the spinal cord is seriously involved. The symptoms of a transverse lesion of the cord appear gradually, beginning with a transient weakness or numbness of the lower extremities, together with a difficulty in controlling the sphincters, progressing in a few weeks to a condition of spastic paraplegia with increased tendon jerks and extensor plantar responses. The loss of power and sensibility is not as a rule complete.

If the lesion is situated in the lumbo-sacral enlargement, the most prominent symptoms are pain in the distribution of the sacral nerve roots together with disturbance of sphincter control.

It is not possible to draw any hard and fast line between cases of acute and chronic syphilitic myelitis of the types described above: disease of the vessel walls is an almost constant feature, so that in a case which has begun with symptoms of gummatous meningitis the signs of a transverse lesion of the cord may develop rapidly as the result of thrombotic softening.

Another somewhat rare form of syphilitic myelitis is that in which the pyramidal tracts alone are affected by the specific toxins, resulting in a gradually progressive motor paralysis of the lower limbs with increase of the deep reflexes, extensor plantar responses and spasticity. This is known as *Erb's syphilitic paraplegia.*

Another rare and obscure disease, known as *toxic myelitis,* is especially associated with pregnancy. The symptoms are those of a gradually progressive transverse lesion of the cord, which is not complete, and the tendency is towards spontaneous recovery. The condition may clear up completely after confinement and recur with subsequent pregnancies.

**Diagnosis.**—The recognition of the acute infective form of myelitis is as a rule not difficult in view of the pyrexia and constitutional disturbance. It must be recognised, however, that such signs of general illness may be present in cases of tuberculous caries or malignant growth of the vertebræ, in which sometimes the onset of signs of compression of the spinal cord may be quite sudden. Careful examination of the vertebral column, together with an X-ray picture of that part of the bony canal which encloses the affected segment, suffices as a rule to differentiate these cases. Acute poliomyelitis and Landry's paralysis may be distinguished by the absence of sensory loss and sphincter involvement.

Both the acute and especially the chronic forms of syphilitic myelitis have to be distinguished from compression of the spinal cord by tumours of the membranes or the vertebræ or by spinal caries. Here again the X-ray plate will help to exclude vertebral disease, whilst in the syphilitic cases the cerebro-spinal fluid constantly shows an increase of cells and albumin, together with a positive

Wassermann reaction. The latter is positive also in the blood. Myelitis may sometimes be confused with peripheral neuritis, but in the latter condition there is no disturbance of sphincter control, and, in spite of cutaneous anæsthesia, there is tenderness of the calves to deep pressure.

An extensive patch of disseminated sclerosis may produce all the signs of an acute myelitis, but there is no severe constitutional disturbance such as occurs in the acute infective conditions; examination of the cerebro-spinal fluid serves to exclude syphilis; and a careful review of the history with perhaps the presence of other signs, such as nystagmus, will make clear the diagnosis.

**Prognosis.**—Complete recovery is the exception. Most of the syphilitic cases make considerable improvement under proper treatment, but there is always a residue of paralysis, usually associated with impairment of sphincter control. Most of the cases of acute infective myelitis progress to a fatal issue either in the early stages from the severity of the initial illness or later from urinary sepsis or bedsores.

**Treatment.**—The general treatment of a case of myelitis is the same as that of any transverse lesion of the cord, and has already been described under the heading of compression lesions (p. 770).

*Treatment of Syphilitic Myelitis.*—In cases due to syphilis, salvarsan should be given, and a course of mercury and potassium iodide should be instituted, and continued over a long period. There is some diversity of opinion as to the dosage of salvarsan which should be employed. It appears, however, that for the efficient treatment of syphilis of the nervous system larger doses are required than in the case of syphilis elsewhere, and that patients suffering from this form of the disease are relatively tolerant of the drug.

The following course is recommended: The initial dose of novarsenobillon for an adult male should be 0·45 gramme intravenously, followed after a week's interval by 0·6 gramme, and subsequently by four doses of 0·9 gramme at weekly intervals. An interval of ten weeks should then be allowed to elapse before any more novarsenobillon is given in order to guard against the dangers of delayed arsenical poisoning. The intravenous injections are then begun again with a dose of 0·6 gramme followed by five doses of 0·9 gramme at weekly intervals. Throughout this course of treatment (including the period of rest from arsenical medication) full doses of potassium iodide and mercury should be given. The mercury is best administered in the form of a sterile emulsion, which is injected deeply into the muscles, 1 grain of the metal being given in this manner at weekly intervals. If this method is not practicable, freshly prepared uncoated mercurial pills may be given by the mouth, commencing with 1 grain thrice daily and increasing the dose to 2 grains thrice daily. The patient should be instructed to take particular care in cleaning his teeth while under this treatment on account of the liability of persons taking mercury to develop septic conditions of the gums and mouth. If symptoms of this kind arise treatment by mercury must be suspended for the time being. Potassium iodide may be safely given to most adults in doses of 60 grains three times a day for a fortnight without producing any ill effects, and appears to be much more effective when given in these large quantities. It is well, therefore, to begin with this dosage, which may subsequently be reduced to 20 or 30 grains thrice daily.

One month after the completion of the course the Wassermann reaction may again be tested in the blood and cerebro-spinal fluid, but even if this is negative the patient should not be considered "cured"; if the progress of the disease appears to have been arrested, he may discard treatment for the time being, but whether the Wassermann reaction is positive or negative, he should submit himself annually to a short course of further treatment.

In any case in which, at the end of the initial course, symptoms appear to be actively progressing treatment should be recommended on the same lines after a suitable interval, while a careful watch should be kept for any symptoms of

arsenical poisoning. The dosage of novarsenobillon described above is that recommended for a healthy adult male. In the treatment of individual cases the dose to be employed must be carefully considered in relation to the general condition of the patient, and it should be remembered that some persons are intolerant of arsenic in much smaller quantities than those recommended. The early signs of poisoning to be looked for are severe constitutional disturbance within twenty-four hours of each injection, continuous malaise and gastro-intestinal irritability, jaundice and skin disturbances such as erythema and exfoliative dermatitis. The occurrence of any such symptoms is a warning to discontinue arsenical treatment at once (*see* p. 123).

## HÆMORRHAGE INTO THE SPINAL CORD

This is a very rare occurrence, and contrasts remarkably with hæmorrhage into the brain, which is one of the most common causes of cerebral paralysis.

**Ætiology.**—It occurs in younger persons than does cerebral hæmorrhage, and in males more often than in females. The chief causes are (1) injuries, falls upon the feet, strains, etc.: these form nearly 90 per cent. of all cases of hæmorrhage, and the cervical region of the cord is the part most commonly affected; and in the recent war hæmorrhages were a constant result of direct injury by gunshot or shell, or by concussion, of the spinal cord; (2) alterations in the vascularity of the cord, or structural changes in the walls of its blood vessels; (3) a preceding lesion of the cord, such as a soft ghomatous growth, the vessels of which may rupture, or, perhaps more often, an acute myelitis in its early stage. A primary hæmorrhage is generally confined to the grey matter, and is of small extent, rarely exceeding the size of an almond; but in cases of congestion it is punctiform, and may occupy both white and grey matter. It is also more diffused when secondary to myelitis. Gowers records cases of hæmorrhage into the cavity of congenital syringomyelia; if this is abundant, it will compress or tear up the tissues of the cord.

**Symptoms.**—The onset is often quite sudden: the patient may be seized with acute pain in the back, and then fall, with complete paralysis of motion and sensation below the seat of the lesion. In other cases the symptoms may be more gradually developed in the course of a few hours. The limbs are mostly relaxed, and there may be clonic contraction in the muscles, either immediately or in a few days. The symptoms are subsequently those of an acute local myelitis—paralysis, loss of sensation, the reflexes increased after a short period, during which they are diminished, and the bladder affected. With a central hæmorrhage there may be dissociation of sensations (*see* Syringomyelia); and with hæmorrhage into a syringomyelic cavity sensory effects may be more marked than motor, because the cavity so often occupies the posterior part of the cord. There may be elevation of temperature after a few days from secondary inflammation, and this myelitis may spread upwards and downwards. Secondary degeneration of the lateral and posterior columns frequently follows, and accompanying this is spastic rigidity of the limbs, with increased knee jerk and ankle clonus. But if the grey matter is much destroyed in the cervical or lumbar regions, wasting of the corresponding muscles may supervene. Trophic changes, cystitis, and bedsores are also not infrequent.

**Diagnosis.**—This depends on the sudden onset of the symptoms, and in the vast majority of cases a history of injury, but the disease may be confounded with a *hæmorrhagic myelitis*, and with *meningeal hæmorrhage*. Prodromal symptoms of even very short duration, and fever, make myelitis or softening probable. *Meningeal* or *extramedullary hæmorrhage* is distinguished by the signs of nerve irritation rather than nerve impairment, such as severe pains in the distribution of certain nerves, and muscular cramps, as compared with anæsthesia and paralysis in spinal hæmorrhage. The bladder is less likely to

be affected, bedsores do not occur, and the disease is less fatal. In *acute poliomyelitis* it is possible that there is in some cases hæmorrhage, but the spinal symptoms are less sudden. The absence of back pain, the initial fever or convulsion, the freedom from vesical, rectal and sensory symptoms, and the rapid localised atrophy, readily distinguish this disorder from spinal hæmorrhage.

**Prognosis.**—Death is rarely caused by the primary lesion if the patient survives the first few hours; later on urinary sepsis or bedsores may lead to a fatal issue. Some degree of recovery may be expected in almost all cases, and the final prognosis may be gauged in some measure by the rate of progress in the early stages. If improvement in power sensibility and bladder control manifest themselves within a week or two of the injury, the outlook is good. If there is no return of power at the end of three months, the paralysis is likely to remain permanent. In all cases affecting the cervical enlargement the atrophic paralysis of the hands due to destruction of anterior horn cells is likely to persist in some degree as a permanent disability.

**Treatment.**—The patient should be placed in the prone position, if possible, or on the side, to prevent the spinal cord being in the lowest part of the body. Of greatest importance is attention to the bladder and the prevention of bedsores (*see* p. 771).

## CAISSON DISEASE

(*Compressed Air Illness, Divers' Paralysis*)

Divers and those who work in caissons at great depths below the surface of the earth, such as are equivalent to a pressure of three or four atmospheres, are liable to a form of paraplegia, which supervenes on their return to the surface. The symptoms may be mild or severe, and come on within an hour of the changed conditions. In mild cases there is only a little weakness with numbness of the lower extremities, which passes off in a few hours or days; in other cases the arms are also affected, or the symptoms are more severe and last several weeks: in others again there is coma, and the patient may die in it. Some persons suffer more often from less serious symptoms, such as pains in the ears, deafness, giddiness, severe pains in the legs, arms, and shoulders (called by them the *bends*), nervousness or intense excitement, dyspnœa or choking sensations (*chokes*), and bleeding from the nose, mouth or lungs.

From Dr. Keays' report on the men engaged in the construction of tunnels under rivers in New York, where the pressures averaged an additional 32 lbs. per square inch (that is + 2 atmospheres, or 47 lbs. absolute) and sometimes reached + 42 lbs. (or 57 absolute), it appears that of 10,000 men employed in the course of two years 3,692 suffered from symptoms. The percentage occurrence of such symptoms was as follows: bends, 90; vertigo, 5·33; dyspnœa, 1·62; unconsciousness and collapse, 0·16; spinal symptoms, 2·16; and hemiplegia, only 0·11. Middle-aged and old persons are more susceptible to these troubles than the young, and the fat and alcoholic than the thin and temperate.

The immediate cause of the paralysis, as of other symptoms in various parts of the body, is that the increased pressure forces an excessive quantity of nitrogen into the blood and tissues, and that on the reduction of the pressure, or *decompression*, unless it is conducted with extreme slowness, bubbles of nitrogen form in the blood vessels, and obstruct the circulation, forming emboli. In the ligaments, fasciæ, periosteum, Haversian canals of bone, muscle spindles and nerve sheaths, they cause the bends; in the subcutaneous tissue, itching of the skin, and in severe cases mottling; in the pulmonary capillaries, dyspnœa and oppression; in the labyrinth, vertigo. In the spinal cord bubbles form especially in the white matter, and they have been found to be more frequent in

the antero-lateral columns than the posterior, and more in the cervical region than in the lower parts of the cord. In extreme conditions patches of necrosis are found. Fissures have been found in the cord, filled with leucocytes, and surrounded by small areas of inflammation; and hæmorrhages are sometimes present.

The liability to the occurrence of bubbles in a tissue is proportionate to the inactivity of its circulation, and to the quantity of fat it contains. For fat absorbs five times as much nitrogen as water; and since fat as well as the fibrous tissues is relatively so little vascular, there is greater difficulty in the removal of nitrogen from them when reduction of pressure is rapidly effected.

The symptoms referable to the spinal cord are different in different cases. They usually occur within an hour or two of arrival at the surface, and consist of weakness in the lower extremities, with or without numbness or formication. They may increase rapidly to complete paraplegia. Often there is retention of urine. Sometimes the arms are similarly affected. The reflexes are often increased, and the gait may be stiff or spastic, when the patient can walk. In bad cases incontinence of urine and fæces may occur later, and persistence of the symptoms with the addition of cystitis or bedsores may lead to a fatal result.

Much more rarely a cerebral lesion is evident in the occurrence of hemiplegia, monoplegia, localised facial paralysis, or mental symptoms, as delusions and hallucinations. And in fatal cases of caisson disease there is often a combination of the spinal symptoms with unconsciousness and collapse.

The prognosis is on the whole good. Bends are generally quite temporary. Paraplegia, unless complicated by cystitis and bedsores, does not generally get worse after the first few hours: many divers have had paraplegia more than once. The deaths in Dr. Keays' series of cases of caisson disease numbered twenty, or 0·54 per cent.; but as eight of the fatal cases showed spinal symptoms, the fatality in the spinal cases must have been at least 10 per cent.

**Treatment.**—If any symptoms are developed when the diver reaches the surface, or the workman leaves the caisson, that is, when either has undergone decompression too rapidly, he should at once, or as soon as possible, be subjected to *recompression*: if a diver he may again descend to the former level; or either patient may be placed in a suitable chamber, or *lock*, in which the air pressure may be increased to the desired extent. Under these circumstances the symptoms, even of the most alarming kind, as a rule rapidly subside; and if now decompression be conducted more slowly—by a more gradual ascent to the surface, or by the more gradual reduction of pressure in the lock—the patient may remain perfectly well. Bends may be treated with friction, massage, and hot baths, or, if very painful, by injection of morphia. Paraplegia, if the opportunity for recompression has been lost, requires treatment similar to that of myelitis.

**Prevention.**—Researches and experiments on this subject have been carried out by L. Hill, Haldane, Boycott, and others, in order to solve the problem of proportioning the rate of decompression to the number of pounds of excess of pressure, and to the time during which the workman has been subjected to it. It is agreed that the rate of descent into the water, *i.e.* the rapidity of compression, has no effect upon the result. A rate of decompression which is generally safe appears to be equal to about one atmosphere in fifteen or twenty minutes. But opinions differ on an important point—as to whether the process should be uniform, or should be conducted in stages with intervals of some minutes during which no change takes place. Boycott advocates a rapid reduction of pressure at first with completion at a slower rate. The Admiralty has issued an elaborate table based on the former (stage) system with times proportioned to the depth of the diving operation, and to the duration of stay at the depth chosen. Hill points out that muscular exercise in the lock, or immediately after decompression, helps to prevent symptoms by quickening the circulation and facilitating the absorption of gas.

## DISSEMINATED SCLEROSIS

*(Multiple Sclerosis, Insular Sclerosis, Sclérose en Plaques Disseminées)*

This disease is characterised by the development of numerous patches of chronic inflammation or sclerosis throughout the brain and spinal cord.

**Ætiology.**—The disease is somewhat more frequent in females than in males. The symptoms are mostly noted for the first time in youth or early adult life, and in nearly two-thirds of 200 cases collected by Byrom Bramwell the onset occurred between the ages of sixteen and thirty. The antecedent conditions to which the disease has been in many cases attributed are (1) different acute infectious diseases, such as typhoid fever, malaria, influenza, pneumonia, scarlet fever; (2) mental worry, or shock, grief, excitement; or (3) cold, wet, injury and fatigue. The only way in which it can be safely suggested that the alleged causes operate to produce the lesions is by lowering the vitality of the tissues, so as to render them susceptible to some unknown toxic agent or agents.

Experimental researches upon the nature of the virus have so far yielded negative results. Attempts at transmission of the disease to animals have been fruitless or inconclusive, and the claims of some workers to have discovered the causal organism in the form of a spirochæte remain unproven.

**Pathology.**—Though little can be said with certainty as to the origin of the disease, it is probable that toxins carried to them by the blood cause degeneration of the myelin sheaths, and that simultaneously or somewhat later hyperplasia of the neuroglia occurs, resulting ultimately in sclerosis.

**Morbid Anatomy.**—The surface of the spinal cord, medulla oblongata, pons, and the base of the brain presents a number of irregular patches of pinkish-grey colour, rather sharply outlined and contrasting with the natural white colour of the medulla, pons, and crura. On section the discoloration is found to extend inwards so as to form deposits of a round or oval shape, ranging in size from that of a pea to that of a hazel-nut, generally harder than the normal nervous tissue, and even leathery or cartilaginous, sometimes projecting above the level of the section, sometimes sunken below it. Recent patches are dark grey, older patches more yellowish grey, and less translucent. They affect the white matter more than the grey matter; thus in the spinal cord the greater part of the cornua is unaffected, and in the cerebrum they are best seen on section of the hemispheres, which are dotted with the grey areas, and the walls of the lateral ventricles are often invaded. The Sylvian aqueduct also is commonly surrounded by areas of sclerosis. They are not frequent in the cerebellum, but may invade the olfactory bulbs and the spinal and cranial nerve roots. Under the microscope the outline of the patch or nodule is much less distinctly marked than it appears to the naked eye. The nodule consists chiefly of fibrous or finely fibrillated tissue, developed by overgrowth of the neuroglia; within this area the nerve fibres have lost their myelin sheaths, but many axis cylinders persist. In the earliest stages of the lesions, changes of an inflammatory nature are found in and around the small vessels, namely, vascular congestion, perivascular infiltration with lymphocytes, polymorphonuclear and plasma cells, and in some cases capillary hæmorrhages. In the next stage the myelin sheaths degenerate and are broken up into fatty droplets, which are removed by large mononuclear cells, known as compound granular cells. There is at the same time a reaction on the part of the surrounding glia cells (comparable with the connective tissue cell reaction around inflammatory foci elsewhere in the body). Finally, when the products of degeneration have been completely removed a dense patch of glial overgrowth represents the scar of the original lesion. Owing to the excessive glia reaction, neighbouring foci tend to be bound together in a common sclerotic plaque which is visible to the naked eye.

In sections obtained by the Weigert-Pal method the healthy fibres are stained

# PLATE XLIV.

**Sections from two cases of Disseminated Sclerosis stained by the Weigert-Pal Method. The pale areas represent islands of degeneration and sclerosis.** ***The healthy fibres are stained black.***

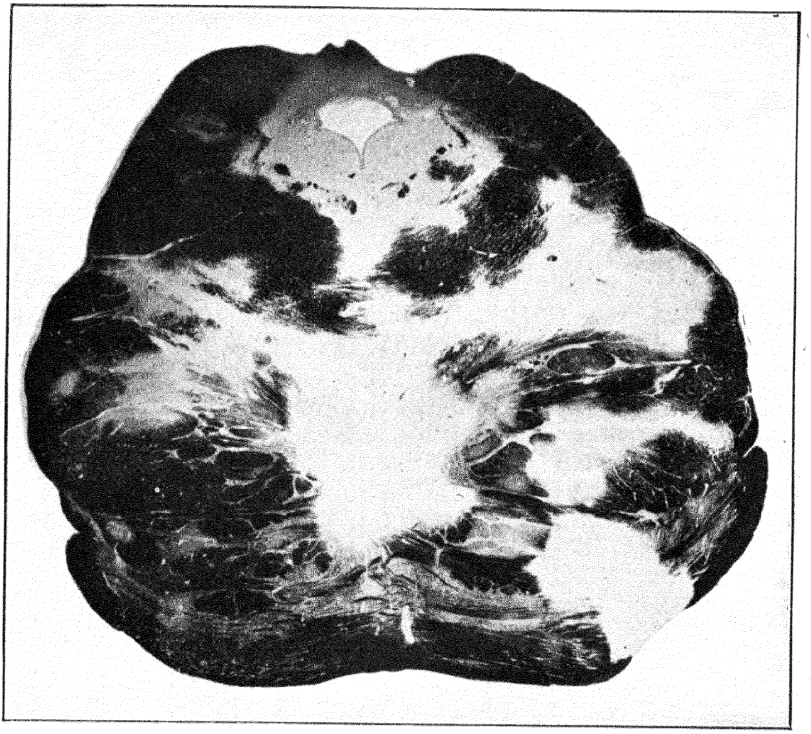

FIG. 1.—Upper Level of Pons × 3½.

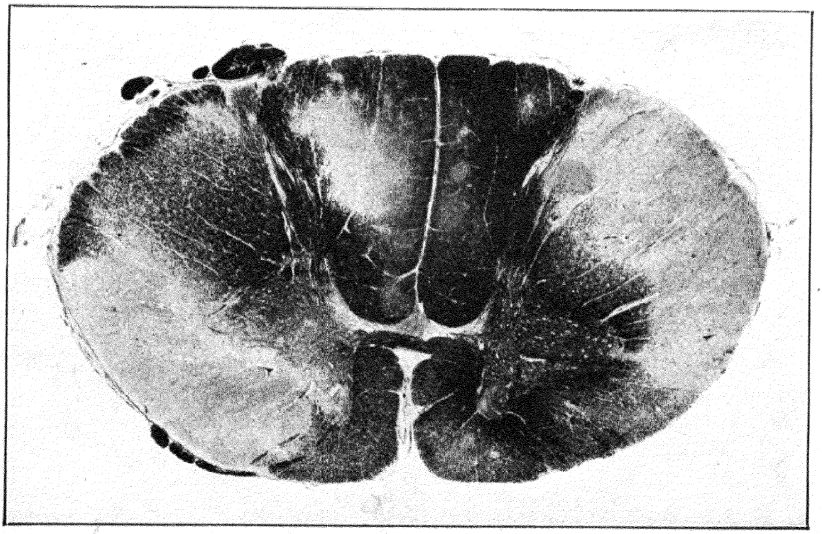

FIG. 2.—Fifth Cervical Segment × 7.
(From photographs taken by Dr. J. R. Perdrau.)

[*To face p.* 781.

black, while the plaques of glial sclerosis, being left unstained, stand out in sharp contrast against the dark background (*see* Plate XLIV., Figs. 1 and 2).

**Symptoms.**—The most characteristic feature of the illness in its early stages is the variety, and often the transient nature, of the symptoms, depending as they do upon a random and haphazard distribution throughout the cerebro-spinal axis of minute inflammatory foci, each of which in its initial stages causes a much greater disturbance of function than ultimately persists. As the disease progresses, with the accumulation and coalescence of repeated patches of sclerosis, the total area of permanent damage steadily increases, so that the symptom picture becomes more uniform and more constant.

The manner of the onset is itself variable; most frequently it is somewhat sudden, with symptoms referable to a small lesion of the longitudinal fibre tracts, the optic nerves or the periaqueductal grey matter. Thus the patient may complain of a numbness in some area of trunk or limbs, a dragging of one foot, some slight difficulty in controlling the bladder, double vision, or a mistiness before the eyes. Examination at the time may reveal slight evidence of organic damage, such as impairment of sensibility, inequality or absence of the abdominal reflexes, or an abnormal plantar response, but these signs, together with the symptoms, commonly disappear in the course of a few days or weeks, to be succeeded after a variable interval of quiescence by the development of new troubles elsewhere or renewal and intensification of the old. In other cases the symptoms begin more gradually, perhaps with progressive weakness or stiffness of one or both legs or an increasing tremor of the hand. Even of these cases, however, the majority show remissions and exacerbations, so that the course of the illness is seldom continuous.

*The nature of the earliest symptoms* depends upon the situation of the first patches of the disease, and is therefore variable in the extreme. It is to be remembered, however, that the morbid process chiefly involves the white matter of the cerebro-spinal axis; that, owing to the great length of the main sensory and motor tracts, these are most likely to suffer damage; and that on account of their functional importance such damage is most likely to result in symptoms. Moreover, on account of their greater length the fibres going to and coming from the lower limbs are more vulnerable than those connected with the arms.

Not infrequently, therefore, the first symptoms take the form of paræsthesiæ —that is, sensations of numbness, tingling, tightness, coldness or heat—referred to various areas of the trunk or limbs. Careful examination may reveal diminished tactile sensibility, but of more common occurrence are signs of damage to the posterior columns, revealed by impairment of the senses of position and passive movement, diminution or loss of the vibration sense, and in the hands astereognosis. In the arms extensive damage of this nature may give rise to symptoms which are somewhat characteristic and by no means uncommon. The patient complains that the hand is useless: he is unable to employ it usefully for purposes of eating, buttoning his clothes, and so on. Examination reveals no loss of power, and as a rule little or no impairment of tactile sensibility, but there is gross loss of sense of position, with consequent ataxia and astereognosis.

The commonest form of motor disturbance is weakness of one or both legs of the type due to pyramidal damage. Thus the group of muscles earliest affected is that of the dorsiflexors of the ankle, so that the foot drags along the ground, and this in the early stages may only be noticed after excessive exercise, as at the end of a long day's walking. With the weakness, which in more severe cases affects the flexors at knee and hip joints also, is associated spastic rigidity of the extensors, so that the patient may complain of stiffness rather than paralysis. With this also there is sometimes complaint of "cramps" in the legs, or of involuntary flexor movements, described as "jumping upward" of the lower limbs, which are especially likely to occur in bed at night.

When symptoms of this nature are prominent they are associated with the signs of a pyramidal lesion, namely, loss of the abdominal reflexes, exaggeration of knee and ankle jerks, and an extensor plantar response, which are most marked on the side chiefly affected.

Further symptoms of common occurrence in the early stages seem to be due to a selective incidence of the morbid process upon certain parts of the nervous system, particularly the optic nerves, and the areas surrounding the Sylvian aqueduct in mid-brain and pons. In relation to the former site complaint is made of dimness of vision, frequently of a quite transitory nature, or a defect of visual acuity which is not due to any error of refraction may be discovered in the course of a medical examination for some trade or profession. The lesion is situated, as a rule, in the optic nerve some distance from the nerve head, and ophthalmoscopic examination therefore reveals pallor of the optic disc, which may amount in the later stages to pronounced optic atrophy with serious impairment of vision. In rare cases, however, the inflammatory patch may be situated actually in the nerve head, when it gives rise to a true optic neuritis.

In connection with the periaqueductal grouping of the lesions reference has already been made to diplopia from involvement of the oculo-motor nuclei or nerve. This is nearly always of a quite transitory nature, lasting a few hours or a few days, and occurs at one time or another in about a third of the cases. It is often one of the earliest symptoms, and may be neglected at the time and afterwards be forgotten by the patient, unless his attention is specially called to the point.

Nystagmus, from interference with the fibres connecting the vestibular with the oculo-motor nuclei, is seldom noticed by the patient, but is present in more than three-quarters of the cases, often at an early stage. It is usually obtained only on lateral deviation of the eyes, is most commonly bilateral, and at first irregular, being well marked at some times, hardly to be noticed at others. It is, however, one of the most characteristic signs of the disease.

Motor inco-ordination of cerebellar type, probably attributable to lesions of the superior cerebellar peduncles at or near the point of their decussation, is sometimes an early symptom, and is present in about half the cases at some time. It is shown most frequently in the form of intention tremor of the arms, and a disturbance of the speech which depends upon inco-ordination of the articulatory musculature. The intention tremor is so called because it is not as a rule present when the arm is at rest, but is observed when the patient attempts to take hold of an object, or in the performance of the finger-nose-finger test. One limb oscillates irregularly to the right and left, or up and down, without regard to the object aimed at, and there is a tendency to overshoot the mark. In addition to this, a nodding, to-and-fro tremor of the unsupported head is frequently observed. The speech has been described as staccato, or scanning, and is of the type already described under Disturbances of Cerebellar Function (*see* p. 714).

As has already been noted, the cerebral hemispheres are commonly riddled with sclerotic patches, and to this must be ascribed the mental changes which are present in the majority of cases before the end, and are often noticeable at an early stage. They are of the general type occurring in other organic diseases of the brain, and consist of emotional instability and lack of control, indifference to the serious nature of the malady, lack of judgment in practical affairs, and defective memory. It is the combination of slight mental changes of this type with a history of transient weakness, or vague sensory disturbances, which so frequently leads to an erroneous diagnosis of hysteria in the early stages.

In addition to those already described, the disease may be ushered in by a variety of other symptoms of less common occurrence. Thus lesions of the cerebral hemispheres may give rise to hemiplegia of sudden or gradual onset, patches involving the central connections of the vestibular nerves may lead to attacks of giddiness or vertigo, or a predominant affection of

the cerebellum may cause symptoms of pure cerebellar disease. There are also cases in which the morbid process begins in the lumbo-sacral enlargement, and loss of control over the sphincters is a prominent and early symptom.

In rare instances also the grey matter of the anterior cornua is affected, with resultant atrophy of the corresponding muscles, usually those of the hands

The *course of the disease* is also variable. In a majority of cases the illness begins in a subacute manner—the patient suddenly notices a numbness of the hand or dragging of the foot—and its progress is characterised by a succession of remissions and exacerbations. The latter may be manifested in the form of new symptoms or intensification of those already present. The duration of the remissions is variable, but may extend over months or years. In some cases, however, the disease runs what appears to be a chronic and progressive course from the beginning. It is doubtful, however, whether one can separate a chronic from a remittent type, for, on the one hand, a case which has begun in the more usual manner with remissions may finally become steadily progressive, and, on the other hand, there may be subacute exacerbations in the course of a case which has for some time shown a chronic progressive character.

*In the later stages* the patient becomes bedridden from paralysis of the lower limbs, which commonly develop the position of spasticity in flexion Control over the sphincters is lost, and bedsores frequently develop. Even at this stage, however, tactile sensibility often remains unaffected, though examination with the tuning-fork usually reveals loss of the vibration sense in the lower limbs. The knee and ankle jerks may now be exaggerated or absent, the plantar responses are extensor, and the abdominal reflexes are lacking.

The disease may last several years—ten, fifteen, or twenty; but about 20 per cent. die within five years of the onset, and another 30 per cent. between five and ten years (Bramwell). Death may take place early from an apoplectic seizure, or the patient may be bedridden for years, with paraplegia, and die from the accidents associated with that condition, or from intercurrent disease.

**Diagnosis.**—There is no disease in which a carefully elicited history is of greater value. The diagnosis rests upon the proof of *multiple* lesions of the central nervous system, and this may only be forthcoming from a consideration of the story of a transient diplopia, numbness, or weakness in conjunction with the physical signs. The danger of a mistaken diagnosis of *hysteria* has already been mentioned. As a rule the detection of some slight sign of organic disease, such as nystagmus, absent abdominal reflexes, an extensor plantar response, or pallor of the optic discs, will permit of the differential diagnosis, but there are cases in the early stages in which it may sometimes be wiser to suspend judgment. The combination of nystagmus with tremor and inco-ordination of the arms may give rise to suspicion of *cerebellar tumour*. In the latter condition, however, the symptoms are all referable to a single lesion, and in the later stages there are symptoms of increased intracranial pressure. The spasticity and weakness of the lower limbs need to be distinguished from similar conditions arising from *compression of the spinal cord* by tumour or spinal caries.

*Syphilis* of the central nervous system in certain of its forms may give rise to a picture closely resembling that of multiple sclerosis, and in any case in which there is a doubt as to the diagnosis the Wassermann reaction should be done with blood and cerebro-spinal fluid. In disseminated sclerosis this reaction is uniformly negative, and the cerebro-spinal fluid shows no changes in its cell or protein content. The colloidal gold curve, however, is often abnormal, resembling that found in neurosyphilis. Some cases of *encephalitis lethargica* in which the spinal cord is extensively involved may at first be mistaken for multiple sclerosis; this mistake is especially likely to occur with a case of encephalitis in which the illness is protracted with comparatively long remissions; the differential diagnosis may be made as a rule by careful consideration of the history of the case,

particularly the nature of the onset and the occurrence of pyrexia or drowsiness in encephalitis.

The **Prognosis** is unfavourable. If the patient be seen in an acute exacerbation it may generally be said that some improvement will follow, but the great probability is that by the accumulation of repeated patches of the disease he will finally become crippled and bedridden. Remissions simulating recovery are not uncommon, but only in very rare instances has the progress of the illness seemed to be arrested. Mental deterioration occurs in a majority of cases before the end.

**Treatment.**—There is at present no specific remedy for the disease. On the ground that the cause is probably a protozoal infection most of the drugs which are known to have an effect in such diseases have been tried. Of these arsenic is generally considered to have some effect in retarding the progress of the disease, and is usually given by the mouth. Theoretically, if of any value at all it should be more effective when given intravenously, and cases treated in this manner appear to have longer remissions and progress less rapidly. A course of four weekly injections of N.A.B. may be given at six monthly intervals and liquor arsenicalis prescribed by the mouth in the intervening periods. A watch must be kept for symptoms of arsenical poisoning. During an exacerbation rest in bed is essential, but the patient should be encouraged to get about as soon as his symptoms have abated. Hurst has shown that in a number of cases of the remittent type a high proportion of the disability is in the nature of a "functional" perpetuation of signs originally organic, and that this may be removed by suitable methods of persuasive re-education. For the spasticity hot baths and exercises are of use. Extreme degrees of mental and physical strain should be avoided, and life should be conducted on sound physiological principles.

Palliative treatment in the later stages of the disease must be conducted on the same lines as those already prescribed for transverse lesions of the spinal cord (*see* p. 770).

## SYRINGOMYELIA

This is a disease in which elongated cavities are formed in the centre of the cord as the result of a combined process of new growth and degeneration. The symptoms depend in the early stages upon destruction of the constituents of the grey matter; in the later stages the fibre tracts are also involved.

**Ætiology.**—The disease occurs more commonly in men than women, and begins as a rule between the ages of twelve and thirty. It is sometimes associated with spina bifida, with cervical ribs or other congenital deformity.

**Pathology.**—The essential feature of the disease is a new growth of glial tissue with subsequent degeneration and cavity formation. This begins as a rule in one of the posterior horns of grey matter close to the central canal and extends forward into the anterior horns, occluding, or sometimes communicating with, the central canal. There is a tendency for the morbid process to spread upwards and downwards in the cord, but the lumen of the tube thus formed may exhibit great variations at different levels (*see* Figs. 83, 84). The portions of the cord chiefly affected are the cervical and upper dorsal regions, the lumbar region and the medulla (syringobulbia).

The **Symptoms** in the early stages are referable to destruction of (1) the anterior horn cells; (2) the fibres conveying sensations of temperature and pain which decussate in the grey matter; (3) the cells of the lateral horn of grey matter from which originate the pre-ganglionic fibres of the sympathetic chain.

Since the disease process usually commences in the lower end of the cervical enlargement, the symptoms are therefore:—

1. Progressive muscular atrophy, commencing in the small muscles of the hand. This is accompanied by fibrillary tremors of the muscles affected, leads frequently

to contractures, and tends progressively to involve the muscles of the arm (*see* Fig. 85).

2. Loss of sensibility to temperature and pain. This is sometimes noticed by the patient in his daily life, in that he may notice that hot sparks from an anvil,

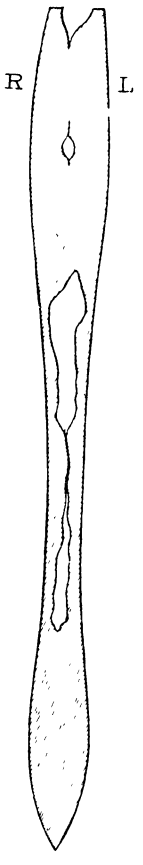


FIG. 83. — Spinal Cord of an Infant showing the Cavity in Syringomyelia. (Diagrammatic)

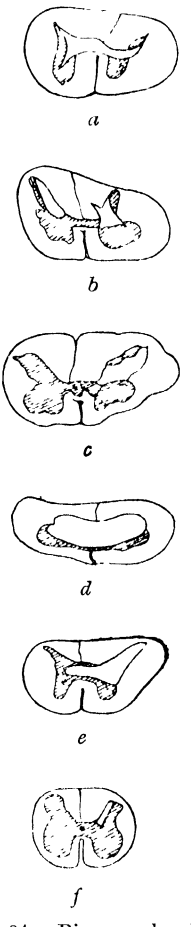


FIG. 84. — Diagram showing Transverse Sections of a Spinal Cord affected with Syringomyelia. *a*, *b*, *c*, *d*. Cervical region. *e*. Thoracic region. *f*. Lumbar region. The asymmetry is produced in the course of preparation.

boiling water, or a lighted cigarette end burn the skin without giving rise to pain, or may be observed by the physician in the shape of numerous scars upon the hands and arms, resulting from such unrecognised injuries.

3. Vasomotor and trophic disturbances of the parts affected. These may take the form of cyanosis, coldness, engorgement and œdema of the hand, bullæ, ulcers, loss of the nails, painless whitlows (Morvan's disease), or degenerative

changes in the joints, especially the shoulder joint, similar to those met with in tabes dorsalis.

In addition, from involvement of the cervical sympathetic there may ensue contraction of the pupil, partial ptosis and enophthalmos (*see* p 721).

The disease may spread upwards into the medulla and pons, and then gives rise to nystagmus, wasting of the tongue, and sometimes diplopia from paralysis of the sixth nerve.

Spinal curvature in the form of scoliosis is also frequently associated with the disease.

In the later stages there is pressure upon the longitudinal fibres of the cord with resultant weakness and spasticity of the lower limbs, with increased reflexes and extensor plantar responses, due to pyramidal involvement, and loss of all forms of sensation, due to destruction of the ascending fibres.

The bladder and rectum are not as a rule affected unless the cavity formation involves the lumbar enlargement. In this case also there will occur atrophy, trophic changes and dissociated anæsthesia in the lower limbs.

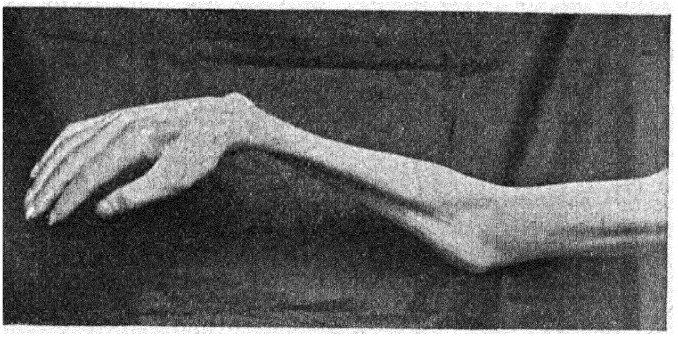

FIG. 85.—Illustrates Muscular Atrophy in a Case of Syringomyelia (*After Turner and Stewart.*)

**Diagnosis.**—In the early stages the disease is likely to be confused with progressive muscular atrophy with cervical rib, or with any of the causes of compression of the cervical cord, such as tumour, spinal caries, and chronic meningitis. Careful sensory examination will, as a rule, serve to complete the diagnosis, but the distinction from an intramedullary tumour may be impossible.

**Prognosis.**—The disease is very slowly progressive, and the patients live, more or less crippled, for many years.

**Treatment.**—This can scarcely be more than symptomatic. Carefully graduated X-ray treatment applied over the spine has appeared to be beneficial.

## TABES DORSALIS

(*Locomotor Ataxy*)

Tabes dorsalis is essentially a gradual progressive degeneration of the lower afferent neurons, first manifesting itself in the posterior columns of the spinal cord; this is accompanied or followed by an overgrowth of the neuroglia. Similar changes may occur in the optic nerve, and in the central portions of the afferent cranial nerves. Tabes is therefore a disease affecting the sensory neurons, the disorder of locomotion from which it was originally named being due to loss of sensations of position and passive movement, with resultant inco-ordination of the movements in walking. Later experience shows that the ataxia is often a very late phenomenon, and, indeed, may be entirely absent; while there

# PLATE XLII.

**Sections of the Spinal Cord from a case of Tabes Dorsalis; stained by the Weigert-Pal Method. The pale areas in the posterior columns represent the degenerated tracts.** ***The healthy fibres are stained black.***

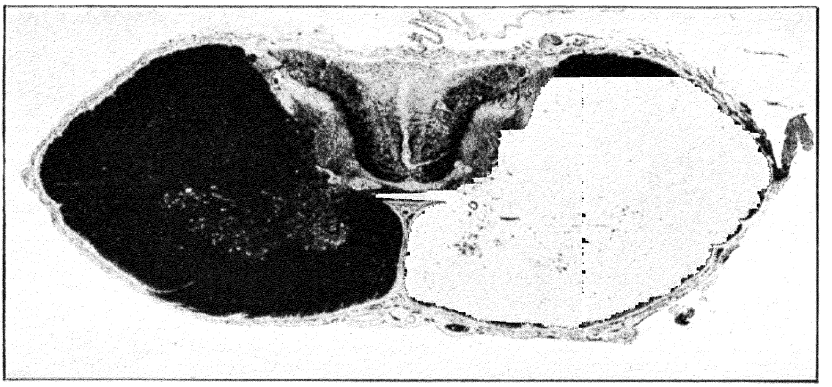

FIG. 1.—Fourth Cervical Segment.

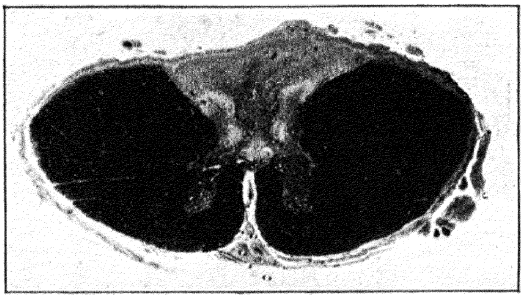

FIG. 2.—Fifth Thoracic Segment.

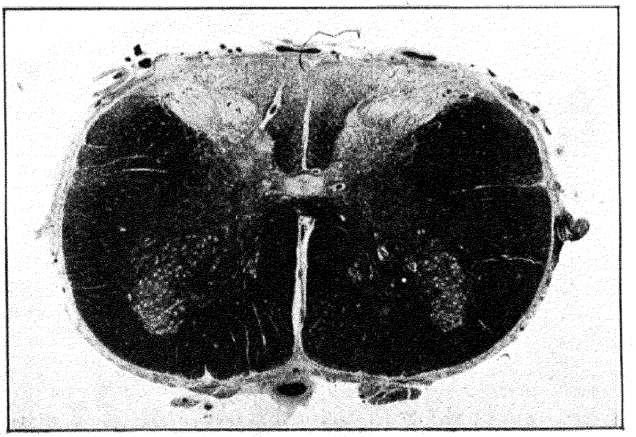

FIG. 3.—Lumbo-sacral Enlargement.
(From photographs taken by Dr. J. R. Perdrfu).

[*To face p.* 787.

are other disturbances in the realm of sensation which are more constant, and are our more usual guides to the diagnosis of the disease.

**Ætiology.**—Syphilis is now considered the only prime factor in the causation of this disease, which is regarded as one of its latest manifestations. Clinical evidence in favour of this conclusion was already abundant, and has been confirmed by the results of the Wassermann reaction in blood and cerebro-spinal fluid. Noguchi has recently demonstrated the *Spironema pallidum* in the spinal cord from a case of tabes. Cold and wet, injuries to the spine, excessive labour, and sexual excesses may act by depressing the general vitality, and by a process of exhaustion prepare the tissues to suffer from the influence of the toxin.

The disease arises generally between five and fifteen years after syphilitic infection, and is therefore commonest in the middle period of life, between thirty and fifty, and it is noteworthy that tabetics comparatively rarely manifest other signs of constitutional syphilis. It is more frequent in men than women, in about the proportion of ten to one. In the juvenile form, however, which appears in a small proportion of congenital syphilitics during adolescence, the distribution between the sexes is more nearly equal.

**Morbid Anatomy.**—The change constantly found in the spinal cord is a degeneration of the ascending nerve fibres, and sclerosis of neuroglia, in the posterior columns. It is seen as a grey discoloration of the white matter in the fresh specimen, whilst in sections stained by the Weigert-Pal method the pallor of the diseased columns stands out in evident contrast with the deep staining healthy tracts (Plate XLII., Figs. 1—3). In ordinary cases it is the fibres entering the lumbo-sacral segments of the cord which are affected, and the degeneration is therefore most evident in the postero-external columns in this region (*see* Plate XLII., Fig. 3), whilst in the cervical enlargement, owing to the displacement inwards of these fibres, it appears in the postero-internal columns. In cases involving the arms the postero-external fibres are degenerated in the cervical region (*see* Plate XLII., Fig. 1). The association fibres in the posterior columns remain intact, the degenerative process being confined to those fibres which represent the direct continuation upward of the processes of the posterior root ganglia. Under the microscope degeneration and disappearance of the nerve fibres, increase of the glial tissue, which is fibrillated, and thickening of the walls of the arteries are observed. In advanced cases changes may also be detected in the grey matter, such as atrophy and degeneration of nerve cells or fibres in the posterior horns, in Clarke's columns, and even in the anterior cornua. The pia mater is often thickened over the posterior columns, or even over the lateral columns as well, or completely round the cord. The cerebro-spinal fluid, as may be shown by lumbar puncture during life, contains an excess of lymphocytes, shows a cloud of globulin by the Nonne-Apelt test, contains more than the normal percentage of albumin, and in about 70 per cent. of cases gives a positive Wassermann reaction, which is, as a rule, found also in the blood. The Wassermann reaction is therefore of great value in confirming a doubtful diagnosis in an early case. But in some 20 per cent. of cases, in which the diagnosis is certain on clinical grounds, even though the patient may admit a history of syphilis, the Wassermann reaction is negative in both blood and fluid.

The posterior nerve roots are generally atrophied down to the spinal ganglia, which are mostly healthy, as well as the mixed nerves beyond them; but atrophy of the peripheral nerves has been also found, mostly of those supplying the skin and the joints, and in the legs more commonly than in the arms. In the optic nerves, in cases alluded to, are found atrophied nerve fibres and overgrowth of neuroglia. Atrophy or degeneration of the nuclei of the third, fourth, fifth, sixth, eighth, and twelfth cranial nerves have in different cases been seen, as well as of the Gasserian ganglion, but the lesion of reflex iridoplegia in tabes is not certainly known, though it is probably situated in the periaqueductal substance of the mid-brain.

**Pathology.**—Little is absolutely known of the pathology of tabes beyond the fact, which has already been stated, that the disease is primarily a process of destruction of the posterior column nerve fibres due to the activity of the *Spironema pallidum* or its toxins. Since the action in tabes dorsalis and in general paralysis of the insane appears to be directly upon the nervous elements, these two diseases are often spoken of as *parenchymatous syphilis of the nervous system* to distinguish them from those forms of syphilis in which degeneration of nerve cells and fibres is secondary to affections of the meninges and blood vessels.

Owing to the chronic nature of the illness, opportunities for pathological study have been limited mainly to end results, but in early cases the point of initial lesion appears to be at the entry of the fibres into the cord, where, as they traverse the pia mater, the neurolemmal sheath is lost. It has been suggested that the poison is conveyed to the cord by way of the perineural lymphatics of the peripheral nerves, the fibres of which are protected against its action up to the point above mentioned by the neurolemma. Orr and Rows have succeeded by subcutaneous inoculation of bacteria in animals in producing lesions of the corresponding posterior roots commencing at this point, and a similar pathological picture has been obtained in animals infected with trypanosomiasis, a disease which is sometimes known in man to produce lesions resembling those of tabes.

The explanation of the delay between the original syphilitic infection and the onset of the disease is even more difficult. Fildes and McIntosh have put forward the theory that in the early stage of general dissemination of the virus the nervous tissues become sensitised in the course of an immunity reaction, and accordingly bear the brunt later of an exacerbation of activity on the part of spirochætes which have been lying dormant in the tissues.

**Symptoms.**—As might be expected from its pathological anatomy, tabes is essentially a disease of sensory symptoms, especially in its early stages. The significance of these is readily missed, and yet, seeing that the best we can hope for our treatment is to arrest the progress of the disease, it is most desirable that we should recognise it before gross defects of muscle and joint sensibility have led to a condition of disablement from ataxy. In the early (pre-ataxic) stage the characteristic symptoms are pains in the limbs, cutaneous hyperæsthesia of the trunk or limbs, loss of knee jerk, and loss of pupil light reflex. This stage may last for months or years.

The pains known as *lightning pains* occur in 95 per cent. of the cases; they are severe shooting, stabbing, or darting pains, as a rule in the lower extremities, sometimes in the arms. They come on suddenly, and, it may be, with such severity as to make the patient start up in bed, or cry out. Each pain lasts only for a few seconds, but they generally soon recur and continue thus coming and going in different parts of the limbs for several hours. They may then disappear, and not return till the next day, or till after an interval of days or weeks. They thus present the greatest irregularity both as to recurrence and duration. The patient frequently puts them down to "rheumatism," but if questioned gives a description so graphic as to be quite unmistakable.

The hyperæsthesia of the skin, usually that covering the upper part of the trunk or of the legs, is often noticed early by the patient, who complains that light-stroking contact, for instance putting on and off the underwear, gives rise to a peculiar, unpleasant sensation, which may also be aroused by extremes of temperature, so that sea bathing becomes intolerable, and he becomes wary of letting himself down into a hot bath.

The knee and ankle jerks are, as a rule, abolished in this stage owing to lesions on the sensory side of the reflex arcs concerned; and it is important to realise that abolition is preceded by sluggishness of the jerk, and that one limb is often affected before the other. A marked inequality therefore between corresponding tendon jerks of the two sides is of diagnostic importance.

Argyll-Robertson pupils are found in more than four-fifths of the cases. In the fully developed condition they are unequal, irregular in outline, excentric, and react to accommodation but not to light, but here again it is to be remembered that disappearance of the light reflex is preceded by sluggishness.

In addition to this, there is often, quite early, slight *anæsthesia* of the feet and lower part of the legs, and occasionally temporary paralysis of one or more of the ocular muscles, leading to diplopia, or squinting, or ptosis, according to the muscle involved.

A slight degree of bilateral ptosis together with compensatory wrinkling of the brow, in a permanent attempt to keep the eyes more widely open, frequently leads to a somewhat characteristic facial appearance (*see* Fig. 86).

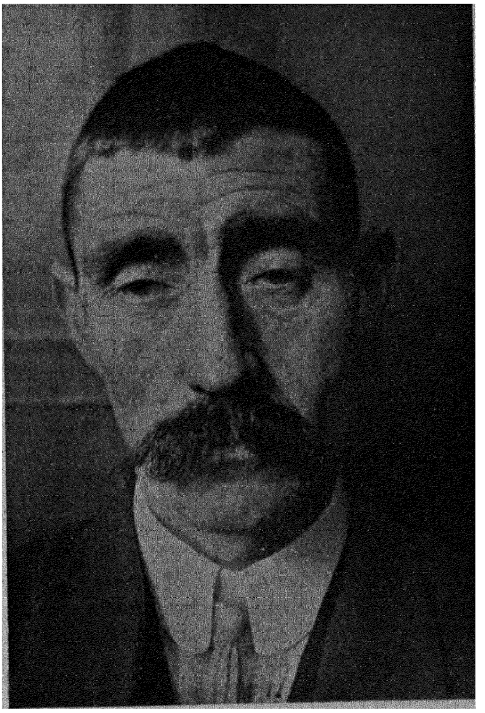

Fig. 86.—Illustrates the Characteristic Appearance of the Face in a Case of Old-standing Tabes Dorsalis—the Tabetic Face. (*After Turner and Stewart.*)

In the second stage, that of the developed disease, or actual locomotor ataxy, the prominent feature is the *muscular incoordination* of the lower extremities, and this is associated with increased anæsthesia and other sensory disorders, and impairment of the functions of the bladder. Other rarer conditions, which are generally first observed in the early stage, are the so-called gastric and other *crises;* certain trophic disturbances; optic nerve atrophy, myosis, and other ocular conditions.

The *ataxy,* as indicated by the epithet *locomotor,* is chiefly and first noticed in the lower extremities, and is confined to them in a great number of cases. At first the patient is only slightly unsteady in his gait, finds a difficulty in walking quite straight, separates the legs a little to meet this difficulty, keeps his eyes carefully fixed on the path he is following, and readily loses his balance when trying to turn. In the dark, when the guiding sensations of sight are removed, he is still more unsteady. If Romberg's test be employed (*i.e.*, standing unsupported with heels together and eyes closed), the patient staggers or falls. In a later stage, walking can still be accomplished, but the legs are drawn up or jerked up in a disorderly way; they are often thrown sharply forwards, and the heels are brought down with force upon the ground. Turning is still more difficult than before, and has to be effected with great care and with the assistance of a stick, a wall, or a friend. Nevertheless the muscular power remains good. If the patient sits in a chair he can keep his leg extended in front of him against any ordinary attempts of the medical man to flex it. Moreover, the muscles are of normal bulk, and give the normal electrical reactions. The distance which the patient can walk is lessened to a mile or two, on account of the great waste of strength involved in these disorderly and, therefore, ineffective movements. In later stages the ataxy may be such that he cannot walk at all without assistance from

sticks, a chair, or a friend on either side; and, finally, he may have to take to his bed.

The arms are sometimes affected late in the disease, but the ataxy is generally less extensive than that observed in the legs. In some cases, however, of so-called cervical tabes the disease may begin as an affection of the upper limbs.

*Hypotonia* is another result of the loss of reflex muscular tone, already shown in the absence of tendon jerks. There is in this condition a remarkable mobility of the limbs, so that joints can be passively flexed or extended to quite abnormal degrees without the resistance and pain which are caused in healthy individuals. Frenkel states that hypotonia is a constant symptom even in the earliest stages.

*Cutaneous Anæsthesia* is variable. It most commonly takes the form of impaired sensibility to cotton wool, pin-prick and temperature, affecting the feet and legs, the ulnar borders of the arms, and the trunk between the level of the nipples and that of the umbilicus. Analgesia to pin-prick is the most prominent feature of this loss, and this is often found also in the skin around the nose. The anæsthesia of the feet gives rise to a feeling which the patient describes as "like walking on cotton wool." Numerous other modifications of sensation are observed in different cases: burning or gnawing pains in the extremities, more continuous than the lightning pains; a sense of constriction in the legs, groins, genitals, or trunk, the latter often described as "girdle pain"; tingling, pins and needles, and sensations of cold or heat.

The deeper tissues are also analgesic, so that the muscles of the calf or the tendo Achillis may be pinched vigorously without giving rise to pain. Loss of the senses of position and passive movement may be demonstrated by the patient's ignorance with his eyes shut of the position of his limbs, and of their movement by the observer, and is also shown by inco-ordination in the performance of more or less complicated muscular actions. Loss or diminution of sensibility to the vibrations of a large tuning fork is an additional sign of value, and is usually demonstrable over the malleoli, shins, and sacrum.

*Visceral anæsthesia* has been noticed in the form of absence of testicular sensation, diminished sensitiveness of the epigastrium to blows, and of the trachea, globe of the eye, and mamma to pressure.

The *bladder* is often affected as follows: In early stages, there is irritation, with frequent micturition, and the necessity of passing urine directly the desire is perceived. Later on the detrusor is weakened, and the urine comes in a sluggish stream, or merely dribbles away. Sometimes there is retention with incontinence from overflow. The *sphincter ani* is generally weakened, or fæces are passed unconsciously from insensibility of the anus. Sexual power is commonly lost, and impotence may be an early symptom of the disease.

The functional disturbances of the viscera, called *crises*, occur in about one-fifth of the cases. The *gastric crisis* (*crise gastrique*) is the most frequent; in each attack there is severe pain in the epigastrium, passing through to the back, or extending from the groins up to the shoulders, accompanied by vomiting, at first of clear liquid, often in great quantity, later of bile, and even of blood. Pain may occur without vomiting, or vomiting without pain. There may be associated cutaneous hyperæsthesia, but there is no constant rigidity of the abdominal muscles such as is found in peritonitis. Often also there is palpitation or irregularity of the heart. These attacks come suddenly without previous symptoms of dyspepsia, and subside leaving the functions of the stomach quite normal. Their duration may be quite brief, or they may last for several days, reducing the patient to a condition of severe exhaustion and collapse. The other *crises* that have been described are a *rectal crisis*, consisting of paroxysmal pain in the rectum with severe tenesmus; sensations referred to the genital organs—*sexual crisis*; paroxysmal diarrhœa, or *intestinal crisis*; paroxysms of renal pain, or *renal crisis*; pain in the bladder or urethra—*vesical* or *urethral crisis*; *laryngeal crisis*, consisting of laryngeal spasm, with inspiratory and expiratory stridor,

cough, and dyspnœa; and *nasal* or *bronchial crisis*, when there are paroxysms of sneezing or coughing.

The *trophic* disturbances which occasionally occur in tabes dorsalis are œdema of the feet, local sweating, ecchymoses under the skin, brittleness of the hair, and herpes, the last three in connection with severe pains. The skin of the sole of the foot becomes thickened, or blisters, or may present a deep circular and conical depression, the *perforating ulcer*. The nails become thickened and furrowed, or fall off, and are slowly renewed. Teeth decay, or may fall out within a short time.

In occasional cases, about 6 per cent., important changes take place in the *bones* and *joints*. The bones become brittle; the compact tissue has been found thinner and more porous; fractures occur spontaneously, or with the slightest amount of force; and a great deal of callus forms in the process of union. The lesions in the joints are known by the name of *Charcot's disease* (*see* Fig. 87). The changes are almost identical with those which occur in osteo-arthritis, or rheumatoid arthritis (*see* p. 669), namely, erosion of cartilage, wasting of the head of the bones, ossification of the ligaments, and new bony outgrowths. Clinically they are characterised by rapid painless swelling from effusion into the joint, and subsequently extreme mobility and grating. While some hold that these changes are the direct result of the withdrawal of trophic influence from the part, others consider them to be due to the external injuries, strains, etc., which ataxic limbs are so much more likely to suffer than healthy ones, and, further, that they are of infective origin (*see* p. 669).

FIG. 87.—Illustrates Arthropathy (Charcot's Joints) of the Right Knee and Ankle in a Case of Tabes Dorsalis. (*After Turner and Stewart.*)

*Primary atrophy of the optic nerve* occurs in about one-fifth of the cases, starting on the temporal side; this results in contraction of the visual field from the periphery inwards, and loss of vision for colours (*dyschromatopia*) in the following order: green, red, yellow, blue, and violet. Ultimately there may be complete blindness. In a very large proportion of the cases with optic atrophy, locomotor ataxia is absent; but the ocular lesion is accompanied by one or more of the following: Argyll-Robertson pupil, loss of knee jerks, pains in the limbs, gastric crises.

Deafness, paralysis of the abductors of the vocal cords, severe headache, glycosuria, and apoplectiform or epileptiform attacks occasionally occur.

**Diagnosis.**—Tabes dorsalis has to be recognised in its early stages before inco-ordination is pronounced, and it has to be distinguished in the stage of ataxy from other disorders affecting the power of the lower extremities. The lightning pains are generally very characteristic, but the absence of knee jerk and the loss of light reflex of the pupil are the distinctive features. In a more

advanced case the same two signs are of service, and in addition the inability to stand with the eyes shut, or to turn with steadiness. Where locomotion is much interfered with, the case contrasts with *paraplegia* from myelitis by the retention of absolute muscular power, and by the normal bulk of the muscles, with absence of rigidity. Where muscular wasting and weakness supervene there may be more difficulty in diagnosis, but the long history and the course of the symptoms will assist. *Cerebellar* disease also causes ataxia, but it is generally of a reeling, staggering kind, the patient swaying from side to side, falling over, crossing the legs to recover balance, and presenting a close resemblance to a drunken man, whereas, in the locomotor ataxy of tabes, for a time the gait may be steady in direction, but the feet are jerked forward, and the heel or flat of the foot is brought down sharply on the ground. The two diseases have also their special accompanying symptoms.

It is important to remember that a patient with gastric crises, a perforating ulcer of the foot, or "Charcot's joint," may be entirely unaware of any locomotor symptoms; and in such cases the knee jerk and the pupils should be at once tested. Otherwise a diagnosis might be formed which would lead quite unnecessarily to an abdominal operation.

*Peripheral neuritis* in its ataxic form may be generally recognised by the atrophy and tenderness of the muscles, the "dropped foot," the high-stepping gait, the altered electrical reactions, the normal pupils, and possibly by an alcoholic history.

The cases of *subacute combined degeneration*, to be described shortly, in which the lateral columns are degenerated as well as the posterior, present spastic phenomena and muscular weakness, as well as ataxia of locomotion, but if the sensory symptoms predominate in the early stages may require careful differentiation from tabes.

The Wassermann reaction should be carried out both in the blood and cerebro-spinal fluid; but it must be remembered that the reaction is negative in 20 per cent. of cases of tabes. The cerebro-spinal fluid should also be examined for the presence of albumin and cells. The diagnosis of tabes is suggested if the fluid contains more than 0·025 per cent. of albumin or more than 5 lymphocytes per cubic millimetre.

**Course of the Disease.**—The symptoms are often stationary for very long periods, and the disease may last twenty years or more. It may be many years before ataxia is observed (pre-ataxic stage), and even when ataxia is developed to a high degree the patient may live to old age. Cases with optic atrophy, which is so constantly present without ataxia, often progress slowly, or even improve for a time. Similarly gastric crises may occur long before any ataxia is present, but with the same associates as above. The majority of patients die, not from the disease itself, but from intercurrent affections, such as pneumonia, phthisis, bronchitis, apoplexy, or other independent ailment. Cystitis and renal complications, bedsores and pyæmia, and rarely laryngeal spasm, are direct consequences of the disease, and may be fatal. Some cases terminate in general paralysis of the insane, a cerebro-spinal disorder resulting from syphilis, in exactly the same way as tabes dorsalis.

**Prevention.**—This consists in the prevention of syphilis in the first place, and in the early adoption of vigorous antisyphilitic measures if the disease is contracted.

**Treatment.**—This may be considered under two heads, specific and symptomatic.

*Specific.*—Of recent years it has become a universal practice to employ salvarsan or similar compounds in the treatment of all forms of syphilis, and cases of tabes are usually treated by such means. The principles to be employed are those laid down for the treatment of syphilitic myelitis (*see* p. 776). In the majority of cases symptoms may be alleviated and the course of the disease retarded or

prevented by efficient treatment. As in the treatment of syphilitic myelitis, mercury and iodides are also given in tabes.

The treatment of tabes by "salvarsanised serum" has also been carried out. After administering salvarsan intravenously, some blood is removed from the patient, and after it has clotted the serum is injected intrathecally. There is not much evidence that this treatment is of value. The procedure involved is laborious and the result often painful to the patient. It is not very commonly used.

*Symptomatic.*—For the lightning pains antipyrin, aspirin, acetanilide or phenacetin may be given singly or in combination at three-hourly intervals while the bouts are in progress. It is important to avoid opium for fear of the real danger of establishing a habit.

A gastric crisis may sometimes be aborted by giving a bath as hot as the patient can stand it, and then putting him to bed with hot water bottles and the administration of morphine, $\frac{1}{4}$ grain, hypodermically. At the height of an attack morphia may be necessary to relieve the pain, or this may be controlled by repeated doses of chloretone in moderate amounts. If the attack is of long duration, rectal feeding is necessary.

In cases in which repeated gastric crises have rendered the patient's life almost intolerable, a surgical procedure which has met with some success is exposure of the spinal cord and division of the antero-lateral ascending tracts in the upper thoracic region.

Attention to the bladder is very necessary, and the catheter should be used if any urine is retained. The expulsive power may be increased by strychnine or incontinence lessened by belladonna.

For the laryngeal crises inhalation of amyl nitrite may give relief.

Much may be done for the ataxia by means of graduated exercises. This is essentially a process of re-education by which the patient is taught to compensate for his loss of sense of position by co-ordination of visual impressions with muscular movements. These exercises may be begun in bed, the patient being instructed to practise simple co-ordinated movements such as raising the leg to a certain height and then placing it nicely in contact with an object set up for the purpose. Thence he is led through movements in the sitting and standing postures to those of orderly progression, full use being made throughout of the visual sense. Thus he is taught to stand opposite a wall and raise the foot to touch various marks upon it, while supporting himself with his hands, to advance by placing his feet accurately upon footmarks outlined on the floor, and so on. In this way it is often possible to obtain for a bedridden patient some measure of ambulatory activity.

## PRIMARY LATERAL SCLEROSIS

(*Primary Spastic Paraplegia*)

The condition in which weak or paretic limbs are rigid from spastic contraction of their muscles is known as *spastic paraplegia*, and is qualified as primary if it occurs spontaneously and independent of any such local disease of the cord as transverse myelitis, disseminated sclerosis, or pressure from tumour, aneurysm, or tuberculous abscess. It is essentially a degenerative change in the pyramidal tract—that is in the upper motor neurons situate in the spinal cord.

**Ætiology.**—The disease is rare; it is more frequent in males than in females, and occurs mostly between the ages of twenty and forty. It has appeared in two or three members of the same family. No adequate cause has hitherto been found; but it is certainly not caused by syphilis.

**Pathology.**—The pathological condition underlying the symptoms is a *sclerosis* of the spinal cord occupying the whole length of the pyramidal tract, or the posterior part of the lateral tract—hence a *lateral sclerosis*. The histological change is identical with that seen in the posterior sclerosis of tabes dorsalis, that

is, degeneration and disappearance of nerve fibres with increase of neuroglial tissue. Whether this is due to a congenital inability to survive, or to toxins, however produced, has yet to be known. Slight lesions of the cerebellar tract and of the columns of Goll may also be present, but do not appear to contribute to the symptoms.

**Symptoms.**—The disease develops very slowly and insidiously. It begins with weakness and stiffness of the lower extremities; the legs feel heavy, the patient soon gets tired in walking, and, as time goes on, the distance he can walk without fatigue gets less and less. The knee-jerk is excessive; ankle-clonus and Babinski's sign are present; and the cutaneous reflexes are usually increased. After a time he has to help himself with sticks; the legs are rigidly extended and firmly adducted, and it is almost impossible to get one in front of the other. The rigidity is nearly constant, or, if the muscles relax slightly, they at once contract on a touch, or the stimulus of attempted movement. The arms are generally unaffected, but if they are involved they take up a position of rigid flexion. In some cases the muscles of the trunk are involved. The muscles mostly remain in good condition, and the electrical reactions are normal; sometimes, however, the excitability of the muscles is slightly increased, at others diminished, to both currents. Sensation is unaffected beyond the occurrence of rheumatoid pains and some tingling or numbness, and the bladder and rectum are in most cases free from the functional disturbances common in myelitis and tabes dorsalis. The course of the disease is very chronic, and it may last twenty or thirty years; indeed, it is less threatening to life than are other forms of chronic spinal disease.

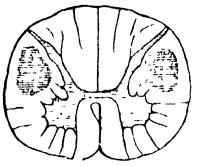

FIG. 88.—Diagram to show the Position of the Lesion in Primary Lateral Sclerosis

**Diagnosis.**—The recognition of a spastic paraplegia is not difficult; weakness of the legs, spastic rigidity, increased reflexes and Babinski's sign are its characteristics; while sensation, the bladder and rectum, and cerebral and ocular functions are normal. The important point to be decided is whether the condition is *primary* or *secondary*, and very careful inquiries and examination should be made before concluding that a case of rigidity is not secondary to local disease of the spinal cord, such as transverse myelitis or softening, or compression. In primary disease of the lateral tracts weakness and stiffness come on gradually and simultaneously, whereas in secondary cases there is at first decided paralysis, rigidity only supervenes later, and sensory symptoms and girdle pain are often present. Many cases which have been at first diagnosed as primary lateral sclerosis prove finally to be instances of the chronic, progressive type of disseminated sclerosis. In others wasting of the hands or bulbar palsy supervening points to amyotrophic lateral sclerosis as the correct diagnosis. In others again involvement of the posterior columns together with anæmia indicates the presence of subacute combined degeneration. Nor must it be forgotten that there is a type of chronic syphilitic myelitis in which symptoms of spasticity are the main features. The Wassermann reaction in blood and cerebro-spinal fluid should, therefore, be obtained in every case. Lateral sclerosis may also form a part of general paralysis of the insane.

A spastic paraplegia occurring in young children or infants is generally of cerebral origin (*see* Infantile Cerebral Diplegia).

**Treatment** by drugs is unsatisfactory. Physical strain should be avoided, and for the spasticity passive movements should be performed daily after a hot bath.

## SUBACUTE COMBINED DEGENERATION

This is a disease *sui generis* characterised by progressive widespread degeneration of the fibres of the posterior and lateral columns of the spinal cord, usually

# PLATE XLIII.

**Sections of the Spinal Cord stained by the Weigert-Pal Method to illustrate the conditions found in Amyotrophic Lateral Sclerosis, Subacute Combined Degeneration, and Friedreich's Ataxia. The pale areas represent the degenerated tracts.** ***The healthy fibres are stained black.***

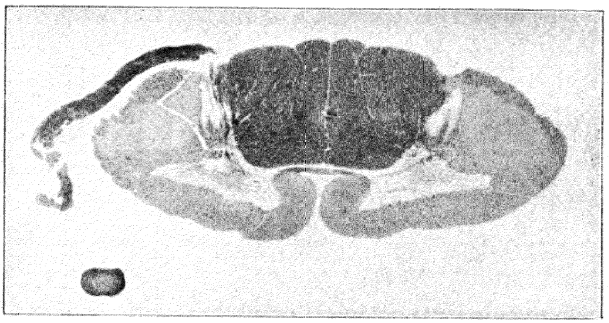

FIG. 1.—Amyotrophic Lateral Sclerosis. Mid-thoracic Level. The fibres of the pyramidal tracts are degenerated, and to a lesser extent the other fibres of the anterior and lateral columns.

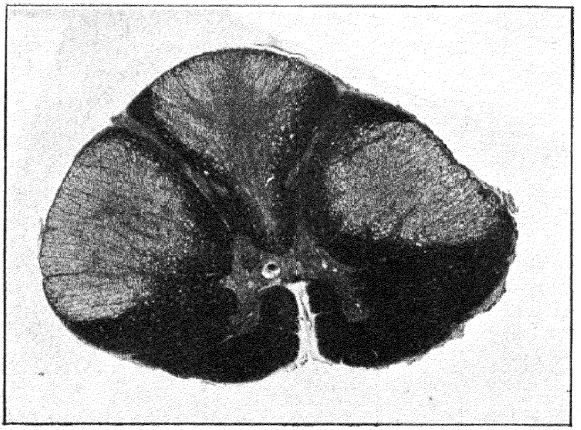

FIG. 2.—Subacute Combined Degeneration. Fourth Thoracic Segment. Degeneration of posterior columns and crossed pyramidal tracts.

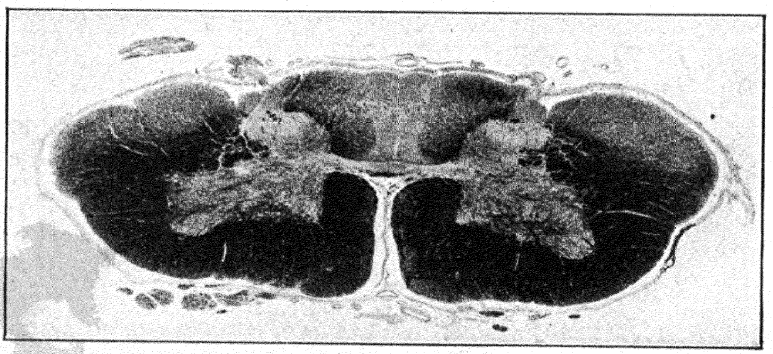

FIG. 3.—Friedreich's Ataxia. Sixth Cervical Segment. Degeneration of the posterior columns, the dorsal spino-cerebellar tracts, and to a lesser extent the pyramidal tracts.

[*To face p.* 795.

associated with pernicious anæmia, and leading as a rule to a fatal termination in from three months to four years.

**Ætiology.**—This disease is most common between the ages of fifty and sixty, the extremes at which the onset has been recorded being thirty and sixty-five. Nothing is known of its true ætiology. Its association in the great majority of cases with pernicious anæmia suggests that in the latter disease the same toxins which are responsible for the blood destruction have a selective action upon the posterior and lateral columns of the spinal cord.

**Pathology.**—The disease may begin with signs of anæmia, but, on the other hand, in a large number of cases the nervous symptoms are well established before any changes are apparent in the blood, and some cases may run a rapid and typical course without showing anæmia at any time. It appears, therefore, that the degeneration of nerve fibres cannot be secondary to the anæmia, but rather that both anæmia and degeneration are concomitant effects of some unknown pathological cause with a selective incidence upon the central nervous system and the blood-forming elements. Combined degeneration of the posterior and lateral columns may also occur in other blood diseases, such as the leukæmias, in the profound conditions of cachexia met with in malignant disease, and in *ergotism* and *pellagra*.

**Morbid Anatomy.**—Macroscopic examination shows the spinal cord to be of normal size, in contrast with the condition found in diffuse sclerotic processes. On cross section the degenerated areas in the white matter appear as greyish patches, which in the mid-dorsal region usually form a complete circle around the central grey matter. The degeneration of the longitudinal fibres is the essential feature of the disease. The Weigert-Pal staining method shows, in cases where death has occurred in the earlier stages, that this process commences in the centres of the ascending posterior and descending lateral columns of the lower thoracic region (*see* Plate XLIII., Fig. 2); thence it extends centrifugally until, in cases where the fatal termination has been delayed, there are no medullated fibres remaining except for the short association fibres surrounding the grey matter.

Similar patches of degeneration occur in the lumbar and cervical enlargements, and the disease process extends longitudinally by coalescence of these patches. The first sign of degeneration is swelling of the myelin sheaths, which then undergo fatty degeneration and break up into droplets which are stained black by the Marchi staining method. With this the axis cylinder breaks up and disappears. The degeneration products are subsequently absorbed, leaving a fine network of neuroglial and connective tissue elements containing empty spaces in place of the destroyed nerve fibres. There are no material changes in the walls of the blood vessels and relatively little overgrowth of neuroglia.

The nerve cells are not affected except as a secondary result of degeneration of their axons. Thus the cells of Clarke's column show vacuolation and chromatolysis consequent upon the degeneration of the spino-cerebellar fibres, and similar changes are found in the Betz cells of the pre-central cortex as a result of destruction of the pyramidal tracts.

In rare cases there are found scattered minute hæmorrhages in the cord, but these probably occur as terminal events, and cannot be considered as a cause of the degeneration of nerve fibres.

**Symptoms.**—The illness is most commonly ushered in by paræsthesiæ of the extremities. These begin with numbness and tingling of the fingers and toes, extending to the hands and feet, and are soon followed by impairment of cutaneous sensibility of the same distribution. The nature of the sensory loss varies; sensibility to pin-prick is generally lost earlier than that to cotton wool, and with extension of the area involved the loss to pain precedes that to touch. In the earlier stages the glove and stocking distribution is retained, but as the anæsthesia progresses from the lower limbs on to the trunk the upper level follows a segmental line. Evidence of affection of the posterior columns is usually

found at an early stage of the disease in the form of loss of sensations of position, passive movement and vibration in the extremities. Other subjective sensory phenomena commonly met with are a sensation of tightness around the waist and lightning pains resembling those of tabes.

On the motor side signs of damage to the pyramidal fibres are usually predominant in the early stages. The patient complains that his feet feel heavy, and drag as he walks, especially when he is tired. Later the lower limbs become stiff, and the gait is that of a case of spastic paraplegia, though careful examination will probably reveal an ataxic element due to the loss of muscular and joint sensibility. As the posterior columns become more completely involved the spastic picture gives place to that of flaccid paralysis, and the gait resembles that of the tabetic. At this juncture the patient, relieved of his stiffness, may be led to the false hope that he is improving, but with the further development of the ataxy soon becomes bedridden. While this is the more usual sequence of events, the posterior columns may be more severely involved from the first with the production of a picture in the early stages resembling that of tabes or peripheral neuritis. In this case, when sooner or later the pyramidal fibres are affected, there is no development of spasticity, since the afferent impulses necessary for the production of this state of reflex tonus are already cut off. The paraplegia does not as a rule reach the upper limits of the regions supplied by the cervical enlargement, and even in the later stages of the disease the affection of the upper extremities remains limited to peripheral anæsthesia and weakness.

The sphincters are always affected in the later stages of the disease. At first there may be difficulty in starting the act of micturition, which is followed by loss of control, with the rapid development of complete incontinence of urine and fæces. At this period cystitis is likely to occur, with the sequence of pyelonephritis, leading to a fatal termination, and bedsores inevitably develop.

The mental state toward the end is one of lethargy, drowsiness and complacency, occasionally developing into a state of feeble delirium. The special senses and cranial nerves are not affected. The reflexes are variously affected at different stages of the disease. In the more usual type beginning with spastic paraplegia there are increased knee and ankle jerks at first with extensor plantar responses; in the later stages the jerks are first diminished and finally lost, while the plantar responses remain extensor. In the type which begins with a flaccid paraplegia from the first the knee and ankle jerks are lost early and do not return. The abdominal reflexes are as a rule retained until a late stage of the disease.

Anæmia is recognisable in about one-half of the cases when first seen. It is of the type met with in pernicious anæmia with low red cell count, high colour index, anisocytosis, poikilocytosis, an absolute decrease in the polymorphonuclear leucocytes, and the presence of nucleated red cells. The skin often shows a yellowish tint, against which the presence of a bright malar flush may give the face a characteristic aspect.

Although anæmia of this type appears in a great majority of cases before death, there are some which run a course typical of that described above to a fatal termination without ever developing the characteristic blood picture.

Gastric achylia is the rule and may be present without anæmia.

The **Diagnosis** in the early stages is often difficult, but complaint on the part of a person between the ages of fifty and sixty of peripheral paræsthesiæ as described above should always arouse the suspicion of subacute combined degeneration, and lead to a careful investigation of the reflexes and a complete blood examination.

The flaccid type of the disease with absent tendon jerks and peripheral anæsthesia has to be differentiated from *polyneuritis*, the presence of tenderness of the muscles in the latter condition being a useful point of distinction. The presence of extensor plantar responses and sphincter disturbance in subacute combined degeneration are of further value.

The spastic type must be distinguished from *disseminated sclerosis*, in which the presence of nystagmus and intention tremor and a history of transient disturbances of sensation, power, or vision are commonly present.

Cases of syphilitic origin in which both lateral and posterior columns are affected in a process of parenchymatous degeneration, *i.e.* some cases of dementia paralytica and of tabes with lateral column involvement, may closely resemble subacute combined degeneration, but may be differentiated by the presence of Argyll-Robertson pupils and a positive Wassermann reaction in the cerebro-spinal fluid and blood. The manner of the onset is sufficient to distinguish the disease from acute myelitis or a spinal tumour. The only other illness of note in which combined posterior and lateral column degeneration occurs is *Friedreich's disease*, from which subacute combined degeneration is easily differentiated by the age incidence and other signs.

**Prognosis.**—There is no evidence that recovery from the disease can take place in any of its stages, though there may be remissions from time to time in the rate of its progress. Those cases in which the development of the early stages is most rapid have usually the shortest time to live, and the onset of severe anæmia precipitates the fatal termination.

**Treatment.**—There is no specific line of treatment. The greatest care should be taken to prevent infection of the bladder, which should not be allowed to become over-distended in the early stages of dysuria, and the patient should be placed on a water mattress as soon as he becomes bedridden with the object of avoiding bedsores. The treatment of the anæmia has been already described (*see* p. 528).

## FRIEDREICH'S ATAXIA

The characteristic feature of this disease is its congenital tendency and its appearance in several members of the same family. Thus a man may transmit it to his children, or it may appear in two or more brothers and sisters without the parents being affected. It affects males only a little more often than females, though males may be especially affected in one family, females in another; and it is generally first noticed at an early age, either about the seventh or eighth year, or at puberty.

**Pathological Anatomy.**—Degeneration is found in the posterior columns in the spino-cerebellar tracts and in the pyramidal tracts (Plate XLIII., Fig. 3). Both sides of the cord are affected symmetrically, and the degeneration is most marked at the mid-thoracic level. The cells and fibre network of Clarke's column show changes in association with the degeneration of the spino-cerebellar fibres. The cerebellum as a rule shows no pathological changes.

The cause of the disease seems to be a congenital want of vitality in the neuron systems or tracts involved.

**Symptoms.**—The onset is as a rule gradual, and the disease is first manifested in a certain clumsiness of the gait and a tendency for the child to fall about when at play or when performing actions requiring a nice balance. These symptoms depend upon inco-ordination, which conforms to the cerebellar type and may be demonstrated by means of the various tests already described (finger-nose-finger and heel-knee tests, diadochokinesis). The arms are affected as a rule rather later than the legs. While at rest the patient may show irregular jerky movements of the limbs somewhat resembling those of chorea, and in particular there is to be noticed a nodding tremor of the head.

The inco-ordination is not confined to the limbs, but also affects the speech musculature, so that the utterance is hesitating and explosive. Nystagmus is usually present, but is not a prominent feature of the illness. The knee and ankle jerks are lost comparatively early by reason of involvement of the posterior columns, and the plantar reflexes are extensor as soon as the pyramidal tracts are affected.

A peculiar feature of the disease is the almost constant presence of certain skeletal deformities. Of these the most characteristic is *pes cavus*, a condition in which the arch of the foot is greatly exaggerated and the toes are hyper-extended at the metatarso-phalangeal, flexed at the interphalangeal, joints. The other is *scoliosis*, or lateral curvature of the spine in the dorsal region, with which is usually associated some degree of *kyphosis* or forward curvature. The causes of these deformities are imperfectly understood.

Sensibility is not as a rule affected to any important extent, but in some cases diminution of both the superficial and deep forms of sensation may be discovered on careful examination. The sphincters are not affected. In patients who show signs of the disease before the age of puberty physical development is usually retarded, and the mentality also remains somewhat childish. The disease is slowly progressive over a number of years, but not directly fatal.

**Diagnosis.**—This has to be made from *juvenile tabes*, in which the common occurrence of Argyll-Robertson pupils, optic atrophy and associated signs of congenital syphilis, together with the Wassermann reaction in blood and spinal fluid, serve to make the differential diagnosis certain. Friedreich's ataxy may also resemble *disseminated sclerosis*, which, however, is seldom fully developed at such an early age, and in which the tendon jerks are exaggerated, not lost. In *cerebellar tumour* the signs of increased intracranial tension are usually evident, but apart from this there are cases of *hereditary cerebellar ataxy* which are distinguished with some difficulty from Friedreich's disease.

**Prognosis.**—This is unfavourable. The patient tends eventually to become bedridden after a period of ten or fifteen years, and as a rule dies comparatively young of some intercurrent affection.

**Treatment** is of little avail, but some improvement may be effected by means of systematic and continued training after the manner of the exercises employed for the treatment of tabes dorsalis.

## PERONEAL MUSCULAR ATROPHY

(*Charcot-Marie-Tooth Disease*)

The main features of this disease are the distribution of the muscular wasting, its slow progress, and the frequency with which a hereditary tendency is manifested by cases occurring in blood relations. As a rule it is transmitted directly to succeeding generations by persons themselves affected. Although isolated cases are reported, careful search often reveals unmistakeable signs of the disease in supposedly unaffected members of the family.

**Pathological Anatomy.**—Degeneration is found in the anterior horn cells corresponding to the wasted muscles, in the peripheral nerves, and in the posterior columns (**3**).

**Symptoms.**—These usually commence in childhood, but in exceptional cases may first appear in middle life. The onset is gradual with wasting and weakness of the small muscles of the feet, and of the anterior tibial group, especially the peronei. This results in characteristic deformity and difficulty in walking. The toes become curled up owing to over-extension at the metatarso-phalangeal and over-flexion at the inter-phalangeal joints; the plantar arch is raised and shortened, producing a *pes cavus*, and the foot is dropped at the ankle joint and inverted. The patient, therefore, has difficulty in clearing his foot from the ground, and is apt to trip over his toes, especially when hurrying.

As the disease progresses the small muscles of the hands are often next affected. The progress of the disease is often extremely slow and may not extend further than this. When it does so the wasting gradually extends up the lower limbs in a somewhat characteristic fashion, affecting first the calves, and then the lower

parts of the thighs. Fibrillary twitchings may occur in the wasting muscles, and the electrical reactions in most cases show R.D.

There may be complaint of numbness and tingling in the feet at the commencement of the disease, but there is no objective sensory disturbance.

Loss of tendon jerks as a rule precedes muscular wasting, so that all the tendon jerks of upper and lower limbs may be abolished at a stage when wasting is confined to the feet. There are, however, exceptions to this rule, and in practice it is found that each family group of these patients, though remaining true to its own type, shows variations from others. The mental condition, special senses, cranial nerves and sphincters are unaffected.

**Diagnosis.**—The differential diagnosis has to be made from Friedreich's disease. In both there are *pes cavus* and absent tendon jerks with a family history of the disease, but in Friedreich's ataxia there are signs of damage to the cerebellar and pyramidal fibres. The absence of such signs in peroneal muscular atrophy, together with the wasting of the anterior tibial group of muscles, as a rule makes the distinction easy.

The distal type of progressive muscular dystrophy is not so easily distinguished. The points in favour of peroneal atrophy are the occurrence of fibrillary twitchings, the early loss of tendon jerks, and reaction of degeneration in the affected muscles.

**Prognosis.**—The course of the disease is so slow that the disability in many cases is slight, and the patient may continue to get about with the aid of special boots and orthopædic apparatus. In some cases, after a period of five to seven years, the affection seems to become spontaneously arrested.

**Treatment.**—Special boots, night splints, and in some cases walking apparatus, are needed to correct deformities. Massage and passive movements are also useful.

## PROGRESSIVE MUSCULAR ATROPHY

This is a chronic disease characterised by wasting of muscles, with weakness consequent thereon, resulting from degeneration of nerve cells in the anterior cornua of the spinal cord. The names *wasting palsy* and *chronic anterior poliomyelitis* have also been used for it, but as it is not an inflammatory lesion, the latter is not suitable. Although progressive muscular atrophy and amyotrophic lateral sclerosis are usually described separately, they are in reality varieties of the same disease, which has been called "motor neuron disease."

**Ætiology.**—Our knowledge of the origin of progressive muscular atrophy is very incomplete. It is a disease of middle life, the age of onset being as a rule over thirty, and it is more common in males than in females. A small proportion of cases appear to be due to syphilis, as evidenced by a positive Wassermann reaction in blood or cerebro-spinal fluid, but in the great majority no cause whatever is to be found for the disease.

**Morbid Anatomy.**—In the ordinary form of progressive muscular atrophy (Duchenne-Aran) changes are found in the anterior cornua of the spinal cord, in the anterior nerve roots and nerve trunks, and in the muscles themselves. The *anterior cornua* are scarcely, if at all, altered in size or shape, thus contrasting with the condition found in acute poliomyelitis; but they are pale, translucent, and almost entirely wanting in the large motor cells or cell bodies of the lower motor neurons. Such of these as remain are smaller than normal, globular in shape, and without processes. At the same time the neuroglial elements are increased. The posterior cornua are always normal. Degeneration of the pyramidal tracts is present in those cases which present the features of amyotrophic lateral sclerosis (*see* p. 802); it is sometimes found even when during life there have been no spastic phenomena. The anterior *nerve roots* are visibly atrophied, being small and grey; but changes in the nerve trunks are not so

obvious, in consequence of the admixture of healthy fibres from the sensory roots. The *muscles* are pale and small. Under the microscope the fibrillæ are found in different degrees degenerated. Some are simply diminished in size; in others the striation is indistinct, or replaced by fatty granules; in others there is a marked longitudinal striation; and others again have undergone a vitreous or waxy change. The intervening connective tissue is increased in quantity.

**Symptoms.**—In the majority of cases (known as the Duchenne-Aran type) the disease begins in the upper extremities, and is seen first as a gradual atrophy of the short muscles of the thumb and little finger, so that the thenar and hypothenar eminences are flattened and disappear. The interossei muscles are also wasted, leading to depressions between the metacarpal bones; and when the atrophy is advanced, the shortening of the extensors, unresisted by the interossei, produces the peculiar claw-like deformity (*main en griffe*) in which the first phalanges are over-extended on the metacarpals, and the middle and terminal phalanges are flexed on the first. Motor weakness accompanies, *pari passu*, the atrophy. These changes may occur in both hands, but often begin in one before the other, so that the hands are affected unequally. Other muscles are then involved; the deltoid is often the next to atrophy, but it may be the muscles of the upper arm, generally the biceps first, or of the forearm when the extensors are affected before the flexors. The trapezius in its lower two-thirds, and the other scapular muscles, may be affected. The disease subsequently spreads to the trunk and neck, and the diaphragm and intercostals are sometimes involved, so as to cause serious difficulties in respiration. In the ordinary form of progressive muscular atrophy the legs are spared until very late; they may then be affected like the arms, or may be rigid, with or without wasting. This rigidity approximates the case to one of amyotrophic lateral sclerosis, and many writers consider the two complaints to be identical. The course of the disease is often exceedingly slow; it may be years before it spreads from the hands to the arms, and years again before other muscles are involved.

The electrical excitability of the muscles fails in proportion to the wasting. Usually faradic and galvanic excitability diminish together, but contractions can still be obtained, except when the wasting of any muscle is extreme. Then a partial form of reaction of degeneration may be found—*i.e.* in the nerves, slightly diminished excitability to both currents; in the muscles, slightly diminished faradic excitability, and increased galvanic excitability, with slow contractions and increase of ACC; or the reactions may fail altogether, that of the galvanic current lasting the longest.

The tendon jerks are sometimes lost in the affected limbs; but when, as commonly occurs, the pyramidal fibres are also affected, the tendon jerks are present, and usually brisker than normal so long as the muscles concerned have power to contract. When the pyramidal fibres are so far affected as to cause spastic rigidity of the lower limbs the knee and ankle jerks are exaggerated and the plantar responses extensor. The abdominal reflexes, however, are usually preserved. A constant feature of this disease is the occurrence of *fibrillary tremors*, which were at one time thought to be pathognomonic; it is now known that they occur in other atrophic conditions. They consist of slight momentary twitchings of a few fibres of the muscle, visible on the surface, painless, though perceptible to the patient, and recurring every two or three minutes. They occur spontaneously, but may be brought out by a tap on the skin over the muscle.

Sensation remains intact, though the patient may complain of a "fluttering" under the skin due to the fibrillation, and the bladder and rectum are normal.

Where progress is very slow and the limbs are the parts chiefly or alone affected, death only occurs from intercurrent diseases, such as phthisis, pneumonia, or

bronchitis. But some cases are fatal through failure of the respiratory muscles, and in others the disease spreads upwards to the medulla oblongata, so that paralysis of the tongue, larynx, and pharynx results, constituting the *progressive bulbar paralysis*, which will be described shortly.

In another type of the disease, the atrophy first attacks the muscles of the legs, then those of the thighs and the glutei, but does not extend any higher.

In a third type (Duchenne's subacute ascending paralysis), the atrophy, which begins in the lower extremities, extends to the muscles of the back and trunk, and then invades the shoulders and arms, finally perhaps ending in a bulbar paralysis, as in the Duchenne-Aran type.

A very rare form, also sometimes familiar, is the *infantile hereditary progressive muscular atrophy* of Werdnig-Hoffman. It begins in early infancy by involving the muscles of the hip girdle, then those of the trunk, and of the shoulder girdle; finally all the muscles are paralysed, except the facial and ocular muscles and the diaphragm. In the limbs the proximal parts are completely paralysed, while the distal parts, fingers and toes, retain some feeble movements. Fibrillary tremors are sometimes present. The deep reflexes, and sometimes the cutaneous, are lost; and the muscles respond only feebly to faradic and galvanic currents. Sensation is generally unaffected, there is no pain or tenderness, and the mental condition is normal. The disease lasts from a few months to four or five years, and generally terminates by broncho-pneumonia.

**Diagnosis.**—Progressive muscular atrophy has to be distinguished from all other diseases accompanied by atrophy of muscles, especially *primary muscular atrophy* and *muscular dystrophy* (*see* Diseases of the Muscles). When the atrophy affects the hand alone, the deformity resembles somewhat the result of lesion of the *ulnar nerve*; but in this last the ulnar half of the hand is more decidedly affected (the radial lumbricales being supplied by the median nerve), and anæsthesia and trophic changes occur; in traumatic cases the history of injury will, of course, help. The presence of a *cervical rib* may cause atrophy of the muscles of the hand, which might be thought to have a spinal origin; but the associated sensory symptoms should prevent errors (*see* p. 757). *Lead paralysis* is recognised by the extensors being first, and generally alone, affected, by the blue line on the gum, the anæmia and punctate basophilia, and perhaps by the occupation, and preceding attacks of colic. *Multiple neuritis* is distinguished by the more rapid onset, the wide extent of the parts affected, the numbness or anæsthesia, and the tenderness of muscles. As contrasted with other diseases of the spinal cord, the important feature of progressive muscular atrophy is the slow commencement of atrophy and weakness together, without pain, spasm, or sensory troubles. This distinguishes it from *tumour* and *meningitis*, which may cause muscular atrophy. In *acute poliomyelitis* the history is quite different. In the typical cases of *amyotrophic lateral sclerosis* the course is more rapid and the reflexes are rapidly increased. The Wassermann reaction in blood and spinal fluid serves to differentiate those cases which are due to a degenerative process of syphilitic origin.

**Prognosis.**—This is unfavourable except in the syphilitic cases. The disease is steadily progressive, but life is not endangered unless the degenerative process extends to the medulla.

**Treatment.**—Drugs have but little value; arsenic and strychnia have seemed to do good sometimes. General hygienic treatment should be pursued: good air, nutritious food, exercise without strain, and freedom from mental worry. In addition to this, the muscles may be locally treated with electricity, massage, and passive movements; but the improvement to be obtained by these means is at best but very slight.

In the syphilitic cases specific treatment should be undertaken from the outset in the hope of arresting the disease. In some cases excellent results may be obtained.

## AMYOTROPHIC LATERAL SCLEROSIS

In this disease there is degeneration of the motor cells of the anterior cornua of the spinal cord, with sclerosis of the pyramidal tracts in the lateral columns (Plate XLIII., Fig. 1). It thus combines at the same time the lesions of progressive muscular atrophy and of spastic paraplegia—that is, lesions of both upper and lower motor neurons. But the lesions are not confined to the spinal cord; the motor centres of the bulb (especially the hypoglossal and vago-accessory nuclei) are also affected towards the end of the disease in the majority of cases; and the atrophy of the upper neurons in prolonged cases extends through the medulla oblongata, crura cerebri, and internal capsules to the motor cells in the cortex of the brain. Atrophy and sclerosis also affect some of the association fibres of the antero-lateral columns near the grey cornua. It is held by some that progressive muscular atrophy and amyotrophic lateral sclerosis are the same disease.

**Ætiology.**—It occurs between the ages of twenty-five and fifty, is more frequent in females than in males (Charcot), but cannot generally be referred to any particular cause.

**Symptoms.**—The first symptom is weakness in the upper extremities, which are soon seen to be affected with wasting. This is not limited to the interossei, or other muscles of the hand, though it may begin in them, but affects the whole upper extremity much more equally (according to Charcot) than in progressive muscular atrophy. Fibrillary tremors often occur, and the electrical reactions, as in progressive muscular atrophy, show only a simple diminution, unless the wasting is extreme, when reaction of degeneration may be present. Quite early in the history the tendon jerks are increased, and can be elicited on striking the tendons of the biceps and triceps, or the lower ends of the radius and ulna. After a time rigidity takes place in the atrophied muscles, and considerable contractures may result. Charcot especially noted a deformity which he regarded as characteristic of amyotrophic lateral sclerosis: the upper arm lies close along the body, the forearm is semiflexed and pronated, whilst the wrist is strongly flexed, and the fingers are bent into the palm. Generally, after from eight to twelve months, the lower extremities become involved, presenting at first the characteristics of spastic paraplegia. Weakness and rigidity appear together, the former being masked by the latter. The knee jerk is increased, and ankle clonus can be obtained; the plantar reflexes are extensor; the abdominal reflexes are diminished or lost; the electrical excitability remains; and walking can be accomplished, though with difficulty. After some time wasting also occurs in the lower extremities, but it is never so complete as in the upper. Sensation and the sphincters are unaffected. As the disease spreads to the bulb the tongue, lips, palate, and laryngeal muscles are paralysed; deglutition and speech are rendered difficult, and the characteristic features of palatal paralysis or laryngeal paralysis may be present. The facial muscles are also atrophied, and in later stages rigidity ensues, with increased jaw jerk or masseter clonus. With the extension to the cerebrum the emotional faculties are disturbed, and the patient laughs or cries without good cause.

The duration is from one to four or more years, and is shortest in those cases in which the bulbar symptoms develop early. Death results from asphyxia, inanition, or more often from pneumonia, caused by inhalation of food particles through the larynx.

**Diagnosis.**—*Progressive muscular atrophy* (Duchenne-Aran type) has a slower course, with no excess of tendon reflexes or muscular rigidity. *Primary spastic paraplegia* begins generally in the lower extremities, and is unaccompanied by atrophy.

**Treatment** may be tried on the same lines as in these two diseases, but the prognosis is very unfavourable.

## LANDRY'S PARALYSIS

In 1859 Landry described cases of paralysis commencing in the lower extremities, rapidly ascending, and soon fatal, for which no pathological cause could be found on examination. Cases of the kind still occur in which the coarser lesions of myelitis and softening are entirely absent.

**Ætiology.**—The disease affects males more than females, and is most frequent between the ages of twenty and forty. It has occurred after exposure to cold, in convalescence from acute diseases, and in patients addicted to alcohol; and a few cases have been recorded after cystitis or other forms of urinary sepsis. In the majority of instances, however, no adequate predisposing causes are to be found.

**Pathology.**—In some rapidly fatal cases the spinal cord, nerves, and muscles have been found completely free from disease. In others there have been varying degrees of degenerative change (chromatolysis and displacement of the nucleus) in the cells of the spinal cord, especially those of the anterior cornua, and of Clarke's columns, with more or less vascular engorgement, but no hæmorrhage or perivascular infiltration of leucocytes. There may be changes in the myelin of the anterior roots and of the white columns of the cord, but none in the peripheral nerves. Occasionally, when the fatal termination has been delayed, the Marchi method reveals the presence of degenerative changes in the peripheral nerves, which are probably secondary to those in the anterior horn cells. It is clear from the above account that the histological picture in Landry's paralysis is quite distinct from that of an ascending myelitis, peripheral neuritis or anterior poliomyelitis. Micro-organisms, but not always the same, have been found by different observers in the nerves, spinal cord, meninges, and blood.

**Symptoms.**—In some cases there are premonitory symptoms, such as malaise, pain in the head and back, and numbness and tingling, but usually the disease begins with weakness in the legs, often one before the other. This soon increases to definite paralysis, and invades successively, and within a few days (or in acute cases in a few hours), the thighs, trunk, abdomen and arms; and these, like the legs, are not always affected simultaneously. The diaphragm and the muscles of the neck, of the palate, and those subserving articulation are subsequently paralysed. Very rarely other cranial nerves are affected; thus diplopia, paralysis of accommodation, dilatation of one pupil, and facial paralysis have been noticed. As a rule cutaneous sensibility is normal, though there may be some loss to light touch in the peripheral areas of the extremities. The sensations of passive movement and position are unaffected. In rapidly fatal cases the muscles have not wasted, and the electrical reactions have appeared to be normal; but in some cases of longer duration both atrophy of muscles and modifications in electrical properties have been observed. Control over the urinary and rectal sphincters is not lost, and there is no tendency to bedsores; the cerebral functions are perfect, and there is no fear except in a few cases at the very onset. The tendon jerks and skin reflexes are lost as the corresponding muscles become paralysed.

**Diagnosis.**—This has to be made from acute ascending *myelitis*, from infective *poliomyelitis*, and from *multiple neuritis*. The first of these is distinguished by the pronounced loss of sensation, and the early implication of the bladder. In poliomyelitis there is more general disturbance—fever, headache, pains, perhaps convulsions—and the paralysis is rarely quite uniform or symmetrical. In multiple neuritis symptoms come on gradually, paralysis appears in the peripheral parts of the arms and legs almost at the same time, and the nerves and muscles are tender.

The mortality is high—*e.g.* 58 per cent. in cases collected by Ross; these, however, probably included some cases of peripheral neuritis. The duration

of the disease is from two days to two or three weeks in fatal cases, and death occurs mostly from paralysis of the diaphragm and intercostal muscles. On the other hand, the symptoms persist from two to six or seven months in cases which recover; but recovery is generally complete.

**Treatment.**—This may be the same as that of multiple neuritis or acute myelitis.

## SENILE PARAPLEGIA

In people of advanced age walking may become slow and difficult from weakness of the lower extremities. The gait is rather shuffling, and the feet are dragged, difficulty is found in going upstairs or getting into a carriage, and undue fatigue is experienced after any exercise. The conditions may develop rapidly, and may go on to some stiffness and even contracture. It may be accompanied by slight sensory symptoms, pain or numbness, by impairment of the vesical sphincter, by senile trembling of the hands or head, by failing mental power, and by evidences of arterio-sclerosis.

Pathological evidence as to the true nature of these cases is lacking. It is probable that under the heading of senile paraplegia many diseases of different pathology have been included, that in some cases the disability has been due to diffuse degeneration of the pyramidal tracts, in others to the neuritis (crural and sciatic) which is often associated in old people with osteo-arthritis, in others to undetected spinal compression, and in others again to the weakness and rigidity of that type of Paralysis Agitans in which tremor is an inconspicuous feature.

## SPINAL MENINGITIS

The spinal membranes may be affected separately, but it is common for inflammation beginning in one to spread to the other. Inflammation of the pia mater is called *leptomeningitis*, that of the dura mater *pachymeningitis*. The forms of inflammation commonly recognised are—first, an inflammation of the outer surface of the dura mater, usually set up by disease in the neighbourhood: this is called *meningitis externa* or *pachymeningitis externa;* secondly, inflammation of the internal surface of the dura mater, generally chronic and resulting in great thickening of the membranes (*pachymeningitis interna hypertrophica*), or in extravasation of blood, and the formation of fibrinous, pigmented deposits (*pachymeningitis interna hæmorrhagica*); lastly, inflammation, beginning in the pia mater, or *leptomeningitis*, which may be acute or chronic. It will be seen that the symptoms of all these forms present much similarity, as they depend not so much on any alteration in the membrane itself as upon the implication of the nerve roots which pass through them, and of the cord which they enclose.

### ACUTE LEPTOMENINGITIS—ACUTE SPINAL MENINGITIS

**Ætiology.**—Its causes are often obscure, but amongst those to which it can be traced are exposure to injuries, such as fractures and dislocation of the spine, and various forms of infection. Thus it has sometimes arisen in connection with pneumonia, scarlatina, typhoid, septicæmia, or the puerperal state. A tuberculous spinal meningitis not infrequently accompanies tuberculous meningitis of the brain, and the cerebral and spinal meninges are both involved in the disease known as epidemic cerebro-spinal meningitis or cerebro-spinal fever (*see* p. 93). Inflammation may spread from outside the spinal canal, first causing an external meningitis, or from the cerebral to the spinal membranes. It has also followed puncture of a spina bifida, doubtless from local infection.

**Morbid Anatomy.**—In the early stage the pia mater is reddened from increased vascularity, and small hæmorrhages may be present; an exudation then takes place in the meshes of the pia mater and upon the surface, at first grey in colour, subsequently purulent and yellow or greenish yellow. This

deposit is seen often over a large extent of the cord in irregular patches, more upon the posterior than the anterior aspect from the effects of gravitation. The spinal fluid is turbid and opaque. The inflammation affects also the inner surface of the dura mater, and the two membranes may be united by the viscid purulent lymph. The process frequently extends into the cord (*meningo-myelitis*), affecting specially its periphery, in which dilated vessels and extravasated leucocytes occur, and the nerve roots are similarly involved. Bacteria have been found in several cases, especially pyococci, *Diplococcus pneumoniæ*, and the meningococcus.

**Symptoms.**—An attack of acute spinal meningitis usually begins with rigor and elevation of temperature; there is severe pain in the back, which may be localised, or extend along the whole length, and this pain is usually increased by movement as well as by pressure. In addition, there are paroxysmal pains of shooting, darting character, radiating in the course of the nerves arising from the part, and hyperæsthesia of the skin, even of the muscles, in the same areas. Irritation of the nerve roots leads to spasm of the muscles, producing rigidity of the spine, with more or less severe arching, or *opisthotonus*. This may be present in the whole length of the spine, or affect the neck alone, when the occiput is fixed back between the shoulders (retraction of the head, cervical opisthotonus). The abdominal muscles and the muscles of the legs are also affected by spasm, and the spasms are increased by attempts at movement. The reflexes are generally increased, and Kernig's sign may be observed. The bowels are constipated, and the urine is retained by spasm of the sphincter, or later by paralysis of the detrusor. There are the usual accompaniments of pyrexia; the temperature is generally raised, the pulse is quick, and the *tache méningitique*, which is the same as the *tache cérébrale* (*see* p. 130), is well marked. After some time—it may be a few days or longer—the symptoms of irritation, pain, and spasm give place to those of paralysis, anæsthesia, and diminished reflexes; and the case then approximates to one of myelitis, and is either rapidly fatal from exhaustion, paralysis of the respiratory muscles, or acute bedsores, or lapses into a more chronic condition, with atrophy and shortening of muscles. Some patients recover after several months; others die from bedsores, or vesical, renal, or other visceral complications.

The symptoms of meningitis vary considerably, and are not always so manifest as above indicated. The signs of irritation may be of very short duration, and those of paralysis become prominent quite early; and in cases where the cerebral membranes are involved at the same time the spinal symptoms may be entirely masked, as is frequently the case in tuberculous meningitis. In cerebro-spinal fever the symptoms of the two conditions are combined. Further, differences are observable according as the inflammation affects mainly the cervical, the thoracic, or the lumbar region. It is sufficient here to point out that in cervical meningitis retraction of the head, dyspnœa from implication of the diaphragm, dysphagia, inequality of the pupils, and great elevation of temperature are likely to occur; while in dorsal meningitis the trunk muscles, and in lumbar meningitis the muscles of the loins and legs, are especially affected. The duration varies from two or three days to three weeks in the more acute cases, and is occasionally much longer.

**Diagnosis.**—Meningitis has mainly to be distinguished from myelitis and from tetanus. As contrasted with meningitis, there is less fever in *myelitis*, the local and radiating pain, rigidity, and spasms are less marked or absent, and paralysis and anæsthesia come on quite early. *Tetanus* is distinguished by the persistent spasm, aggravated on the slightest peripheral irritation, by its commencement in trismus, by a slight pyrexia, if any, and by the history of wound in the majority of cases. Rheumatism of the lumbar muscles or of the vertebral articulations may to a certain extent simulate meningitis. The diagnosis both of the existence and nature of meningitis may as a rule be settled by means of

the operation of *lumbar puncture*, followed by microscopical and bacteriological examinations of the fluid withdrawn (*see* p. 737).

**Treatment.**—In the acute stage the treatment is similar to that of acute myelitis. Perfect rest and avoidance of all external irritations should be ensured; while morphia, chloral, and potassium bromide or chloroform inhalations should be given to relieve pain. Lumbar puncture should be performed daily during the acute stage, and the cerebro-spinal fluid allowed to drain until it appears to run out under normal pressure. If the meningococcus be identified as the causal agent, intrathecal injections of anti-serum are indicated (*see* p. 97), whilst in streptococcal cases intravenous or subcutaneous injections of anti-streptococcal serum should be employed with suitable precautions against anaphylactic shock (*see* p. 17).

## CHRONIC LEPTOMENINGITIS

**Ætiology.**—This may be only the continuation of an acute process; or it may be chronic from the first, and is then in most cases attributable to syphilis. It is commonly found in association with inflammatory (or degenerative) processes in the cord itself, such as chronic myelitis; and it may also result from lesions external to the cord.

There is another group of cases, in which the condition follows an injury to the spine. In many instances, however, which appear to be due neither to syphilis nor trauma, and in which such degeneration as may occur in the spinal cord seems to be secondary to compression by the meninges, no satisfactory cause is to be found.

**Anatomy.**—The condition is one of fibrous thickening of the pia mater, with dilatation and thickening of the walls of the blood vessels; in syphilitic cases the characteristic gummatous deposits or gelatinous exudations are present. The change may be extensive when it follows upon an acute lesion, more limited and scattered when it is chronic from the first. The parts affected are in some cases determined by the pre-existing lesion of the cord. The pia mater and arachnoid may be adherent to the dura mater; the cord itself often shows inflammatory changes in the connective tissue at the surface, or a more extensive myelitis; and the nerve-roots may be compressed and atrophied.

In the condition known as *meningitis serosa circumscripta* (**4**), which may follow an injury to the spine, with or without involvement of the cord, or may appear spontaneously in the absence of a traumatic history, the adhesions between pia arachnoid and dura may lead to cyst-like collections of fluid which compress the spinal cord.

**Symptoms.**—These are the same in kind as those of acute meningitis—namely, symptoms due to local irritation of the membranes, others due to implication of the nerve roots, and others again which result from implication of the cord; but they are gradual in their onset, they are not accompanied by fever, the muscular spasms and rigidity are much less marked than in the acute form, the sensory symptoms are often more localised, and paralysis may occur early. Ultimately the case may assume all the features of chronic myelitis or compression of the spinal cord (*see* p. 768).

**Treatment.**—Specific treatment is indicated in the syphilitic cases, which may be distinguished by means of the Wassermann reaction in blood and cerebro-spinal fluid. The condition of *meningitis serosa circumscripta* may be greatly relieved by laminectomy and incision of the membranes with release of the localised collections of fluid.

## INTERNAL PACHYMENINGITIS

It has been already stated that the inner surface of the dura mater is often involved in both acute and chronic leptomeningitis; but there are two forms of

pachymeningitis which require separate mention—namely, *pachymeningitis interna hypertrophica* and *pachymeningitis interna hæmorrhagica*. In the former, which is probably in most cases due to syphilis, the dura mater becomes immensely thickened, so as to cause very severe compression of the cord and nerve roots.

**Symptoms.**—Charcot has described it as especially affecting the cervical region (hypertrophic cervical pachymeningitis). After a first stage of shooting pains in the back of the neck, shoulder, arms, and upper part of the thorax, with muscular twitchings and spasms, there are gradually developed anæsthesia, paralysis, and atrophy, and loss of electrical reactions in the distribution of the compressed nerve roots. Charcot observed that the median and ulnar nerves were more affected than the musculo-spiral, and that consequently there arose a deformity of the upper limb, characterised by extension at the wrist and metacarpo-phalangeal joints, and by flexion of the phalanges. This appears to be due to implication of the lower part of the cervical region. In a case affecting the upper part of the cervical region the muscles supplied by the musculo-spiral were paralysed, and a condition of extreme flexion was the result. As the compression of the cord increases, paraplegia, secondary degenerations, and spastic rigidity of the paralysed parts occur. In rare cases, hypertrophic pachymeningitis affects the lumbar region, or the cauda equina, when the pains, paralysis, and atrophy are situate in the lower extremities, and the sphincters are involved early (*see* p. 766).

**Treatment.**—A vigorous antisyphilitic treatment should be employed.

In *pachymeningitis interna hæmorrhagica*, or *hæmatoma of the dura mater*, the inner surface of the membrane is covered with a reddish-brown exudation, consisting of fibrin, connective tissue, recently extravasated blood, and, it may be, cysts containing blood in different stages of change. This form of meningitis affects the greater part of the cord, and is associated mostly with a similar condition in the cerebral dura mater (*see* p. 847). The disease has been attributed to the abuse of alcohol, and it is seen in some cases of mental disorder.

The **Symptoms** are those of a slight chronic meningitis, but they are often masked by others which result from the cerebral lesion.

### EXTERNAL PACHYMENINGITIS

As already stated, this is mostly secondary, and arises from any inflammation in the neighbourhood of the dura mater: thus deep bedsores over the sacrum may slough into the spinal canal, or the dura mater may be inflamed by caries of the spine (the most common cause), by retropharyngeal abscess, or abscess in other situations, by cellulitis of the neck, or inflammation of the subpleural tissue; or it may result more directly from injury. It may be acute or chronic. In the acute form, such as occurs from sloughing bedsores, the external surface of the dura mater is reddened, or presents lymph upon the surface, or is covered with a layer of pus. In caries of the spine the process is generally more chronic, and the outer surface of the membrane is covered with caseous or semi-caseous deposit, and the membrane is itself thickened. More or less inflammation or compression of the nerve roots commonly co-exists, and the cord may be much narrowed. In the purulent cases especially the process may extend to the external surface.

**Symptoms.**—These are not essentially different from those already described under Leptomeningitis. They are pain in the back at the level of the lesion, stiffness of the back from rigidity of the muscles, and pain on movement, pains in parts corresponding to the nerves arising from the region affected, hyperæsthesia, jerking and tension of the muscles, and later the symptoms of compression of the cord—namely, paralysis and anæsthesia, in varying degrees, of the parts below the seat of the lesion. In acute cases the symptoms of irritation are likely to predominate, in chronic cases those of compression.

**Diagnosis.**—The important point is the recognition of an external cause, otherwise it may be impossible to distinguish it from other forms of spinal meningitis.

The **Prognosis** is unfavourable in acute cases, but more hopeful in caries of the spine, of which a large number of cases make a more or less perfect recovery.

**Treatment.**—The chief indication is to remove the original cause, if possible; the others, to aid in the absorption of inflammatory products and to treat the myelitis resulting from compression.

## SPINAL MENINGEAL HÆMORRHAGE

This is a rare occurrence. It arises from injuries, such as blows, stabs, falls on the feet or back, and traction on the spine of new-born children during delivery. Blood effused into the cranial cavity sometimes runs down into the spinal canal, and very rarely an aneurysm of the aorta or of the vertebral artery has burst into it. Hæmorrhage into the membranes may form part of purpura and scurvy, or of other general conditions in which hæmorrhage occurs, such as alcoholism; and in this connection it has already been mentioned as part of pachymeningitis hæmorrhagica.

**Symptoms.**—Like those of hæmorrhage into the cord itself, the symptoms are distinguished by the suddenness of their onset. There are severe pains in the back, pains radiating along the nerves, and spasms and rigidity of the muscles supplied by the nerves. Subsequently there is loss of power and sensation, and in some cases complete paralysis and anæsthesia occur. The paralytic symptoms follow quickly upon the signs of irritation, and reach their height in a period varying from a few hours to a few days. Death also may happen in a few hours, or symptoms characteristic of meningitis may supervene.

**Diagnosis.**—Spinal meningeal hæmorrhage is distinguished from *hæmorrhage into the cord* chiefly by the fact that signs of irritation, such as muscular cramps, spasms, rigidity and hyperæsthesia, precede paralysis, and the paralysis is less complete. *Spinal meningitis* is more gradual in its onset, and is accompanied by fever from the first. A lumbar puncture may give valuable information.

**Prognosis.**—Many cases are fatal; but recovery is more frequent than in hæmorrhage into the cord itself.

**Treatment.**—This must be conducted in the same way as that of intramedullary hæmorrhage. The later treatment is that of spinal meningitis.

# DISEASES OF THE MEDULLA OBLONGATA

The medulla oblongata, or bulb, is subject to similar diseases with other parts of the central nervous system, such as hæmorrhage, inflammation (bulbar myelitis), and tumours. The symptoms are determined by the anatomical structure of the medulla oblongata, which, besides transmitting the motor and sensory tracts, contains the special nerve centres of the lower cranial nerves, from the fifth to the twelfth. Hence, on the one hand, there may be paralysis of the trunk and limbs; on the other, impairment of the functions of phonation, articulation, mastication, and deglutition. These symptoms make up what is commonly called "bulbar paralysis"; and it will be best to describe first a chronic form known as progressive bulbar paralysis, and subsequently the more acute results of hæmorrhage and embolism, and the effects of the growth of tumours.

## PROGRESSIVE BULBAR PARALYSIS

*(Labio-glosso-laryngeal Paralysis)*

In this disease there is a slowly developed paralysis of the lips, tongue, larynx, and pharynx, resulting from degeneration of the nuclei of the nerves which supply the muscles of these parts.

**Ætiology.**—It occurs in middle and advanced life, between the ages of thirty and seventy, and is more frequent in men than in women. The true ætiology is unknown. Bulbar paralysis is intimately related to progressive muscular atrophy and amyotrophic lateral sclerosis, occurring often as the last stage of either of those diseases; and sometimes an illness which has begun as bulbar paralysis has at a later date affected the spinal centres.

**Pathology.**—To the naked eye the medulla oblongata may show but little, or there may be some want of symmetry, or slight shrinking, or on section discoloration or blurring of the outlines. Changes are more obvious in the nerve roots proceeding from the medulla; those of the hypoglossal and facial, the vagus and accessorius, are grey in colour and shrunken, and the microscope shows that a number of fibres are atrophied and degenerated. Microscopic examination of the corresponding nuclei reveals degeneration and atrophy, or complete disappearance of the nerve cells, some increase of the neuroglia, and thickening of the vessel walls. These changes are most marked in the hypoglossal nucleus and the lower part of the facial nucleus, and then in the vago-accessorius nucleus. Less commonly the glosso-pharyngeal nucleus may be affected, and rarely the nucleus of the sixth nerve, and that of the motor division of the fifth. Degeneration of the pyramidal fibres is usually present, even though there may have been no signs during life to indicate this.

The atrophied muscles present appearances identical with those seen in progressive muscular atrophy.

**Symptoms.**—The disease is generally first evident in the movements of the *tongue*, and the articulation of sounds which depends upon it becomes faulty. These are first of all *e*, and then *s*, *l*, *k*, *g*, *t*, *d*, *n*, *r*, and *sh*. The paralysis increases, and may become so complete that the tongue cannot be protruded, but lies always at the bottom of the mouth. After a time atrophy takes place, and the organ becomes wrinkled and furrowed. Fibrillary contractions are often observed in it. Shortly after the tongue begins to be paralysed the same change occurs in the *lips*. The articulation of *o*, *u*, *p*, *f*, *b*, *m*, and *v* is impaired, and whistling, blowing, and pouting are performed with difficulty. The lower lip drops away from the teeth, the naso-labial folds are more marked, and saliva dribbles from the angles of the mouth. Atrophy here can also be detected in the lips becoming thinner, and fibrillary contractions may be seen. Food also collects between the teeth and the cheek; but the paralysis of the facial nerve is confined to the lower half of the face, the upper half remaining entirely free. After the tongue and lips the *palate* is paralysed, and as a result liquids may regurgitate through the nose, and the voice acquires a nasal quality. The articulation of *o* and *p* is also impaired by this, since the volume of air which is required for their production is diminished by its escape into the nasal cavity. Paralysis of the *larynx* produces hoarseness, and, finally, complete aphonia; and during swallowing food is apt to enter the larynx from the paralysis of the tongue and certain muscles of the larynx (arytænoideus, thyro-ary-epiglottideus, and thyro-arytænoideus externus), by the combined action of which the two passages should be cut off from one another. As a result choking takes place, and small particles are inhaled, which may set up bronchitis or lobular pneumonia. The laryngeal paralysis also renders coughing and hawking difficult or impossible. Dysphagia is further aggravated by paralysis of the *pharyngeal* muscles.

The progress of the disease is very slow, but the condition of the patient in an advanced stage is highly characteristic. The lower lip falls; from the angles of

the mouth dribbles saliva, which the patient is constantly wiping away with a pocket-handkerchief; the tongue cannot be protruded, and the only sound uttered by the patient is a hoarse grunt as the air is forcibly driven through the flaccid glottis. With all this, the intelligence and memory, appetite and digestion, the functions of the bladder and rectum, are perfect, and, as a rule, the special senses, the movements of the eyes, the sensibility of the skin of the face and mucous membrane of the mouth, and motor and sensory power in the limbs are unaffected. Only in rare cases does the disease extend to higher centres, so as to produce deafness or ocular paralysis, or to affect the sensation of the face; and if motion of the limbs is affected, it is by the lesion of progressive muscular atrophy or amyotrophic lateral sclerosis. The electrical excitability of the affected muscles is, in part at least, retained, but in advanced cases is much diminished. Erb says that the muscles of the chin, lips, and even the tongue, show a marked reaction of degeneration, while the electric irritability of the nerves is normal or but slightly diminished. The reflexes are generally diminished, so that the palate, pharynx, or larynx may be irritated without exciting retching, vomiting, or coughing; but they sometimes persist till late in the disease, and Erb describes reflex contractions in the muscles of the chin and lips. Fever is absent, vasomotor disturbances are not necessarily present, nor has glycosuria or albuminuria been observed as part of the disease. Occasionally, towards the end, the pulse becomes very rapid (140 to 160). Death takes place by exhaustion from inanition, by choking, by dyspnœa or sudden cardiac failure, or by bronchitis, pneumonia, or gangrene set up by the inhalation of particles of food.

**Diagnosis.**—This is generally quite easy, from the chronic course and the limitation to the bulbar nerves specified. Tumours growing in or compressing the medulla are mostly accompanied by other symptoms, such as headache, noises in the ears, deafness, sickness, or convulsions. Bilateral lesions situated more centrally (*i.e.* in the motor tracts nearer the cortex of the brain) may cause paralysis of the same nerves, but the symptoms on the two sides may not run parallel, and there will be no atrophy, electrical changes, or loss of reflexes, showing that the nerve nuclei are intact; further, the limbs will probably be paralysed, and the mental functions seriously impaired.

**Prognosis.**—This is absolutely unfavourable, and the duration is rarely more than three years.

**Treatment.**—Drugs are of little value, but antisyphilitic remedies should be tried when there are syphilitic antecedents or a positive Wassermann reaction. The important thing is to secure proper nutrition, and to prevent the inhalation of particles into the lungs. It may become necessary to feed the patient by an india-rubber tube, which he can himself pass down the pharynx into the stomach, and liquid food can then be poured into a funnel connected with the free end of the tube. Sometimes, in the early stage, solid food in large boluses can be more easily swallowed than liquid, being less liable to pass into the larynx or nares. Atropine has been given to lessen salivation. The general health of the patient must be, as far as possible, maintained.

## ACUTE BULBAR PARALYSIS

Contrasting with the chronic progressive form, there occur occasionally cases in which the symptoms of bulbar paralysis come on suddenly, or at least rapidly, as a result of *hæmorrhage*, *embolism*, or *acute inflammation* of the medulla oblongata.

**Ætiology.**—Bulbar hæmorrhage is more frequent than spinal hæmorrhage, less so than cerebral hæmorrhage. It occurs in the same circumstances as the latter. The same may be said of embolism or thrombosis of the arteries of the medulla oblongata; here, however, the distribution of the vessels becomes of

interest, since, according to Duret, the nuclei of the hypoglossal and accessory nerves are supplied by the anterior spinal and vertebral arteries, those of the vagus, glosso-pharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries, and the nuclei of the facial, the trigeminal, and the three oculo-motor nerves by branches of the basilar. These anatomical associations may help to distinguish vascular obstruction from hæmorrhage, otherwise not always easy to discriminate. Cases of an inflammatory nature are closely related to acute encephalitis (*see* p. 829) and to infective poliomyelitis (*see* p. 97), and may be due to the same virus.

From progressive bulbar paralysis this form differs not only in the rapidity of onset, but in the greater frequency of premonitory indications, in the irregularity of the symptoms, and in the accompanying paralysis of the limbs, since the lesions are not necessarily restricted to motor nerve nuclei, as in the "progressive" cases, but are more or less indiscriminate, affecting the motor and sensory tracts as well.

Thus with paralysis of the tongue, difficulty of articulation, and inability to swallow, there may be paralysis of all four limbs. The occurrence of the lesion on one side will produce a more or less unilateral distribution of the symptoms. On the other hand, crossed paralysis may take place, for instance paralysis of the arm on one side, and of the leg on the opposite, from a lesion in one-half of the medulla affecting the lowest pyramidal fibres going to the opposite limb just before their decussation and the highest fibres coming to the same side just after their decussation. Or a hæmorrhage situated higher in the medulla may damage the facial nerve fibres or nucleus on the same side, and the pyramidal tract of the same side, before its decussation to the opposite, producing a crossed hemiplegia such as results from lesions of the pons Varolii (*see* p. 828). Sometimes also there is severe respiratory disturbance, or rapid and irregular pulse, or vaso-motor derangement, shown by rise of temperature. Albumin and sugar have been noticed in the urine. There may, however, be little time for the observation of such symptoms, as, especially in hæmorrhage, the patient may fall down suddenly, with or without a cry, and death may take place at once. In other cases there are headache, vomiting, noises in the ears, and epileptiform convulsions. In fatal cases the temperature sometimes rises to 107° F. or higher.

Acute bulbar myelitis is usually less rapid in its occurrence than the other lesions, hæmorrhage, and embolism; vertigo, headaches, and pain in the muscles of the back, may precede the more obvious bulbar symptoms. The limbs may be paralysed from implication of the pyramidal tracts. The temperature is sometimes raised, and the pulse is mostly rapid. Death takes place in from four days to two or three weeks.

**Treatment.**—This must be similar to that of the same lesions in the brain and spinal cord.

## PSEUDO-BULBAR PARALYSIS

This condition may be briefly mentioned here, though it belongs more properly to diseases of the brain. The term has been used to designate cases in which a succession of small vascular lesions on opposite sides of the brain has given rise to weakness or paralysis of the muscles innervated from the medulla. As already mentioned, the labio-glosso-pharyngeal group of muscles is not affected in an ordinary hemiplegia for the reason that their upper motor neuron supply comes from both hemispheres, and the fibres from one side of the brain alone appear to be capable of maintaining functional efficiency in the muscles of both sides. When, however, a vascular lesion producing hemiplegia is followed by another on the opposite side of the brain, the upper motor neuron supply to this group of muscles may be partially or completely cut off.

The resulting symptom picture simulates that already described as occurring in

progressive bulbar paralysis, but there is no atrophy of the muscles, some of the signs of hemiplegia may usually be found on one or both sides of the body, and a history is generally obtained of two or more slight "strokes." The age incidence of pseudo-bulbar paralysis is also later than that of progressive bulbar palsy, and signs of arterial degeneration are commonly found in association with the condition. Moreover, in pseudo-bulbar paralysis the mental condition is nearly always affected to a far greater degree than in the other disease.

### COMPRESSION AND TUMOURS OF THE MEDULLA OBLONGATA

The medulla oblongata may be slowly compressed as a result of caries of the occipital bone or of alterations of its shape, by enlargement of the odontoid process, by tumours such as gumma of the dura mater, growths on the choroid plexus, aneurysms on the vertebral or basilar arteries, and lastly, perhaps most frequently, by tumours of the cerebellum.

The rare occurrence of disease and rupture of the transverse ligament is followed by sudden and fatal compression of the medulla by the odontoid process. Tumours in the medulla oblongata are comparatively rare; they include tuberculous masses, glioma, glio-sarcoma, myxoma, and fibroma.

**Symptoms.**—In compression, the symptoms characteristic of bulbar lesions may be preceded by those of irritation, such as pain in the distribution of the fifth nerve, and twitchings in muscles supplied by the facial. Convulsions, vomiting, hiccough, and dizziness may also be present, and later the special lesions of the bulbar nerves, and probably weakness in the limbs. The symptoms may begin on one side and spread to the other. Tumours in the substance of the medulla are not accompanied by irritative symptoms; but headache, vomiting, and convulsions may occur.

## DISEASES OF THE BRAIN

What has been said of the nervous system in general is equally true of the brain —namely, that the symptoms of disease are largely determined by the locality of the lesions, and to a much less extent by their nature. A further knowledge, therefore, of the localisation of the functions of the brain is eminently desirable in the study of its diseases, and some remarks on this subject, with an account of the result of lesions, of whatever kind they may be, upon the different parts of the brain, must precede the description of the diseases which produce them. At the same time it must be recognised that, though the symptoms of a purely destructive lesion correspond accurately to the area destroyed, a tumour will, as it grows, press first upon adjacent parts, and ultimately upon every part of the encephalon, and hence the symptoms may be in excess of those which correspond to the primary seat of the lesion. Thus cerebellar tumours often press upon the motor tracts in the bulb, and occipital tumours upon the cerebellum. An irritative lesion also will produce effects which may on some occasions correspond to the actual site of the lesion, and on others will be such as to show that the irritation has spread to adjacent parts. Thus convulsions started by a lesion in the arm centre will sometimes spread to the leg of the same side, or even to the limbs of the opposite side.

A further point of importance is that a large proportion of the cerebral substance is given over to the provision of neuronic pathways for the performance of reactions individually acquired, and therefore to some extent variable. Destructive lesions of these parts may give rise to changes in the personality as a whole which can be measured by no standard methods of examination and are more readily detected by the patient's friends than by the physician.

## LOCALISATION OF FUNCTIONS AND EFFECTS OF LESIONS

**Motor Centres and Tract.**—The greatest importance and interest attaches to the position of the motor centres. The cortical areas responsible for voluntary movements, as mapped out by experimental stimulation, have already been described. These experimental results are confirmed by pathological evidence, certain (irritative) lesions causing convulsions, and other (destructive) lesions causing paralysis, in the corresponding parts. In accordance with this is the fact that the Betz cells, the largest pyramidal nerve cells (upper motor neurons), are found exclusively in the grey matter of this area. Sherrington and Leyton in their researches upon the brains of anthropoid apes have found that even within this area the localisation of function is to some extent variable, and that the form of the response obtained from stimulation of the motor area depends not only upon the exact position of the spot stimulated, but also upon the state of activity of the grey matter in the immediate neighbourhood. Stimulation, for instance, of adjacent spots, A, B, C, D, in this order, may result in a movement of flexion of the fingers at C, but if the order of stimulation be changed to D, A, C, B, a movement of a different order, say extension of the index finger, may be obtained from spot C. The chart constructed by these observers to illustrate the distribution of the various motor centres in the pre-central cortex of a gorilla has already been referred to (*see* p. 708 and Fig. 64).

Fibres from the motor convolutions gradually converge through the white matter of the centrum ovale to the base of the brain, and there pass between the lenticular nucleus and the optic thalamus, occupying the genu and the anterior two-thirds of the posterior limb of the internal capsule (*see* p. 708 and Fig. 65). Thence the pyramidal fibres pass into the lower part of the crus cerebri, at first somewhat on the outer side, and then in the middle; and they can be traced through the pons Varolii into the medulla oblongata. Here a *decussation* takes place, the greater part of each pyramidal tract crossing to the opposite lateral column, and a small portion remaining on the same side at the inner margin of the anterior column, forming the direct pyramidal tract, the fibres of which successively decussate lower down in the cord. The pyramidal fibres, the axons of the upper motor neurons, ultimately terminate in the anterior cornua by arborisations forming synapses with the dendrites of the lower neurons.

A sufficiently large destructive lesion of any part of the pyramidal fibres between the motor centres and the pons causes *hemiplegia*, or paralysis of the face, arm, and leg, on the *opposite* side of the body. This is a common result of a hæmorrhage into the internal capsule; but disease above this, where the fibres are spread over a wider area, may lead to a more limited paralysis. Extensive lesions of the cortex, such as suppurative meningitis, or meningeal hæmorrhage, will also cause complete hemiplegia, but smaller lesions may occupy only particular centres, and give rise to the form of paralysis called *monoplegia*. Thus a lesion of the facial centres results in paralysis on the opposite side of the facial muscles alone, *facial monoplegia;* and in corresponding manner there may be a *brachial monoplegia*, or a *crural monoplegia*. If the adjacent centres of the face and arm are involved together, *brachio-facial monoplegia* is the result, and if those of the arm and leg together, a *brachio-crural monoplegia*. But the centres for the face and leg can scarcely be affected at the same time without those for the arm, which lie between them, being involved; and, as a fact, a *cruro-facial monoplegia* has never been recorded.

The internal capsule is frequently involved in cerebral hæmorrhage, and paralysis of the limbs of the opposite side is usually the result of it. Hemianæsthesia may be present if the posterior fibres are affected, and hemianopia if the optic radiation is involved.

**Corpus Striatum.**—It has long been thought that few, if any, symptoms could be attributed to lesions of the corpus striatum; but recent researches have

shown that the motor cells of the lenticular nucleus form an important part of an extra-pyramidal motor system, lesions of which are associated with muscular tremor and rigidity (*see* pp. 708, 840).

**Sensory Cortex.**—The relations of the sensory neurons in the brain have been mentioned (*see* p. 707). The centres in the sensory cortex for different parts of the body do not appear to be localised so precisely as the motor centres. Head has emphasised the fact that cortical representation is determined by physiological rather than anatomical conditions, and points out that in man the hand, being the chief explorer of space, is widely represented in the sensory cortex. In presumably minimal lesions of the cortex due to glancing gunshot wounds he has shown that loss of the power of spatial recognition, or of ability to distinguish differences and similarities between small objects, or of appreciation of differences of intensity in the case of pressure, temperature and pin-prick, may exist in almost pure form. Since the sensory fibres spread out fanwise in their path from internal capsule to cortex, it is clear that the deeper the lesion, the more likely will be wholesale loss of sensory function as opposed to these finer differentiations. According to Head, if the lesion be situated above the optic thalamus crude appreciation of touch, temperature and pain is not affected.

**Optic Thalamus.**—The posterior part of this grey mass contains the central ends of some optic nerve fibres, and lesions may produce complete hemianopia. But it is in the optic thalamus that all the afferent fibres, passing up from the mid-brain, terminate by forming synapses with neurons which carry on the impulses to the sensory cortex. Roussy has formulated the symptoms which result from a lesion of the optic thalamus—his *syndrome thalamique*. They affect, of course, the opposite side of the body, and are (*a*) persistent hemianæsthesia, more or less marked for superficial sensations (touch, pain and temperature), but always very pronounced for deep sensation; (*b*) slight hemiplegia, usually without contracture and rapidly recovering; (*c*) slight hemiataxia and more or less complete astereognosis; (*d*) acute, persistent, paroxysmal pains, of burning or stabbing character, often intolerable and yielding to no analgesic remedies; (*e*) choreic and athetotic movements. Hemianopia may be present if the posterior or lower part of the thalamus is involved. But the sensory loss and the pains are the only symptoms due to the lesion of the thalamus itself; the others result from pressure on surrounding parts. Head and Holmes show that in addition there is often an excessive reaction to unpleasant stimuli, so that pin-pricks, scraping, tickling, pressure, and extremes of heat and cold were all more unpleasant on the affected side than on the healthy side. Similarly a pleasant sensation of warmth was appreciated more on the affected side.

The explanation offered for the occurrence of this syndrome as the result of a destructive lesion is that it is a manifestation of unbalanced thalamic activity due to removal of inhibitory control from above (*see* p. 707).

**Frontal Lobe.**—The posterior extremities of the upper two frontal convolutions, adjacent to the pre-central, contain, according to Ferrier, centres for rotation of the head and eyes; the posterior extremity of the third frontal convolution is connected on the left side with speech movements, so that a lesion there situated causes motor aphasia (*see* p. 718). The remaining portion of the frontal lobe seems to be related to the higher functions of the brain, and lesions may lead to loss of memory, of the control of emotions, and of the powers of attention, of association, of ideas, and of judgment, depending to some extent upon the previous intellectual and temperamental assets of the individual.

**Parietal Lobe.**—Besides the sensory centres already referred to, this contains on the left side in right-handed persons areas in and around the supramarginal gyrus which are connected with the reception of speech through auditory and visual channels, and with the elaboration of thought in terms of speech.

**Temporal Lobe.**—The upper convolution contains the centre for hearing, but destructive lesions do not give rise to deafness, since the lower auditory centre

on each side is connected with both sides of the brain. A lesion on the left side in a right-handed person may cause word deafness, in which words are heard as sounds, but their meaning is not understood (*see* p. 718). Irritative lesions of the superior gyrus cause subjective sensations of sound. Irritative lesions of the uncinate gyrus of the temporal lobe give rise to peculiar subjective sensations of smell or taste. An attack of this kind is frequently preceded by a dreamy state, with indefinite visual hallucinations; this is followed by a sense of an unpleasant taste or odour, which lasts a few seconds. There is as a rule no loss of consciousness with such an attack. The posterior part of the superior temporal gyrus on the left side is included in the area referred to above under Parietal Lobe as being concerned with the functions of speech.

**Occipital Lobe.**—This contains cortical centres for vision, and their destruction on one side causes hemianopia of the opposite side. The researches of Holmes and Lister show that the centre for macular or central vision lies in the most posterior part of the visual areas, probably on the margins and in the lateral surfaces of the occipital poles. The macula has not a bilateral representation. The centres for vision subserved by the periphery of the retina are situated in the anterior portions of the visual areas, and the serial concentric zones of the retinæ from the macula to the periphery are probably represented in this order from behind forwards in the visual areas.

**Corpora Quadrigemina.**—A lesion of these bodies causes vertigo, incoordination of the gait of cerebellar type and double ophthalmoplegia. The latter is of the nuclear type and in the beginning may be limited to ptosis and paralysis of upward movement of the eyes, with which may be associated myosis and inactivity of the pupils. Deafness may also occur from involvement of the inferior corpora quadrigemina.

**Pituitary Body** or **Hypophysis.**—The nervous symptoms which may result from a tumour of this organ are explained by its pressure upon adjacent parts. The most characteristic are double temporal hemianopia from pressure on the inner side of each optic tract, optic atrophy, the hemiopic pupillary reaction, and some ocular paralysis. Acromegaly and other effects upon growth and development may accompany these symptoms or occur alone. (*See* Diseases of the Ductless Glands.) Polyuria may be caused by a lesion of the brain tissue at the base of the infundibulum.

**Pineal Body.**—Here also the nervous symptoms if present are due to pressure on surrounding parts. They are the usual pressure symptoms of intracranial growths (*see* p. 849) together with those seen in the lesions of the corpora quadrigemina, viz. double ophthalmoplegia, ataxia, vertigo, and tremors. The other results are changes in growth and development. (*See* Diseases of the Ductless Glands.)

**Crus Cerebri.**—Paralysis of the limbs on the opposite side and of the third cranial (oculo-motor) nerve on the same side is characteristic of a lesion of the cerebral peduncle at its inner and posterior part; this has been called the syndrome of Weber. Tremor of the opposite limbs with oculo-motor paralysis on the same side, or Benedikt's syndrome, is also due to a lesion of the crus cerebri. Gordon Holmes notes that lesions of the *mid-brain* (which includes the corpora quadrigemina and the crus cerebri, with the substantia nigra and red nucleus) have not infrequently been accompanied by definite, rather slow, tremors of the limbs.

**Pons Varolii.**—This portion of the brain contains the pyramidal tracts, and the nuclei of the fifth, sixth, and seventh nerves. Large central lesions may paralyse all four limbs from the proximity of the two tracts to the middle line. A one-sided lesion in the upper part produces hemiplegia of the ordinary type (*see* p. 817) on the opposite side of the body; but a lesion in the lower part, while involving the same pyramidal fibres for the arm and leg, is below the facial fibres for the opposite side, and destroys the facial nerve roots of its own side.

There is then produced a variety of hemiplegia known as *crossed hemiplegia*, the face being paralysed on the side of the lesion, but the arm and leg on the opposite side. A lesion involving the nucleus of the sixth nerve on one side will give rise not only to paralysis of the external rectus on this side, but also to paralysis of the internal rectus of the opposite eye in attempted conjugate deviation of both eyes towards the side of the lesion (*see* p. 820).

**Cerebellum.**—The results of destructive lesions of this body have already been considered in discussion of its functions (*see* p. 714). Irritative processes may in rare instances give rise to so-called cerebellar fits in which the trunk assumes the position of opisthotonus, the head is bent backwards, the arms are fully extended by the sides and pronated with wrists and fingers flexed, while the lower limbs assume a position of extreme extension with toes pointing downwards. The picture is akin to that seen in decerebrate rigidity, and perhaps depends upon similar causes.

**Doubtful Regions.**—There are some parts of the brain, known also as silent areas, or latent regions, the destruction or irritation of which *may* produce no distinctive phenomena. Of cortical regions the frontal lobe, part of the inferior parietal, most of the right temporal lobe, and some of the left temporal lobe are of this nature and in the deeper parts, some portions of the centrum ovale. It is probable that the extensive meshwork of association fibres contained in these silent areas has to do with individually acquired and variable reactions, and that localisation of these functions is less constant. It may be also that when the destruction is of slow development (as in the case of tumour) neuronic pathways still intact may function vicariously for those damaged.

## THE ARTERIES OF THE BRAIN

In connection with the frequency with which hæmorrhage, embolism, and arterial thrombosis are causes of cerebral disease, the distribution of the arteries of the brain has much importance.

The *anterior cerebral* artery supplies the superior frontal convolution, and the inner surface of the hemisphere nearly as far back as the occipito-parietal fissure. The *middle cerebral* artery supplies the greater part of the outer convex surface of the brain, all, indeed, except the superior frontal and the occipital convolutions; that is, it supplies the second and third frontal, the pre-central and post-central, the parietal, and upper and middle temporo-sphenoidal gyri. The *posterior cerebral* supplies on the outer surface the occipital lobe, and all the inner or tentorial surface from the limit of the anterior cerebral distribution to the tip of the temporal lobe, which is supplied by the middle cerebral. The *vertebral* and *basilar* arteries supply the cerebellum and the pons Varolii.

The central parts of the brain, including the great ganglia, are supplied by branches, which come off from the circle of Willis and the origins of the three cerebral arteries, pass vertically into the nerve substance, and do not anastomose with each other or with the arteries of the cortical system. These are divided into groups: one, the *internal striate*, supplies the two inner parts of the lenticular nucleus, and the anterior part of the internal capsule; another, the *lenticulo-striate*, supplies the outer part of the lenticular nucleus; and a third, the *lenticulo-optic*, supplies the anterior part of the thalamus. One large vessel in the lenticulo-striate group was called by Charcot the *artery of cerebral hæmorrhage*, from the frequency with which its rupture was the cause of apoplexy.

The relations of the central ganglia to the larger arteries are as follows: The *caudate nucleus* derives its supply from the anterior and middle cerebral arteries; the *lenticular nucleus* from the anterior choroid and middle cerebral; the *optic thalamus* from the posterior communicating central branches, and from the posterior cerebral; the *corpora quadrigemina* and *geniculate bodies* from the pos-

terior cerebral; the *internal capsule* in its anterior half from the anterior cerebral and middle cerebral, in its posterior half from the posterior communicating, anterior choroid, and middle cerebral arteries.

## HEMIPLEGIA

Before dealing with the diseases of the brain it is desirable to describe in more detail the form of paralysis known as hemiplegia.

Though this term seems to imply paralysis of one-half of the body, as a matter of fact some muscles are not paralysed in ordinary cases of hemiplegia, and of those that are paralysed some suffer much more than others. Paralysis is most marked in the arm, leg, and face, on the side opposite to that of the lesion; the muscles of the trunk, chest, and abdomen are but little affected, and the ocular muscles not at all. It is stated that, on careful testing, some weakness of the muscles on the apparently unparalysed side can also be shown.

Even in the face, arm, and leg, many differences exist. In severe cases the arm and leg may be completely motionless, but in milder cases the leg is less paralysed, and in course of recovery the leg nearly always gets better first. The facial muscles are never paralysed to the same extent as they are in a lesion of the nucleus or trunk of the facial nerve (*see* p. 749); the upper half of the face is always less affected. Thus the eye can always be shut, and the forehead wrinkled, though it may be obvious that the frontalis and the orbicularis are not so strong as those on the opposite side. In the lower part of the face the paralysis is marked: if the patient is told to smile, or to show the teeth, the angle of the mouth is drawn up on the healthy side, and on the paralysed side it remains unmoved or is drawn inwards. But these paralysed muscles under the influence of emotion will contract more powerfully than they do when stimulated by voluntary effort. Usually there is a slight degree of paralysis of the tongue shown by the tip being turned, during protrusion, to the paralysed side by the unbalanced action of the healthy genio-hyoid and genio-hyoglossus muscles. The action of the respiratory muscles varies with the nature of the movement. In many cases during ordinary breathing, the upper part of the chest on the paralysed side moves more than that on the other side; H. Jackson said this was a result of injury to fibres between the cortex and medulla on the opposite side, which act as continuous inhibitors of the respiratory centre. But during forced inspiration the movement on the paralysed side is less than that on the other. The weakness of the abdominal muscles on the paralysed side may be shown when the patient coughs, and by the divergence of the umbilicus to the unparalysed side when he tries to sit up. But paralysis of the spinal muscles is not commonly observed, and the muscles moving the eyeball, the muscles of mastication, and those of the larynx are unaffected. The explanation commonly adopted for these variations in the paralysis is as follows. It is first to be observed that the parts that are least paralysed, or not paralysed at all, are those which rarely or never act independently of their fellows on the opposite side, whereas the parts that are most paralysed are much more independent, and may be capable of performing acts of which the corresponding muscles on the opposite side are incapable. As an extreme instance may be mentioned the eyes, of which one never moves except in association with the other; their muscles are not affected. In contrast with these are the hands, of which the right may be able to do things the left cannot, and *vice versâ*; these parts are most affected. There is of course a close association by commissural fibres both in the brain and in the spinal cord between the two sides; and we must suppose that when particular movements involve synchronous (or alternating) actions of the same muscles on the two sides of the body, such as the ocular movements, phonation, and locomotion, the commissural connections have become functionally active in the highest degree, so that in the case of a lesion of the brain

on one side the corresponding muscles can be stimulated by the opposite undiseased centres, whereas in unilateral movements the commissures are not functionally active, and no help can be derived from the healthy centres. In other words, the bilaterally associated muscles are represented in, and can be stimulated by, both hemispheres, whereas the muscles acting independently are excited by the opposite hemisphere only.

**Causes of Hemiplegia.**—This form of paralysis may arise from any lesion involving, either directly or by pressure, the pyramidal tract from the cortex to the upper part of the pons Varolii. If the lesion is mainly cortical, it must be sufficiently extensive to include the centres of the face, arm, and leg. With these on the left side, the motor speech centre may be involved, when *aphasia* will result; and if a right hemiplegia occurs without aphasia it is possible that the cortical lesion is nearer the middle line, and then the leg may be more paralysed than the arm. The most frequent causes are hæmorrhage, embolism, and thrombosis, involving cerebral vessels; others are infective encephalitis and more localised abscess; tumours; meningitis, whether suppurative or tuberculous; meningeal hæmorrhage; injury to the surface of the brain, as from blows, or from compression of the infant's skull during birth, whether by forceps or not (*birth palsies*).

**Course and Associated Conditions.**—The duration of hemiplegia is very variable. It may get completely well in the course of a few weeks, the power of movement being gradually restored in the face first and the leg afterwards, so that the patient can walk about, while the arm is still useless until this also recovers. Sometimes recovery progresses up to a certain point during the first two or three months, until a stage is reached beyond which no improvement takes place. The paralysis is then generally accompanied by rigidity or contracture of the muscles, which develops during the first few weeks. The fingers become flexed into the palm of the hand, and the elbow is slightly bent; any attempt to overcome the flexion is resisted, and causes considerable pain. In the lower extremity the knee is only slightly flexed, and the foot is often extended at the ankle. With this so-called *late* rigidity—to contrast it with the rigidity of the apoplectic state described below (*see* p. 823)—the deep reflexes are increased, the knee jerk is greater, and ankle clonus is readily obtained. The superficial abdominal reflexes are weak or absent on the affected side, and the plantar reflex is extensor. The muscles may lose a little of their bulk from disuse, but are not degenerated, and the electrical reactions, faradic and galvanic, remain normal. This condition is similar to what may be seen in such lesions of the spinal cord as involve the pyramidal fibres, or upper neurons, alone (primary spastic paraplegia); but it differs from what occurs in the lesions of the greater transverse extent, which myelitis usually involves. In this latter, if the transverse lesion is complete, the paralysed lower limbs tend to assume the position of flexion; in the cerebral lesion now under consideration, there is spastic paralysis in extension. The increased reflexes are due to loss of higher control, and are present from an early stage of the paralysis; the rigidity is attributed to *hypertonus* conveyed to the extensor muscles by descending fibres from tonic centres in the mid-brain (*see* p. 715).

Some involuntary associated movements occur also in hemiplegia. When the patient grasps, for instance, the observer's hand with his unparalysed hand, an associated movement takes place in the hand of the paralysed side, unless the paralysis is very complete. When he tries, with folded arms and legs widely separated, to sit up in bed, he flexes the thigh of the affected side strongly on the trunk, the healthy thigh not rising from the bed level (Babinski). Strümpell observed that when the patient, lying on his back, flexes the leg on the thigh, while the physician opposes this flexion by pressure on the front of the thigh, the foot assumes the position of talipes equino-varus from contraction of the tibialis anticus.

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More rarely there occur in the partially paralysed muscles other disorders of movement, of which the most important is *athetosis*. In this there is a constant involuntary slow movement of the fingers, which are abducted, adducted, flexed, and extended in the most irregular way (*see* Fig. 89). Similar, but generally less extensive, movements may affect the arms and the toes.

The occurrence of these post-hemiplegic disorders of movement is especially frequent when hemiplegia arises in early life, either at birth or within the first ten years (*infantile hemiplegia*); this is often the result of encephalitis. The paralysis frequently recovers up to a certain point, when some muscles become rigid and others become the subjects of athetosis. This is most marked in the arm, which is flexed at the elbow, with the hand dropped, and the fingers and thumb twisting and writhing. The leg of the same side is stiff, with slight flexion of the knee, talipes equino-varus, the tendo Achillis rigid, and the toes moving about in a purposeless manner. The gait is limping, and the pelvis is tilted to give room for the swing of the stiff leg. Another important feature is this, that as the child grows the affected limbs do not keep pace with the others, and the arm may be found years after to be 1½ or 2 inches shorter than its fellow, while the hand is narrower and altogether more delicate in form. The legs show similar but less marked differences. The muscles of the affected arm and leg may be less bulky than those of the opposite side, but are never decidedly wasted; and they show no diminution of the electrical reactions or reflexes, thus differing from the muscles in acute poliomyelitis. Sometimes the muscles are extraordinarily hypertrophied, probably from the constant involuntary contractions. In some cases convulsions occur in the paralysed limbs; in others the patients are dull, stupid, epileptic, or idiotic; but in others, again, the mental condition is perfectly normal. Some of these cases are described as *infantile spastic hemiplegia*.

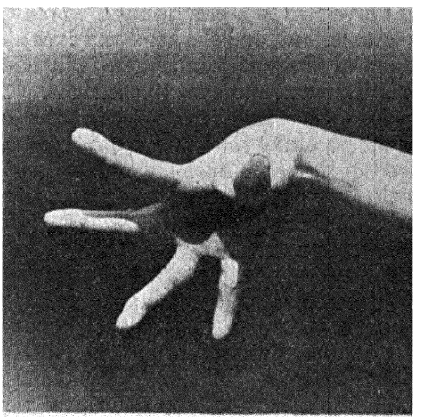

FIG. 89.—One of the Positions which the Hand may assume in Athetosis. (*After Turner and Stewart.*)

A pronounced *anæsthesia* is not common with hemiplegia, and if it occurs with the onset of the attack it generally passes off within a few days. It is on the same side of the body as the motor paralysis. Dr. Gordon, of Philadelphia, says that in nearly all cases there is some affection of cutaneous sensation, but its complete loss is uncommon, and hyperæsthesia in any form is still rarer. The sense of pain is diminished most of all (*analgesia*), that of temperature is less so, and the sense of touch still less. *Astereognosis* is also often present. These changes are more marked in the upper limb than elsewhere. With hemianæsthesia there is sometimes associated hemianopia.

*Conjugate deviation*, or forcible rotation of the head and eyes to one side, is occasionally associated with the sudden or apoplectic onset of hemiplegia: it may continue after consciousness has returned; but it generally subsides after a few days or a week. Ferrier has found that there is a region of the cortex situate in the *frontal* lobe, irritation of which causes deviation of the head and eyes; but pathological results show that conjugate deviation is not restricted to lesions of one locality alone. There is, however, an important connection between the position and the nature of the lesion and the side to which the

deviation occurs. When the lesion of the *left* side of the brain occurs, giving rise to *right* hemiplegia, the eyes and head are turned to the *left* side ; that is, the eyes are turned *away from the paralysed side*, or the patient is said to *look towards his lesion*. But if, as a result of this lesion, there should occur convulsions in the paralysed limbs, or if a cerebral lesion of any kind causes convulsions on the opposite side with rotation of the head, neck, and eyes, the rotation will be *towards* the convulsed side—that is, the patient will appear to *look away from his lesion*. And similarly, with right-sided lesions, there will be left-handed paralysis with right-sided deviation, or left-sided convulsion with left-sided deviation. Always, then, in *cerebral* lesions, deviation with paralysis is away from the paralysed side, deviation with convulsion is towards the convulsed side. The explanation given for this phenomenon is as follows : The median position of head and eyes is normally maintained by the perfectly balanced action of two opposing forces, which depend upon tonic innervation of the eye muscles in combination, together with the rotators of head and neck. The upper motor neuron centre for rotating head and eyes to the right is situated in the left cerebral hemisphere. A destructive lesion of this centre or the pathway leading downwards from it will give rise to a condition in which the continued action of the opposite centre is unbalanced, and the head and eyes are therefore rotated to the left. An irritative lesion of the left hemisphere will cause temporary over-action of the centre, rotating head and eyes to the right.

The fibres from the upper motor centres for conjugate deviation decussate before reaching the lower motor centres in the pons. These are in the neighbourhood of the sixth nuclei, each of which sends fibres across the mid-line to connect with the motor cells innervating the opposite internal rectus. A destructive lesion of the left side of the pons therefore will give rise to paralysis of the rotators of head and eyes to the left, and there will result, from unbalanced over-action of the intact opposite centre, conjugate deviation away from the side of the lesion. An irritative lesion of the pons will produce conjugate deviation towards the side of the lesion.

Mental symptoms are not very infrequent accompaniments of hemiplegia, from whatever cause. There is often confusion of mind, loss of memory, difficulty in fixing the attention, and emotional weakness, so that the patient readily cries or laughs, especially the former. If speech is affected (*aphasia* or *dysarthria*) the mental condition may be difficult to appreciate ; it will probably appear defective, and, according to some, the occurrence of an *aphasia*, involving loss of memory for words, must of itself impair the mental processes.

## APHASIA

The term *aphasia* means loss of speech from a cerebral lesion, and must be distinguished—

(1) From *aphonia*, or voicelessness, which is due to failure of the laryngeal muscles, and is not indeed a loss of speech, since words can be uttered by whispering ;

(2) From *dysarthria*, or defect of articulation, which is due to imperfect action of the muscles of the lips and tongue, consequent upon paralysis of their motor supply, whether from upper or lower motor neuron lesions, or to loss of co-ordination of these muscles from a lesion of the cerebellum or its efferent pathways (*see* p. 716) ;

(3) From purely *mental* aberrations independent of demonstrable lesions of the cerebral centres.

With rare exceptions, aphasia is due to a lesion on the left side of the brain, and consequently, if it is associated with hemiplegia, it is with a *right hemiplegia*, and not with a left hemiplegia. The only explanation of this is that the left side

of the brain is alone or chiefly educated for speech purposes; and this view receives support from the rare cases of aphasia associated with left hemiplegia, in which it has often been found that the persons were left-handed. It is thus suggested that the side of the brain which is educated for the most extensive use of its associated opposite arm also develops the functional activity of its speech centres, while those of the opposite side are comparatively inactive; but there is good reason to believe that they have some share in speech processes: for instance, cases of *crossed aphasia* have occurred in which a left-handed person has had aphasia in association with a right hemiplegia. As an explanation of some of these cases, however, Foster Kennedy has put forward the theory that right-handed individuals of strongly left-handed stock may have their speech centres in the right hemisphere, and *vice versâ*.

In aphasia, then, the muscles of the lips are used perfectly so far as the utterance of any letter or even of any syllable is concerned. The words which the patient can speak are clear, distinct, and natural, or if words and syllables are mixed unintelligibly, it is obviously from imperfection of the higher centres or from want of perfect automatism in them rather than in the action of the muscles of articulation. There is none of the blurred utterance or thick speech of bulbar paralysis, general paralysis, or alcoholism.

The various types of aphasia, together with the localisation of the lesions producing them and methods for their investigation, have already been described (*see* pp. 718, 723).

## CEREBRAL HÆMORRHAGE

In dealing with diseases of the brain prominence must be given to diseases of the vessels of the brain, which are responsible for the majority of cases of cerebral paralysis. Rupture of the vessels, with escape of blood (*hæmorrhage*) into the brain, and obstruction of the arteries by *embolism* and by *thrombosis*, are the forms of vascular lesion which have to be considered.

**Ætiology.**—Cerebral hæmorrhage occurs more frequently in men than in women, and more in advanced life than in youth. Thus it occurs, but is very infrequent, below the age of thirty, and has, indeed, occurred in one as young as nine; but nearly four-fifths of the cases occur after the age of forty. A large proportion have granular kidneys and hypertrophied hearts; the arteries are generally at the same time atheromatous, sclerosed, or calcareous, late results of endarteritis; and frequently the actual cause of hæmorrhage is the rupture of minute (miliary) aneurysms, which are found on the branches of the cerebral arteries. Alcohol, gout, and syphilis have their share in the production of these arterial lesions. Hæmorrhage is sometimes associated with heart disease and endocarditis, and this is specially the case when it occurs in quite young people. Probably in these instances an embolism has been the first lesion, around which the artery has softened, and then it has ruptured, or the artery has yielded so as to form an aneurysm which has subsequently burst—a process similar to what will be described as occurring in the hæmorrhage from the lung in phthisis. Hæmorrhages often occur in the substance of tumours of the brain, and they may be so large as to render their source uncertain; smaller hæmorrhages take place in conditions of general tendency to bleeding (scurvy, purpura), and as a local result after ligature of the common carotid artery.

**Seats of Hæmorrhage.**—Scarcely any part of the brain is exempt from the risk of hæmorrhage, but it is much more frequent at the base in the neighbourhood of the corpus striatum and optic thalamus, which are mainly supplied by the branches of the middle cerebral artery, to which reference has already been made (*see* p. 816). Vessels may also burst in the lateral ventricles (*ventricular hæmorrhage*), or on the surface of the brain (*meningeal hæmorrhage*); but when blood

is found in these situations, it has often proceeded from a hæmorrhage primarily in the cerebral substance.

**Anatomical Changes.**—In different circumstances the blood effused may be small in quantity or amount to several ounces. In the latter case it tears up the cerebral tissue, destroying, for instance, the great ganglia and the internal capsule, and extending thence into the centrum ovale, or it may burst through the optic thalamus or caudate nucleus into the lateral ventricle. Thence the blood flows by the aqueduct of Sylvius into the fourth ventricle. Such cases are rapidly fatal, and post-mortem examination reveals a mass of dark red clot, filling the ventricle and occupying much of the hemisphere, surrounded by brain substance, which is ragged and discoloured by blood. In cases which have lasted a few days there is the same black-red clot, and the tissue around is soft and discoloured yellow, from absorption of hæmoglobin (*yellow softening*). The effect upon the parts of the brain not immediately destroyed by the hæmorrhage varies with the amount of the effused blood, and with the time which has elapsed since the hæmorrhage. If the clot is sufficiently large, it presses upon the sound cerebral tissue; the hemisphere, whether right or left, in which it has taken place, is enlarged; the convolutions are flattened against the interior of the skull; and the sulci are obliterated. After some hours or a few days secondary œdema of the affected hemisphere may occur, and further increase the size of this half of the brain. Either from the extent of the hæmorrhage or from the occurrence of the secondary œdema, this increase may be so great as to cause considerable compression of the healthy hemisphere, also with flattening of the convolutions and closure of the sulci. To this compression of the healthy side of the brain P. Marie attributes the occurrence of the coma which is so common in cases of cerebral hæmorrhage (*see* Fig. 90). In later stages the clot becomes brown or brownish yellow, consisting of disintegrated blood and nerve structure; and the surrounding tissue is frequently softened (*white softening*), and contains large mononuclear phagocytes full of granular *débris*. Finally, in patients who survive the blood becomes absorbed, and leaves a tawny or orange coloured spot, in which crystals of hæmatoidin can be found; or a cyst may remain, containing serous fluid, or a distinct, tough, fibrous scar, discoloured also by the remains of blood pigment.

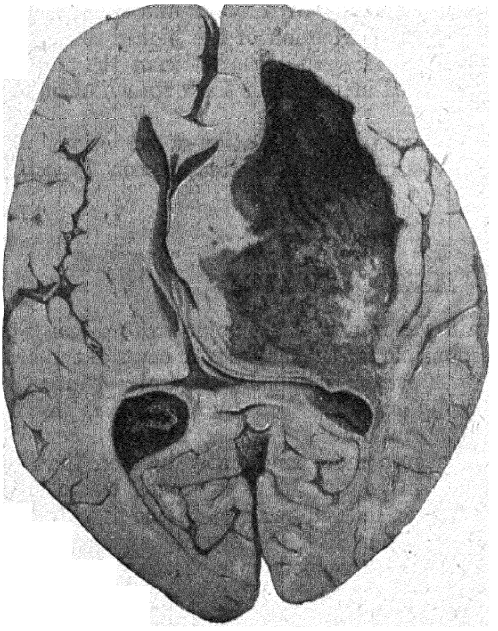

FIG 90.—Section of Brain in Cerebral Hæmorrhage with Coma, showing compression of the healthy side. (*After Pierre Marie.*)

**Secondary Degeneration.**—Permanent lesions of the pyramidal tract, or of the cortical motor area, are followed by secondary descending degenerations, like those which occur in disease of the spinal cord. Such degenerations follow the course of the pyramidal fibres below the lesion; thus a lesion of the internal capsule causes this change to take place in the middle third of

the crus cerebri, in the anterior part of the pons, in the pyramid of the medulla oblongata on the same side, in the direct pyramidal tract, also on the same side, but in the posterior part of the lateral column of the spinal cord (crossed pyramidal tract) for its whole length on the opposite side. Lesions of the corpus striatum or optic thalamus alone are not followed by secondary degeneration.

**Course and Symptoms.**—*Apoplexy.*—Cerebral hæmorrhage may be preceded for days or weeks by occasional giddiness, numbness or twitching of the fingers, headaches, insomnia, or some diminution of mental capacity; but these are not so much indications of the severe attack to come as evidences of existing disease of vessels, and perhaps due themselves to slight hæmorrhages. On the other hand, it may come on without any warning whatever. Sometimes also it seems attributable to a definite cause, such as emotional excitement, muscular effort, violent coughing, or straining at stool; but at others it occurs when the patient is perfectly quiet, or even during sleep. In numerous cases cerebral hæmorrhage causes a group of symptoms known as *apoplexy*—that is, the patient is struck down suddenly unconscious, or he quickly becomes so (ἀποπλήσσειν, to strike to earth). Patients have died in five or ten minutes from the first symptom. More often the symptoms come on slowly. The patient is seized with intense pain in the head, becomes faint or slightly collapsed, may be sick or have a slight convulsion, and then, after half an hour or more, gradually sinks into a condition of coma. Or the first symptoms may show themselves in the motor system: the patient mumbles in his speech, or his arm drops powerless, and he gradually leans over to one side, falling if not supported, and then lapses by degrees into coma. Or the coma may be developed in a few hours through stages of increasing drowsiness, or the attack may begin with convulsions. In some cases the collapse is extreme, and the pulse is barely perceptible; but after an hour or two it improves and becomes full and bounding as the patient assumes the condition described below with complete coma and paralysis. Cases in which some hours elapse before the coma is complete have been called *ingravescent apoplexy*. When the patient is found by the friends alone, or is picked up in the street unconscious, or is unable to be roused in the morning from sleep, it is of course impossible to say what the onset has been. But undoubtedly cerebral hæmorrhage may occur without apoplexy; a very slight bleeding into the motor tract alone may give rise to paralysis without loss of consciousness, but this is uncommon.

The patient suffering from hæmorrhagic coma lies completely unconscious, and cannot be roused by shouting or any form of stimulation of his skin. The face is flushed; the pulse is full and tense; the breathing is *stertorous*, a loud, snoring noise being made in consequence of the palate or tongue falling back and impeding the passage of air into the chest. The condition of the limbs varies: both legs and arms may be quite flaccid, falling at once when raised, or it may be obvious that the leg and arm on one side are more flaccid than those on the other. The muscles of the face share in the paralysis, and the cheeks are puffed out and sucked in with the processes of respiration; this may also occur only on one side. Sometimes, however, the limbs of one or both sides are in a condition of rigidity (*early rigidity*), the muscles contracted and resisting extension or flexion. The deep reflexes are commonly increased, and the skin reflexes are absent. The pupils are variable: they are sometimes contracted, at others dilated or unequal. Conjugate deviation may occur. The temperature shows a slight fall, which may continue till death, or if life is prolonged it rises a little above the normal. A greater or persistent fall of temperature, like a rapid or excessive rise of temperature, is of ominous significance. The latter is often associated with rupture into the lateral ventricles. Sugar is occasionally found in the urine, from pressure on the medulla oblongata. A trace of albumin is commonly present in association with primary contracted kidneys. In very severe

cases the pulse and breathing are rapid, there is profuse sweating, and intense flushing of the face and skin generally; then after a time, two or three hours or more, the patient becomes livid, râles occur in the larger bronchi and trachea, the pulse gets weaker, the breathing slower, and finally death takes place. The fatal termination may, however, be delayed for several days, during which the lungs are very apt to suffer from œdema or pneumonia; and the occasional passage of particles of food or fluids through the glottis probably contributes to the inflammation of these organs. In more favourable cases, the patient lies simply comatose, with but little disturbance of his pulse or respiration, and gradually regains his senses in the course of a few hours or two or three days. In a large proportion of cases, the patient is then found to be suffering from *hemiplegia*, which may itself slowly recover or be permanent (*see* p. 817).

**Diagnosis.**—This has to be made from thrombosis, from embolism, from uræmia, from alcoholic intoxication, and from other conditions causing apoplexy on the one hand and hemiplegia on the other. It will be best considered after cerebral embolism has been described.

**Prognosis.**—This is generally unfavourable in proportion to the extent of severity of the first symptoms. That apoplexy is often fatal is well known, and death may take place at different intervals after the onset. The cases are grave in which the coma is profound, with much stertor, flushed or congested face, full bounding pulse, and complete relaxation of all the limbs.

If, after recovery from the first coma, headache continues, and the patient again becomes drowsy, the result is likely to be serious.

**Treatment.**—The first thing to consider is whether there is any possibility of arresting the hæmorrhage. During the attack the patient should be in the recumbent position, with the head and shoulders slightly raised. Absolute rest must be insisted upon. In the very acute cases there may be no time to do anything else; but in other cases, with a raised blood pressure, venesection is the correct treatment. Usually a cerebral hæmorrhage is formed gradually by a small leak from the artery. This leak continues so long as the blood pressure remains high; but it may stop if the blood pressure can be a little lowered, even though this is only temporary. The rapid removal of a pint of blood often accomplishes this, and in any case no harm is done. To be effective, the venesection must be carried out at the earliest opportunity.

Stertor is often diminished by placing the patient on one side, and this is further beneficial by allowing the full play of at least one lung, viz. that which is uppermost. An ice-bag should be applied to the head. If the bowels are known to be confined, or not recently opened, a drop of croton oil or a few grains of calomel may be placed on the tongue, or an enema of castor oil or turpentine may be administered. If the coma continues, the catheter may be required to empty the bladder. In later stages, pain in the head requires the continued application of ice; the patient should be carefully nursed to prevent the formation of bedsores, and a water-bed may be needed. If hemiplegia occur without loss of consciousness, the patient should be kept quiet in bed, the bowels should be attended to, and a light diet of fish, milk, custard pudding, etc., should be enforced for some days or weeks. Neither here nor in the cases beginning with coma can the treatment of the paralysis be entered upon until all evidences of active mischief in the brain have subsided. As soon as this is the case, the limbs may be treated in order to delay as long as possible the onset of rigidity, contracture, or stiffness of joints. Massage, passive movements, and re-education are the means to be employed, and the patient should exercise his muscles as far as he can. Splints should be ordered to be worn at night in order to prevent deformity. The possibility of a second attack should be remembered, and the patient should have always a light simple diet, should abstain from alcohol, take only moderate exercise, and keep

from business or other mental worry as much as possible, and in particular avoid sudden exertion.

## EMBOLISM AND THROMBOSIS OF CEREBRAL ARTERIES

**Pathology.**—The usual cause of *embolism* is mitral or aortic endocarditis; the former is far more common. In either case particles of fibrin are detached from the surface of the valves, or in the case of a contracted mitral orifice fibrin may be deposited in the dilated left auricle, and subsequently detached and carried into the cerebral vessels. The middle cerebral artery is more often obstructed than others, and the left more often than the right; the reason for these differences is not clear.

*Thrombosis* is most frequently caused by disease of the vessel wall, such as atheroma, by which the surface is roughened, and fibrin is consequently deposited. Syphilitic disease of the arteries produces considerable narrowing of their channels, and thus favours thrombosis. In addition, thrombosis may occur from several conditions weakening the circulation, such as those resulting from enteric fever, typhus, cancer, phthisis, and other severe illnesses.

Embolism and thrombosis, by obstructing the circulation of the blood, alike lead to *softening* of the districts of the brain to which the vessels correspond, unless the vascular supply is maintained by means of anastomoses. These are not abundant in the case of the cerebral vessels, and, indeed, the vessels going to the central ganglia are really terminal vessels, while those going to the cortex of the brain anastomose more or less. At least, this is true of the distribution of the middle cerebral artery—the vessel most often obstructed. A part of the brain in which softening has taken place has generally lost the smooth, glistening surface of a normal brain section, is more opaque, or grey, or speckled; it breaks down readily under a stream of water; or it is milky or diffluent. It shows under the microscope drops of myelin, portions of nerve fibres, large mononuclear phagocytes containing granular *débris*, and free fat globules. It sometimes has a yellowish or brownish colour from altered blood pigment; or minute extravasations of blood may be present in cases of sudden obstruction, and a form of *red softening* results. In cases of rapid death after embolism, the brain substance may look perfectly healthy, as there has not been time for any changes visible to the naked eye to take place. Occasionally an embolus sets up inflammatory changes in its neighbourhood; sometimes it leads to aneurysm and cerebral hæmorrhage, as already described (*see* p. 821). Rarely actual infarcts are formed. The later stages of softening consist in the absorption of the disintegrated tissue, and the formation of the cyst; or, if the softening is small, a cicatrix may be produced.

Embolic lesions, involving the motor tract, are followed by the same secondary changes as are hæmorrhagic lesions. A persistent lesion of the brain, whether embolic or hæmorrhagic, causing hemiplegia in infancy or early childhood, has the remarkable effect of checking the growth of one-half of the brain, or, it may be, of other parts of the central organs, so that years after it is smaller than the other half, and is described as atrophied (*cerebral hemiatrophy, unilateral atrophy*). If the lesion is in the motor cortex, the hemisphere is atrophied on that side, and there is sclerosis of the pyramidal tract; if it is in the basal ganglia, there is in addition atrophy of the middle fillet in the pons and medulla, and of the antero-lateral region of the spinal cord on the *same* side, and atrophy of the cerebellum, superior cerebellar peduncle, and dentate nucleus on the *opposite* side (Mott and Tredgold). The growth of the paralysed limbs is also impaired.

**Symptoms.**—The results of *embolism* are not very different from those of hæmorrhage; but it more often causes sudden hemiplegia without loss of consciousness than does hæmorrhage. Obstruction of a large vessel will cause

sudden loss of consciousness, and death may take place soon after. In other cases, coma comes on more gradually, and may be preceded by pain in the head. When the patient comes out of his coma, he is often found to be paralysed on one side, and if the paralysis is on the right side, aphasia may be present. Since the softening occurs only in the areas supplied by the vessel beyond the seat of obstruction, the symptoms are more likely to correspond with the distribution of the artery than in hæmorrhage, where the extravasated blood ploughs up the brain with little discrimination. If the middle cerebral be obstructed near its origin, there will be hemiplegia of the opposite side, and if the lesion is on the left side, aphasia also, since this artery supplies the internal capsule, Broca's convolution, the greater part of the motor area of the cortex, the first and second temporal convolutions, and the angular gyrus. Persistent hemiplegia is accompanied by the conditions already mentioned (*see* p. 817).

*Thrombosis* is usually less rapid in its effects, though with the same results – apoplexy and hemiplegia; but sometimes a sudden coma occurs, indistinguishable from that of hæmorrhage. There are often premonitory symptoms—headache, dizziness, loss of memory, drowsiness, numbness, or formication of an arm or leg, or of one side of the body. Senile forms of disease are frequently of this nature. The symptoms are aggravated from time to time by fresh lesions, not necessarily confined to the motor tracts, and they are often followed by mental weakness, or dementia—the "softening of the brain" of old people.

**Diagnosis of Cerebral Vascular Lesions.**—This may be divided into two heads—the diagnosis of apoplexy from other conditions simulating it and the diagnosis from one another of the different causes of apoplexy or hemiplegia.

1. In the former the history is of great importance. Causes of coma occurring in the course of severe illnesses, such as meningitis, cerebral abscess, cerebral tumour, typhoid fever, etc., may be readily excluded by the history; it is coma coming on suddenly or rapidly which may be confounded with apoplexy. In *pyæmia* a sudden coma has sometimes occurred closely resembling that of apoplexy. More commonly the conditions to be discriminated from it are coma from injury, poisoning by opium, alcoholic poisoning, uræmia, diabetes, epilepsy and heart-block. In hot countries it will be necessary to remember that coma arises from heat stroke and pernicious malaria.

Cases of *injury*, in the absence of history, may present the greatest difficulties, as, even with the external evidence of injury, it may remain uncertain whether the patient has fallen as a result of apoplexy, or has injured his brain in consequence of the fall. Even after death the problem may be insoluble. The position of a scalp wound in relation to the weaker side, if paralysis can be recognised, may sometimes help; and the age of the patient, or other circumstances of his bodily health, may render a spontaneous lesion of the brain more or less likely. In cerebral concussion the patient is pale, with dilated pupils and low blood pressure. His limbs are flaccid. Recovery is usually heralded by vomiting and convulsions. In sub-dural hæmorrhage there are symptoms of *compression*, with slow pulse; there is gradually deepening coma with remissions, during which the patient is quite conscious.

*Opium-poisoning* is generally distinguished by the minutely contracted pupils, the slow pulse, and slow respiration; but it may be closely simulated by hæmorrhage into the pons Varolii. Evidences of the unilateral lesion, such as greater flaccidity or rigidity of limbs on one side, or unequal pupils, are in favour of hæmorrhage.

The same may be said of *alcoholic poisoning*. The condition is one of profound coma, without any one-sided symptoms. Evidence of alcohol may, of course, be obtained from the breath, or from the stomach by means of emetics or the stomach tube. But a patient may have drunk freely or sufficiently just before an apoplectic attack, or if the attack has come on gradually he may have taken a glass of spirits as treatment.

*Asthenic uræmia* is accompanied by albuminuria, but the detection of albumin in the urine does not exclude cerebral hæmorrhage, for, in the first place, hæmorrhage occurs often in those who have granular kidneys; and, secondly, hæmorrhage may itself produce albuminuria in those who have healthy kidneys. In uræmia sometimes the coma is less profound; the patients are more easily roused for a time by shouting, to relapse again into coma. There is no paralytic weakness, no vasomotor disturbance, and no flush of congestion, such as occur in some cases of apoplexy. Addison used to call attention to the hissing nature of the breathing. Uræmia will be indicated by a large increase of urea in the blood (*see* p. 629).

*Diabetic coma* develops very slowly, and is not profound till near the end; it is often preceded by severe abdominal pain, so severe sometimes as to have led to a diagnosis of perforation of the intestine. The pulse is rapid and feeble, the breathing is often slow, deep, or sighing, and the breath has a sweet odour. The urine contains sugar and aceto-acetic acid; but here also a mistake is possible, for a hæmorrhage involving the fourth ventricle may produce glycosuria.

*Epilepsy* is sometimes followed by coma, which is more like natural sleep than that of apoplexy; the patients are more easily roused, and there are no unilateral symptoms. Early age would be opposed to apoplexy. On the other hand, a gradual onset excludes epilepsy. Occasionally *hysterical* patients will lie unconscious for long periods, but the cases are generally distinguished by other characteristic symptoms.

The chief feature in *heart-block* is the extremely slow pulse (*see* p. 305). There may be convulsions, and the breathing is deep and rapid.

2. In the diagnosis of the causes of apoplexy, one has to consider the nature of the attack and the associated condition of the patient. It will have been seen that the nature of the attack often gives but little help. Hæmorrhage, embolism, and thrombosis may all produce a sudden or rapid coma. The more severe and prolonged the coma the greater the probability of hæmorrhage, whereas a pronounced hemiplegia, occurring without coma or with very transient unconsciousness, is more likely to be due to embolism. Age is in favour of hæmorrhage, and youth almost excludes it unless an antecedent embolism is possible; but in persons between forty-five and sixty years of age positive indications in one or other direction are often wanting. In ventricular hæmorrhage evidence of blood will be found in the liquid withdrawn by lumbar puncture.

The associated conditions of hæmorrhage are albuminuria and other evidences of renal disease, or arterial degeneration, with tense and rigid or thickened arteries, high blood pressure, hypertrophied heart, and arcus senilis. Senile changes in the arteries may also be recognised in many cases of thrombosis. Hemiplegia in young subjects free from heart disease is often due to syphilitic arteritis, of which further evidence may be found in the history, or in the Wassermann test. In embolism there is generally a mitral or aortic murmur, or some evidence of dilatation of the left cavities of the heart, which serves as a source of the embolus, or there may be signs of embolism in other parts of the body, such as enlargement or tenderness of the spleen, blood in the urine, the characteristic appearances in the retina, or obstruction of an artery in one of the limbs. Other less common causes of apoplexy with hemiplegia are general paralysis of the insane, hæmorrhage with a cerebral tumour, and infective encephalitis.

**Treatment.**—If cerebral embolism or thrombosis can be certainly recognised, the treatment is similar to that of cerebral hæmorrhage; but venesection must not be thought of. Absolute rest, milk diet, ice to the head if there is pain, and gentle laxatives or enemas if the bowels are confined, are the main indications.

The treatment of the resulting hemiplegia is also the same. In "infantile" cases little can be done; bromides may be given when there are fits (*see* Epilepsy), and impaired gait may be assisted mechanically.

## CEREBRAL MENINGEAL HÆMORRHAGE

Hæmorrhage in connection with the cerebral membranes may be between the bones of the skull and the dura mater (*extra-dural*), within the dura mater but outside the arachnoid membrane (*sub-dural*), or within the sub-arachnoid space (*sub-arachnoid*). Owing to the delicacy of the arachnoid membrane a sub-dural hæmorrhage usually ruptures into the sub-arachnoid space. The causes of hæmorrhage in these situations are (1) direct injury by a fall or blow on the skull, or in the case of infants from birth injury; (2) disease of the cerebral vessels; (3) capillary hæmorrhages in acute infective meningitis (*e.g.* anthrax).

Extra-dural hæmorrhage proceeds mostly from the middle meningeal artery, and is caused by direct injury producing fracture of the skull. The effused blood presses upon the brain, and causes symptoms which vary with the extent and seat of pressure, such as coma and paralysis. These cases are dealt with in works on surgery.

Sub-dural hæmorrhage may also result from direct injury. The hæmorrhage in new-born infants is more often sub-dural, and forms a clot which spreads over the surface of the brain. The blood probably proceeds from meningeal veins entering the longitudinal sinus, which are torn during compression of the cranial bones. In such cases the new-born infant is drowsy and cyanosed, sucks badly or unwillingly, breathes irregularly, or has unilateral or general convulsions. A lumbar puncture shows that there is blood in the cerebro-spinal fluid. Death takes place in a few days, or, if the child survives, it may subsequently suffer from paralysis and rigidity, and the brain undergoes local atrophy and sclerotic changes (*see* p. 838).

Subarachnoid hæmorrhage from disease of the vessels occurs mostly in persons of middle or advanced years, under pathological or ætiological conditions precisely similar to those of intra-cerebral hæmorrhage, but with much less frequency (*see* p. 821). The symptoms are variable. In the case of a large effusion signs of cerebral compression predominate and the patient, after an initial seizure of headache and vomiting, passes rapidly into a state of coma in which he commonly dies. These cases are not clinically distinguishable from those of intra-cerebral hæmorrhage. In other cases, however, the patient recovers from the initial ictus and then displays symptoms due to the irritation of the meninges by the effused blood, which becomes diffused throughout the sub-arachnoid space. Headache, pain, and stiffness in the neck and a positive Kernig's sign, together with irregular pyrexia, are the signs of this condition, and may easily be mistaken for those of an infective meningitis. Examination of the cerebro-spinal fluid, however, reveals the true diagnosis, altered blood being present usually in considerable quantity (*see* p. 736). The optic discs are frequently engorged and sometimes show large retinal and sub-hyaloid hæmorrhages.

Where there is clinical evidence of cerebral compression, as evidenced by deepening coma, withdrawal of large quantities (25–50 c.c.) of spinal fluid should be performed. This should, however, be done carefully, the pressure of the escaping fluid being watched by means of a manometer, since if it is allowed to fall too low there is a danger of provoking fresh hæmorrhage (**5**).

## HÆMORRHAGE INTO THE PONS VAROLII

If hæmorrhage takes place into the central region of the pons, there is generally profound coma, with minutely contracted pupils and complete paralysis of all four limbs, a condition which may easily be mistaken for poisoning by opium. Death is often very rapid, but it may be delayed some hours or three or four days. Convulsions and vomiting are frequent, and sometimes the temperature rises to a great height before death. If a slight hæmorrhage permits of recovery, there

will probably be some degree of paralysis of the limbs, with anæsthesia, irregular facial paralysis, paralysis of the tongue and articulation, and dysphagia.

Hæmorrhage into the lateral regions causes the forms of paralysis previously described (*see* p. 814); and conjugate deviation, if it occurs, is towards the paralysed side (*see* p. 820). If the posterior or upper surface of the pons is involved, sugar or albumin may appear in the urine, and the urine may be abnormally abundant (*polyuria*). The symptoms are, in part, the same as those which result from hæmorrhage into the medulla oblongata, since these two portions of the nervous system are continuous with one another.

## CEREBELLAR HÆMORRHAGE

This is not common, and the symptoms present much variety. In some cases there is a close resemblance to cerebral hæmorrhage, the patient having coma with complete resolution of all the limbs, or with paralysis of one or other side. The hemiplegia may be opposite to the side of the cerebellum involved, and is then regarded as being due to pressure on the pons; vomiting is frequent. Death may occur in a few hours or days, or the patient may recover from the apoplexy and remain hemiplegic.

## ENCEPHALITIS

Whereas in the case of the spinal cord the tendency of older writers was to regard every softening as inflammatory, in the case of the brain every softening was held to be degenerative (pressure or embolism), and suppuration or abscess was the only inflammation described. But different forms of non-suppurative inflammation are now recognised which are probably due in all cases to infective organisms or their toxins, affecting often limited areas of the encephalon, and having corresponding, and more or less distinctive, groups of symptoms.

### ACUTE ENCEPHALITIS

**Ætiology.**—The most important feature in the ætiology of encephalitis appears to be the influence of intoxications and infections. Among the former chronic alcoholic intoxication holds the first place, and is responsible for many of the cases described as acute hæmorrhagic encephalitis, while in some of the other forms influenza has been often observed as an antecedent, less often and with less obvious connection other infectious diseases, such as scarlet fever, measles, pneumonia, diphtheria, syphilis, gonorrhœa, and erysipelas. It has already been stated that in a certain proportion of cases of epidemic poliomyelitis encephalitis is present, due of course to the same infection (*see* p. 98). Injury may be a cause of encephalitis, probably by facilitating infection. The disease lately recognised in epidemic form under the name of *encephalitis lethargica* is of subacute rather than acute onset, and in view of its comparative frequency will be separately described. In the following paragraphs certain rare forms of encephalitis will be described other than those due to the virus of acute poliomyelitis and encephalitis lethargica.

**Pathology.**—The inflammatory change may occur in all parts of the brain, but in some forms it is almost limited to the grey matter (*polio-encephalitis*), whereas in others it has a wider distribution; the grey matter may be that of the cortex or of the basal ganglia, or that which surrounds the third and fourth ventricles, whence it may extend to the grey matter of the cord.

As a result of inflammation the colour of the grey matter becomes grey-red, violet, or dark-brown red, and the white matter becomes reddish, or pink, or grey-red; it is often finely speckled with red points of hæmorrhage. Moreover, the brain is swollen, prominent above the section, infiltrated with serum, moist and shiny,

Microscopically there are hæmorrhages near the vessels, and round-cell infiltration The nerve cells are pale with a swollen nucleus and turbid contents, and later they may atrophy or undergo fatty or calcareous changes; the nerve fibres show swelling and varicosities of the axon. Influenza bacilli, pneumococci, and other organisms have been found in the inflamed area in different cases.

Associated with the hæmorrhagic form of encephalitis there is often pachymeningitis hæmorrhagica, and the pia mater is injected with blood.

**Symptoms.**—These may be described under the following forms:

*Polio-encephalitis acuta hæmorrhagica superior* (Wernicke).—This is a lesion of the grey matter about the third ventricle and of that extending back from this point to the fourth ventricle as low as the sixth nerve nucleus. The onset of the symptoms is generally sudden, and they consist of somnolence or, it may be, unrest, excitement, or delirium, headache, giddiness, vomiting, and stiffness of the neck. There is ocular paralysis, and double ophthalmoplegia is a marked feature, though the sphincter pupillæ and levator palpebræ superioris are spared; and there is optic neuritis or hæmorrhage into the disc. The gait is staggering, reeling, or uncertain, like the ataxy of drinking; the speech is trembling and hesitating, the pulse rapid and the temperature normal or subnormal. Death may take place in from ten to fourteen days.

This form of polio-encephalitis may be accompanied by multiple neuritis.

*Polio-encephalitis acuta inferior.*—Here the lesion affects the lower part of the medulla oblongata, and the symptoms are mainly those of a bulbar paralysis such as have been already described (*see* p. 810)—namely, paralysis of the face, tongue, and palate, with dysarthria and dysphagia, but with more or less extension upwards or downwards in different cases. Such cases are much more often due to infectious disease than to chronic alcoholism, and have been not infrequently observed in the course of influenza epidemics, though the anatomical proof of their nature has often been wanting in cases that recover.

The symptoms of this condition may be combined with those of Wernicke's form, thus constituting a *polio-encephalitis superior et inferior.* In other cases, again, the lesions about the third or fourth ventricle have been combined with an extension to the spinal cord—*polio-encephalo-myelitis.* In these the lesions have not always been symmetrical, nor always confined to the central grey matter, and sensation has been sometimes involved; the cases are generally acute or subacute.

In some cases ataxia of cerebellar type is the chief symptom; and in a patient of Sir Frederick Taylor's, aged four years, who had recently had whooping-cough, there was ataxia of the arms and legs, with tremor of the trunk and head, nystagmus, and indistinct speech. The pathological changes in all these cases are thrombosis of minute vessels followed by perivascular exudation, minute hæmorrhages, and small-celled infiltration. The symptoms are then determined by the position of the lesion, some cases presenting mental changes (frontal area), hemiplegia (motor centres) or ataxia (cerebellum); others, symptoms referable to the cranial nerve centres; and others again, when the cord is involved, producing the familiar symptoms of atrophic spinal paralysis, as seen in infective poliomyelitis.

*Acute primary hæmorrhagic encephalitis* (Strümpell) is another form, which is specially liable to occur after infectious diseases, and has even been called influenza encephalitis. It involves the cerebral hemispheres, but is not confined to the grey matter. It occurs in youth, or even in early childhood, affecting females especially. There is an acute onset, and when it follows influenza there is a distinct interval between the two events. The symptoms are headache, giddiness. nausea, sickness, sleepiness, and prostration, then suddenly a rigor, intense headache, vomiting, occasionally convulsions with rigidity of the neck or limbs, and generally some fever. Either at the onset, or more frequently after the first stage of dulness, occurs hemiplegia, or paralysis of an arm or leg, or

aphasia, or conjugate deviation of the eyes and head. The duration is from three or four days to two or three weeks, and it is mostly, but not always, fatal. It is probable that in some cases of infantile hemiplegia the lesion has been of this kind.

Acute inflammation of the brain also occurs as a part of the acute disseminated encephalo-myelitis already described (*see* p. 774).

**Diagnosis.**—The differential diagnosis of these somewhat rare cases presents considerable difficulties, which must be met by a careful consideration of the symptoms in each. The diseases likely to be confounded with them are myasthenia gravis, tuberculous and other forms of meningitis, thrombosis of the cerebral sinuses, and hysteria.

**Prognosis.**—This is not absolutely unfavourable: acute cases may be rapidly fatal, but recovery, partial or complete, has not infrequently occurred. Sir Frederick Taylor's case recovered in three or four years, and was well at the age of thirty-three.

**Treatment.**—This can be little more than symptomatic. Rest in bed, cold compresses to the head, leeches to the temples or mastoid in severe cases, and purgatives, are the measures which may be employed.

## ENCEPHALITIS LETHARGICA

(*Epidemic Encephalitis*)

This is an acute or subacute non-suppurative inflammatory process affecting the nervous system and meninges in a widespread disseminated fashion. It most commonly involves the structures of the mid-brain, giving rise to symptoms of lethargy and paralysis of the oculomotor nerves.

The disease first appeared in epidemic form in Vienna in 1917; one year later it became widespread throughout the whole of Europe, and since 1919 has been prevalent also in America.

Persons of all ages are liable to the infection. It attacks the population in a widespread random fashion; only in rare instances has more than one case been reported from the same household, and there is no evidence so far to show that it is directly communicable from one person to another. Since its first appearance there have been mild outbreaks in epidemic form every year in the winter months, with the occurrence of sporadic cases during the intervening periods.

Although the disease was not recognised as a specific entity until 1917, small epidemics of a similar nature were reported in Central Europe following the influenza pandemic in 1890, and it is probable that isolated cases have passed unrecognised since that time.

**Ætiology.**—In a number of cases the injection into monkeys or rabbits of nervous material obtained from fatal cases has been followed by death, and the brains of these animals have revealed under the microscope changes which are regarded as characteristic of the disease.

Strauss and Loewe have recently claimed to have discovered that the causal organism is a globular body similar to that of acute poliomyelitis, which may be grown under anaerobic conditions on Noguchi's medium, and may be obtained from the nervous tissues, naso-pharyngeal mucous membrane, blood and cerebro-spinal fluid of persons suffering with the disease. Rabbits are more easily infected than monkeys, which is a point of distinction from poliomyelitis. Their results, however, still await confirmation.

**Morbid Anatomy.**—On macroscopic examination the brain and spinal cord show in the majority of cases little abnormality beyond congestion of the capillaries, which is generally best marked in the grey matter of the basal ganglia, mid-brain, and cerebral cortex. In some instances extravasations of blood are seen either in the subdural or subarachnoid spaces, or in the substance of the mid-brain.

Under the microscope in a case proving fatal within ten days of the onset there may be nothing more to see than intense capillary congestion. Later in the course of the infection, in addition to congestion, there appear other changes characteristic of the disease. The walls of the smaller vessels are seen to be infiltrated with small round cells, and the perivascular spaces filled with them, which leads to the appearance, on cross section of a capillary, of a cuff of lymphocytes surrounding the vessel. Among these small cells are seen occasional large cells containing granular pigment. The nerve cells in most cases show evidence of degeneration, but are attacked in a somewhat random manner, so that in a single nucleus of grey matter only a small proportion of the cells are affected. The changes seen are swelling and excentricity of the nucleus, breaking up of the Nissl granules (chromatolysis), and invasion by small round cells (neuronophagia). These changes are most constantly found in the grey matter of the mid-brain and pons and the basal ganglia, but may also be discovered in the cerebral cortex and the spinal cord. Capillary congestion and round-celled infiltration have also been described in the roots of the cranial and spinal nerves. No constant changes are found in the other organs of the body.

**Symptoms.**—1. *Early Stage.*—These may be divided into those of general intoxication and those referable to localised lesions in the central nervous system. Under the former heading are included pyrexia, malaise, anorexia, vomiting, general pains, and occasionally an erythematous rash. The nervous symptoms are extremely variable, depending upon the localisation of the virus, which may involve any part of the nervous system or its coverings. In the large majority of cases, however, the main incidence is upon the mid-brain, and gives rise to characteristic signs. The disease as a rule commences insidiously with a feeling of lethargy and drowsiness. With this are frequently associated minor signs of meningeal irritation, such as headache (often suboccipital) and stiffness of the neck. In the course of a day or two the nuclei of the third nerves become affected, leading to dimness of vision for near objects due to paralysis of accommodation, ptosis, and weakness of the ocular movements; at this stage complaint is sometimes made of diplopia. Subsequently the drowsiness progresses, until the patient appears to be continually in a stuporous condition, from which he must be roused to take his food. When awakened, however, his mental condition is quite clear, and he is able to answer questions in a rational manner. At this period of the illness the picture presented is a very striking one. The face appears to be smoothed out and expressionless, and this, combined with the ptosis, gives rise to an appearance of inanimate stupidity, against which the lucidity of the mental state often stands out in remarkable contrast. Careful observation will often reveal the occurrence of irregular twitchings at this stage, affecting individual muscles or their fasciculi in random fashion, but seldom powerful enough to move the limbs. These may involve any part of the musculature, including that of the trunk and face. The tongue is dry and coated, and frequently tremulous. The patient may complain occasionally of pains in the limbs.

The temperature at the onset is raised, and oscillates in an irregular manner between 99° and 101°, with corresponding alterations in the pulse rate. In a fatal case the temperature as a rule rises, the stupor deepens into coma, incontinence of fæces and urine follows, and death occurs in from ten to twenty-one days from the onset.

Physical examination when the disease is well developed may reveal additional signs. Thus the pupils are frequently unequal, and respond sluggishly to light. If the patient can be induced to co-operate in the examination, it is usually found that the pupillary reaction in accommodation is lost, and all ocular movements depending upon the integrity of the third nerves are weak or absent. Nystagmus is often present; not infrequently there is complete paralysis of all ocular movements. The Argyll-Robertson pupil may occur.

The other cranial nerves are not affected as a rule. No anomalies of sensation

are discovered beyond the complaints of pain already referred to. Examination of the motor system reveals general muscular weakness, combined sometimes with plastic rigidity, so that the limb will remain almost indefinitely in any attitude in which it is placed, however bizarre (catatonia). The reflexes vary from case to case; the tendon reflexes may remain unaffected; they may be increased; more frequently they are abolished; the abdominal reflexes seldom show any abnormality; the plantar responses are as a rule flexor.

If recovery ensues, the temperature begins to fall during the third week from the onset, and the patient gradually awakens from his stuporous condition. With the end of the third week the period of pyrexia terminates, but in a severe case lethargy persists, the cranial nerve palsies clear up slowly, and it may be three months or more before the normal functions of the nervous system are again fully established, if indeed complete recovery is attained.

Since the symptoms in a case of nervous disease depend almost entirely upon the localisation of the infective process, and since in the disease under consideration the distribution of the virus may be widespread, one must be prepared to find almost any combination of signs and symptoms in the course of an epidemic: with the extension of the inflammatory process from one part to another the appearance of a single case may change from time to time, and among half a dozen cases there may be no outstanding nervous symptom common to all. Thus, with regard to the onset, in addition to the common type already described there may be instances in which meningeal symptoms at first predominate; in other cases restlessness, irritability, or even a noisy delirium may take the place of the more usual lethargy; the illness may begin acutely with an apoplectiform seizure, or the picture may be entirely dominated by the presence of muscular twitchings (the so-called myoclonic type), usually associated with delirium. In another group of cases the disease may be ushered in by severe neuralgic pains of peripheral nerve or root distribution; these may be followed by wasting and weakness of isolated groups of muscles. Or, again, in some instances absence of tendon jerks and subjective sensations of numbness in the extremities may give rise to the appearance of polyneuritis.

This account by no means exhausts the symptomatology of the disease. Among signs of minor importance may be mentioned dysarthria, dysphagia, paroxysms of dyspnœa, hiccough, excessive salivation, excessive sweating, sensations of choking or stifling, retention of urine, and occasionally spasmodic contracture of the jaw muscles.

The extent and duration of pyrexia are variable; the temperature is probably always raised during the initial stages, but on account of the insidious nature of the onset may have subsided before the patient comes under medical observation. In mild cases the signs of involvement of the nervous system are often fleeting, and special inquiry is necessary to elicit a history of transient diplopia, temporary paralysis of accommodation, pain and stiffness in the back of the neck, neuralgic pains or muscular twitchings.

The cerebro-spinal fluid is always clear; in about one half of the cases it shows no abnormality by the time it is examined, but in the other half there is an increase in the number of lymphocytes, which varies from ten to eighty cells per cubic millimetre; this lymphocytosis is more commonly observed in cases in which lumbar puncture is performed early in the course of the disease. There is, as a rule, no increase in the protein content, but the colloidal benzoin test may give curves of the paretic or luetic types. The Wassermann reaction in blood and cerebro-spinal fluid is negative.

2. *Late Stage.*—In a large proportion of the cases after the acute stage has subsided further symptoms develop, probably as the result of a smouldering process of infection, which may continue to involve fresh areas of the brain for months or even years after the onset. In some cases the patient passes directly from the early into the late stage of the disease, but more often there is a latent

interval during which he may appear to have made a satisfactory recovery—and this interval may be of several months' duration. The severity of his late symptoms bears no relation to that of the early stage, so that cases of a mild or ambulatory nature, which may not in the first place have been recognised, are often subsequently the victims of serious physical or mental disability. The commoner symptoms of the late stage may be grouped under the headings of (1) Weakness, rigidity and tremor of the paralysis agitans type, (2) Involuntary movements, (3) Mental changes.

(1) Symptoms resembling those of paralysis agitans probably develop in a high proportion of the cases which survive the early stage of the illness. The most noticeable feature as a rule is the stiffness of the facial muscles with consequent loss of expression; the rigidity often involves also the muscles of trunk and limbs; the gait is then slow and shuffling, with bent back and bowed head, the arms are held stiffly with elbows slightly abducted and fingers semi-flexed. Automatic movements are diminished or lost, and the patient has difficulty in adjusting his centre of gravity, and his balance therefore is easily upset. Inhibition of the blinking reflex adds to the staring facial expression, and of the swallowing reflex to the dribbling of saliva. The voice is frequently monotonous and speech slow. Tremor of the paralysis agitans type may also be present, but is usually slight in comparison with the rigidity.

(2) Involuntary movements of all kinds may occur, but the proportion of cases in which they are a striking feature is small. Tremor of the paralysis agitans type has already been mentioned. Choreiform and athetoid movements are also seen. Perhaps the commonest type, however, is that in which there are rhythmic movements of relatively large amplitude and slow rate involving the musculature of the proximal joints (shoulder and hip), often affecting a single limb or the arm and leg on one side of the body. These are unlike those seen in any other known disease.

(3) Mental impairment of some degree is an almost constant sequel of the disease. In adults it is manifest in slight cases as an increased tendency to fatigue and diminished emotional control; in more severe cases symptoms of a neurasthenic character develop with constant complaint of headache, feelings of inadequacy and morbid anxiety. In the cases of the paralysis agitans type, on the other hand, profound apathy is often present. Children are often more seriously affected, the disease in them leading to deterioration of character without any corresponding impairment of intellect. They are apt, therefore, to develop habits of lying, stealing and violence, which make it difficult to control them at home and lead not infrequently to the police court.

Insomnia at all ages is a common and distressing sequel. The patient is drowsy and apathetic during the day, but at night time becomes restless and agitated, often excited, and gets off to sleep only in the early morning.

**Diagnosis.**—In cases of the common type beginning insidiously with drowsiness, low fever, and ocular palsies, this is comparatively easy, but may be a matter of extreme difficulty when the incidence of the infection is more widespread. The diagnosis of cerebral abscess has to be considered, and, with this possibility in view, the ears should be examined for signs of suppurative otitis. Cerebral tumours also, especially if situated in one of the silent areas of the brain, may produce signs resembling those of encephalitis lethargica, but in this case examination of the optic discs may clear up the diagnosis, papilloedema being seldom absent in cases of tumour, rarely present in encephalitis.

Cases in which the illness is ushered in with meningeal symptoms may simulate meningococcal or tuberculous meningitis, but the neck stiffness is hardly ever so important a feature of encephalitis as in these illnesses. In the rare instances in which the onset is apoplectiform, the condition can hardly be differentiated from that caused by a cerebral hæmorrhage or thrombosis.

In the later stages, after the decline of pyrexia, the presence of signs of wide-

spread involvement of the nervous system may suggest the possibility of syphilis, when the Wassermann reaction in blood and cerebro-spinal fluid will be of use in settling the point. Cytological and bacteriological examination of the cerebro-spinal fluid is always of value in making the differential diagnosis. Finally, there are instances in which it may be impossible to discover at the bedside whether a given case should be regarded as one of encephalitis lethargica with involvement of the spinal cord or acute poliomyelitis with involvement of the brain and brain stem.

**Prognosis.**—The death rate appears to be about 20 per cent. if note is taken of the abortive cases. The notification returns show a considerably higher rate of mortality. The large majority of deaths occur within the first three weeks of the illness, and after this period the prognosis as regards life is good.

Signs of ill omen are a rising temperature (above 102·5°), deepening of stupor into coma, or noisy delirium with extensive muscular twitchings (the acute myoclonic form).

In the cases which recover the cranial nerve palsies as a rule clear up, relatively soon leaving, perhaps, inequality of the pupils with sluggish contraction in accommodation. Lethargy, weakness and slowness of movement may persist for many weeks and develop gradually into the clinical semblance of paralysis agitans.

Symptoms of the order already described as belonging to the late stage may continue to make their appearance for as long as two or three years after the onset, but it may be said as a rule that a patient who has recovered from the initial attack and has thereafter remained completely free from fresh symptoms for six months is unlikely to have further trouble.

Of the late symptoms the paralysis agitans group show a tendency to progress slowly; they eventually come to a standstill, and there may be a slight remission, but the patient is left considerably disabled. The involuntary movements develop slowly (over a period of months) to their climax, and as gradually recede, often with but slight residuum. The mental changes, both in adults and children, though there may be some remission, appear for the most part to be of a permanent nature.

**Treatment.**—There is no specific treatment for the disease. Urotropin in full doses should be given in view of its possible antiseptic action, and symptoms should be attended to as they arise. In most cases the patient can be roused to take his food, which should be confined to a light and easily assimilable diet with plenty of fluids. Occasionally the stupor is so deep that tube feeding has to be resorted to, and this may also be rendered necessary by dysphagia, due to glosso-pharyngeal paralysis apart from stupor. The method of choice is by the nasal tube, through which an ample diet may be given in the shape of milk, beaten-up eggs, malt, and orange juice. The bladder should be watched carefully, since retention with overflow may occur, and if neglected lead to cystitis. In prolonged cases there is, as a rule, much emaciation, with probable development of bedsores, so that it is wise to have the patient upon a water bed if the course of the illness appears likely to be protracted. For the insomnia, which is sometimes a distressing sequela, hot baths (for one hour before bedtime) and various drugs may be tried, of which the most useful are paraldehyde in full doses and the veronal compounds. For rigidity of the limbs after the acute stage has subsided passive movements should be carried out daily, preferably after a hot bath.

In view of the grave risk of a smouldering infection leading to further symptoms, convalescence should wherever possible be prolonged, and the patient advised not to resume his full round of activities for a period of six months after the progress of symptoms has ceased. During this period regular exercise should be prescribed for body and mind, but this should stop short of fatigue.

**Prevention.**—The Ministry of Health makes the following recommendations (1924):—

"The other occupants of a house in which a case of encephalitis lethargica has occurred, or is being treated, may be assured that the disease is one of low infectivity, and that very slight risk is run by association with the patient. At the same time it is desirable that such association should be limited to what is necessary for proper care and nursing, and the patient should be well isolated in a separate room.

"School children in the affected household may be kept from school, as a precautionary measure, for three weeks after the isolation of the patient. There is no necessity to place restriction on the movements of other occupants provided they are frequently examined and remain well. Those in contact with the case, however, may be advised to use antiseptic nasal sprays or douches and to gargle the throat with solutions such as those advised for influenza.

"For example, any of the following:

(1) One per cent. solution of peroxide of hydrogen.
(2) A solution of permanganate of potash, 1 in 5,000, in 0·8 per cent. solution of chloride of sodium (common salt).
(3) Liquor sodæ chlorinatæ, 0·5 per cent.

"These solutions can be used as ordinary throat gargles or snuffed up the nostrils, or applied by an efficient spray.

"Any persons in the infected household who suffer from sore throat or other symptoms suggesting an abortive attack should be treated from this point of view and isolated as far as possible until they have recovered.

"The sick room should be thoroughly cleansed and disinfected at the end of the illness."

### CHRONIC ENCEPHALITIS

Of this as a separate disease little can be said. Wilks described some cases of *diffuse sclerosis*, or induration of the brain, which were probably inflammatory in origin (Guy's Hospital Reports, 3rd ser. vol. xxii., 1877). In one the convolutions were flattened and compressed; on section the white matter was firm, hard, grey in colour, and encroached on the normal grey matter, pushing the convolutions away from one another. In another there was *meningo-cerebritis*, and the brain substance was tougher than normal. In a third case the membranes were also inflamed; the left hemisphere presented some red softening, but the right was hard and brick-red in colour, the convolutions were swollen to twice their size, and the cortical was scarcely distinguishable from the medullary matter. Under the microscope the tissue showed an "absence of anything like nerve structures, and appeared to consist mostly of vessels and a dimly fibrillated substance." In the case of an infant, recorded by Fagge, the brain was indurated, the white matter was of a yellowish colour, the grey matter appeared normal, but the pia mater was firmly adherent. The only histological change noted was a slight excess in the cells of the neuroglia. In other cases atrophy of nerve fibres and increase of the neuroglia are described.

The early symptoms in Wilks' cases were stupidity, inability to speak, indifference to food, loss of sight, hearing, and memory, tingling and pain in the extremities, followed by drowsiness and unconsciousness, with ill-defined paralysis of one or other side of the body.

Chronic encephalitis, partial and probably secondary to degenerative changes, occurs in *disseminated sclerosis* and in *general paralysis of the insane*.

## ABSCESS OF THE BRAIN

**Ætiology and Pathology.**—In the great majority of cases abscess can be shown to be the result of direct infection by pyogenic organisms. It often arises in consequence of chronic suppurative disease of the ear. For instance, otitis occurs during convalescence from scarlatina, the membrana tympani is perforated,

and there is a discharge of pus, which may continue for months or years. Ultimately, and sometimes without any apparent cause, the symptoms of cerebral abscess develop. In some such cases, the bone forming the wall of the tympanum is necrosed, the dura mater over it inflames or sloughs, and the pia mater becomes adherent; in others the bone may be healthy, and the infection seems to have been carried by channels in the bone to the interior of the skull. Even where the membranes are directly inflamed, the abscess may not be in immediate contact with them. The pus from the ear is sometimes fœtid, but not necessarily so. Another cause of abscess is disease of the nasal fossæ or frontal sinuses, and it may follow any other lesion involving the cranial bones, such as direct injury, syphilitic caries or necrosis, or tumour of the bones.

Cerebral abscesses occur in general pyæmia, and in some pyæmic cases patches of red softening have occurred side by side with developed abscesses. Another source of suppuration is occasionally seen in inflammatory lesions in the lungs, such, for instance, as tuberculosis, pneumonia, gangrene, empyema, and especially bronchiectasis. It is possible here that particles of thrombus are carried from the lungs into the general circulation, and so to the brain. In some cases of cerebral embolism from infective endocarditis the softened tissue breaks down into a fluid indistinguishable from pus.

The position of the abscess is determined, to a certain extent, by its cause. Thus, if due to otitis, it is mostly in the temporal lobe; or in the cerebellum, if the mastoid cells are especially involved. Figures given by A. Starr show that abscess is nearly three times as frequent in the former situation as in the latter. Disease of the nose may give rise to abscess in the frontal lobes. Generally there is only a single abscess, but in pyæmia there are often two or more, situated indiscriminately. They are, however, commonly located in the white matter of the hemispheres, or of the cerebellum, and rarely in the grey matter, or at the base of the brain. They vary in size, and may reach 2 inches in diameter. Recent abscesses have a shreddy wall; those which are older have a definite and often thick cyst wall or capsule, composed of fibrillated, if not fibrous, tissue. The pus is mostly pale green, viscid, and acid in reaction; but in long-standing cases it becomes more mucoid still, alkaline, and of a bright green colour. Sometimes, especially when due to bone disease, it may be extremely offensive. The brain tissue outside the abscess may be softened. Though in many fatal cases the abscess is found intact, it may rupture on the surface and set up meningitis, or into the lateral ventricles, or it may form a communication, through diseased bone, with the tympanum, and discharge externally (*otorrhœa cerebralis*).

**Symptoms.**—These are often extremely obscure. The most constant is *pain*, of a continuous dull aching character, or more severe, so that the patient holds his head with his hands, or bores his head into the pillow, or cries out constantly. Exacerbations of the pain occur from time to time. The seat of pain often, but not always, corresponds to the position of the abscess. Sometimes there is elevation of the temperature, sometimes *rigors*, either occasionally or following with such regularity as to suggest malaria, and sometimes profuse perspiration; but the temperature may be very nearly normal. Convulsions and vomiting may occur. Papilloedema is much less frequently present than in cases of tumour. Alterations in manner, dulness, listlessness, loss of memory, and emaciation are also sometimes observed. Drowsiness is a common symptom. It may occur early and progress gradually into coma, or there may be striking intermissions during which the mental condition is normal for several hours. The common seat of the abscess renders localising symptoms on the side of the motor tract or nerve trunks rather improbable; but there may be ill-defined hemiplegia, or, if the situation is in the left temporal lobe, aphasia of a kind in which the patient cannot give the name of an object shown to him, or cannot call to mind the object of which the name is mentioned (word forgetfulness), or uncinate attacks may occur (*see* p. 814). An abscess in the cerebellum may cause symptoms

of a destructive lesion; and if cranial nerves are pressed upon, the corresponding paralysis will occur.

The duration of the symptoms is very variable; they may last for months, or they may end fatally in a few weeks. Death is often quite rapid, the patient becoming delirious, or quickly drowsy and comatose. Respiration may cease before the pulse, as in the sudden deaths from cerebral tumour.

**Diagnosis.**—The diagnosis of abscess of the brain is not always easy.

The most important factor in diagnosis is the presence of a primary cause, and chronic discharge from the ear is the most frequent of these. It must not, however, be too hastily assumed that acute pains in the head and pyrexia, occurring in a patient the subject of chronic otitis, are due to cerebral abscess, even though rigors and optic neuritis be present as well. For otitis may produce, besides abscess of the brain, subdural abscess, or meningitis, or suppuration of the mastoid cells with or without thrombosis of the adjacent veins and sinuses, or encephalitis. All of these are accompanied by severe head pains and fever; with mastoid abscess there may also be rigors. Moreover, in mastoid suppuration there is sometimes double optic neuritis, with an entire absence of meningitis or of abscess, as proved by post-mortem examination, and by recovery after simply trephining the mastoid cells. The cause is probably thrombosis of some cerebral sinuses. This warning, however, applies almost more to meningitis than to abscess, since optic neuritis is more frequent in the former. Suppurative meningitis—the form most likely to be confounded with abscess, since they have a common origin—is more rapid in its course, and is more likely to be accompanied by paralysis and fits: the temperature is more uniformly high, and shivering is absent; and lumbar puncture may show micro-organisms and polymorphonuclear leucocytes in the cerebro-spinal fluid.

**Prognosis.**—Many cases have been cured by evacuation of the abscess; without the help of surgery recovery cannot be expected.

**Treatment.**—Where an abscess can with reasonable certainty be recognised, and its locality accurately determined, the attempt to evacuate the pus should be made.

For exploratory purposes bone may be removed with the trephine or with the gouge, and a fine trocar can then be introduced. In cases arising in connection with diseases of the ear, it is desirable that before trephining a thorough exploration and antiseptic treatment of the tympanum and mastoid cells should be undertaken, both to exclude the possibility of the symptoms being entirely due to these parts, and also to minimise the risk of meningitis during the longer operation.

Apart from surgical interference, the treatment of abscess of the brain must be purely symptomatic; the relief of pain may be attempted by local anodynes, by ice to the head, and by bromide of potassium, aspirin, or even morphia, internally.

## INFANTILE CEREBRAL DIPLEGIA

(*Infantile Spastic Paraplegia, Diplegia Spastica, Congenital Spastic Paraplegia, Birth Palsy*)

This title includes all cases of bilateral spastic weakness of the extremities, of cerebral origin, occurring in infancy or early childhood.

These cases may be further divided into two groups according to their pathological and clinical features (**6**).

*Group I.* comprises those cases in which the symptoms are due to lesions of both cerebral hemispheres resulting from injury or infection, at or after birth. Of such causes the commonest are meningeal or cerebral hæmorrhages as the result of birth injury, and acute encephalitis (*see* p. 829).

The brain in such instances provides post-mortem evidence of injury or inflammation in the shape of meningeal scarring and cyst formation in the traumatic cases, and perivascular scarring in those of infective origin.

Clinically in many cases no definite history of injury or infection may be obtained, but a series of convulsions occurring shortly after birth is suggestive of meningeal hæmorrhage, or a history may be elicited of an acute febrile illness in infancy.

The symptoms are as a rule asymmetrical, one limb or one side of the body being more involved than the other. Actually the paralysis often escapes notice until the child proves backward in sitting up, walking and talking.

The damage in these cases is of an irreparable nature, but a great deal may be done for these children by means of carefully planned remedial exercises and drill. There is almost always some degree of mental impairment, which is irremediable. Nevertheless these children are much more educable than may appear at first sight and will usually repay patient instruction.

*Group II.* comprises the majority of cases of cerebral diplegia, and consists of cases in which as the result of some obscure cause acting as a rule in utero, but sometimes after birth, the development of the cortical cells is arrested or retarded.

In a severe case of this kind the brain is small and atrophic, with wide sulci and wrinkled convolutions, and is sometimes compared to a walnut kernel. Microscopic examination reveals a diminution in the numbers of the cortical nerve cells with secondary overgrowth of neuroglia. The process is symmetrical in the two hemispheres and frequently includes the cerebellum. The optic nerves also are frequently degenerated.

Clinically these cases are distinguished by the remarkable symmetry of the spasticity and weakness. Nothing may be noticed at birth, although a history is often obtained of premature, sluggish, or precipitate labour, which is possibly due to the same cause affecting the reproductive mechanism of the mother as is responsible for the disease in the child. After birth the child may suck feebly, but often nothing is noticed until he proves backward in sitting up and slow at learning to walk, owing to the stiffness of the lower limbs. Ultimately the condition is much like that seen in the spastic paraplegia of adults. The limbs are extended and rigid with increased tendon jerks and persistent extensor plantar responses. Sometimes spasm of the adductors is extreme and the legs are crossed over one another, in spite of which the child manages to walk—*crossed leg progression*. The arms are never as rigid as the legs, and may escape entirely. The picture may be complicated by the addition of cerebellar ataxia and nystagmus, and optic atrophy is of frequent occurrence. Some degree of mental impairment is usually present.

In the cases which are of congenital origin (and these are in the majority), although the development of some nerve cells would seem to have been arrested in other cells it is merely retarded, and as the result of suitable treatment by means of remedial exercises and patient education considerable improvement may be expected. In a few instances, however, the disease process is still active and progressive and may end in complete paralysis and death; in these the optic nerve and retina are as a rule involved. When the disease commences in infancy or childhood it is usually slowly progressive, but may after some years come to a standstill with some degree of improvement.

**Treatment.**—In cases of both groups this is the same, and must consist of regular exercises and drill with suitable educative measures. Deformities must be treated by manipulation, splinting, and tenotomies when necessary. A small proportion of cases of Group II. are of syphilitic origin and should be treated accordingly.

### HEREDITARY CEREBELLAR ATAXY

This disease affects members of the same family, and is hereditarily transmitted. The symptoms generally set in after puberty, and only slowly progress. The essential feature is the reeling, unsteady gait characteristic of cerebellar disease; in addition, chorea-like movements, impaired articulation and pallor

of the optic discs. The cerebellum has been found in a condition of atrophy without sclerosis. The disease is distinguished from Friedreich's ataxy by the later age of onset, the presence of knee jerks, and the absence of trophic disturbances and spinal deformities.

### PROGRESSIVE LENTICULAR DEGENERATION

This is a rare disease of the corpus striatum, which has been fully described by Dr. Kinnier Wilson. Most of the patients have been young, and it has affected more than one member of the same family. Its onset is gradual, and the first symptoms observed have been clumsy movements, trembling of the limbs, stiffness of gait, impaired articulation, and sometimes defective memory or mental feebleness. In its completer development in the course of some months, the patient shows generalised tremor of the upper and lower limbs and sometimes of the face and trunk, increased by voluntary effort; muscular rigidity, spasticity, with spasmodic contractions and, later, contractures; dysarthria; dysphagia; emaciation; spasmodic movements and emotionalism. Together with the tremor, spasticity and contracture there is some muscular weakness; but there is very little affection of sensation, and no definite changes in the reflexes, which are active, but not exaggerated. In many cases the mental state is altered after a time. The patients have been described as dull, stupid, untidy, silly, childish, slow in answering questions, easily provoked to laughter; and towards the end they are often docile, childish, and easily amused. In the later stages, the arms are fixed to the sides of the chest by contraction of all the flexors, there is often a vacant smile on the face, the mouth is wide open, and saliva is constantly flowing from it.

The disease is progressive and fatal in from two to seven years.

**Pathology.**—The essential, and perhaps the only, change in the brain is degeneration of the lenticular nucleus of the corpus striatum, most pronounced in the putamen, and less in the globus pallidus. This shows itself in different degrees as discoloration and sponginess, shrinkage, atrophy, or complete disintegration and excavation. Microscopically there is no small cell infiltration, but there is glial overgrowth, disintegrating and breaking down, with increase of glial nuclei and the presence of large mono-nuclear phagocytes containing granular *débris*. The optic thalamus and other grey nuclei are not affected, and the pons Varolii, medulla oblongata, and spinal cord are healthy.

A remarkable feature in these cases is that the *liver*, though showing no signs of symptoms during life, is always found after death to be the subject of *cirrhosis*. This is sometimes multilobular, and sometimes of a mixed type.

In explaining the relation of the symptoms to the cerebral lesion, Dr. Wilson attributes the tremors to the loss or abolition of the steadying influence which, he believes, the corpus striatum exercises upon the cells of the anterior cornua, by means of an extra-pyramidal efferent path (lenticulo-rubro-spinal), which passes from the lenticular nucleus through the red nucleus to the anterior cornua of the spinal cord.

Or the steadying influence may possibly be exerted by means of strio-thalamic fibres upon the thalamus, and so upon the motor cortex.

From the constant presence of cirrhosis of the liver in these cases it may be considered highly probable that a toxin is the cause of the lenticular degeneration.

## PARALYSIS AGITANS

*(Shaking Palsy, Parkinson's Disease)*

This disease consists, in its fully developed form, of rhythmical contractions of the muscles of the limbs associated with weakness and rigidity.

**Ætiology.**—It is a disease of advanced life, rarely occurring before the age

of forty-five years, but, on the other hand, not often commencing after the age of sixty-five. It occurs in men twice as often as in women. It cannot always be traced to any definite cause; emotion, fright, injuries, acute diseases, and exposure to cold have been the determining factors in some cases.

**Pathology.**—Recent researches have indicated that the symptoms of paralysis agitans may be produced by lesions of certain areas of the brain, and that these lesions may be the result of various pathological processes. The exact site of the areas affected is a little uncertain. On the one hand, symptoms in many ways resembling those of paralysis agitans have been observed in association with progressive degeneration of the lenticular nuclei (*see* p. 840), and in typical cases of juvenile paralysis agitans Ramsay Hunt claims to have found atrophy of the motor cells in the globus pallidus. Many Continental observers, however, point to changes in the substantia nigra of the mid-brain as being the most important pathological features. The truth probably is that we are dealing with an *affection of a system of extra-pyramidal motor fibres* in which cell stations and neuronic pathways both in the corpus striatum and the mid-brain play an important part. As to the *nature* of the pathological process, there can be no doubt that the virus of encephalitis lethargica may be responsible in a certain number of cases, especially those occurring in young people (*see* p. 835). In progressive lenticular degeneration the association with cirrhosis of the liver suggests some unknown toxic factor as the primary cause. The common form of the disease beginning in old people may possibly be due to arteriosclerotic changes in the brain, or to a system degeneration akin to that seen in progressive muscular atrophy. But of this at present we have no certain knowledge.

**Symptoms.**—In some cases the first symptom is the tremor, after which the disease is named; in others rigidity is observed before any tremor has occurred; but ultimately they are both present.

*Tremor.*—In the former case, it is noticed that one hand and arm are the subjects of a tremulous movement, due to rhythmical contractions of antagonistic muscles. The movement is most marked in the hand; the fingers are generally flexed, with the thumb resting against the forefinger; and the constant slight flexion and extension of the fingers and thumb produces a movement like that required for rolling pills. Similar slight movements of flexion and extension occur at the wrist and elbow joints. After the tremor has existed for some time in one arm it generally spreads to the leg of the same side, and then in succession to the arm and leg of the opposite side. The trunk may also be affected, though it is not always easy to say how much the tremor is due to the movements in the legs; and, finally, in some cases, there is a slight movement of the head. Occasionally even the muscles of the jaw and tongue, but very rarely those of the face, are affected. These movements vary in extent; in rapidity they range between $4\frac{1}{2}$ and 7 to the second. As a rule the movements continue even during rest; thus, if the patient sits with the arm resting on the knee, both the leg and the hand and arm will continue to tremble. In early cases, however, support may check the tremor for a time, and in advanced cases, with the rigidity to be presently described, the tremor may only occur on movement. By voluntary efforts fixing the limb the tremor may also for a time be stopped, and it ceases during sleep.

*Rigidity.*—This differs from that met with in spastic conditions following pyramidal lesions in that the rigidity is more evenly spread, affecting both extensor and flexor groups, and more evenly maintained in opposition to passive movements. When it is the first symptom it may be observed that the thumb and forefinger are rigid without any tremor: rigidity extends to other parts, and tremor is developed later. In a case which has begun with tremor in one limb, the rigidity may appear first in another limb, and be followed by trembling. The muscular weakness is shown by deficient power of grasp, and fatigue on exertion, so that in the early stages, for instance, a man may find difficulty in

buttoning up his collar, and a woman in doing her hair. The rigidity imposes a characteristic posture, which is most marked when the patient is standing. The head and body are bent forwards; the elbows are flexed nearly to a right angle, and stand out a little from the side; the hands are in the position above described; and the legs are slightly bent at the knees. The gait in fully developed cases is very peculiar: the patient rises from his seat slowly and with apparent difficulty, and his first steps are hesitating; but soon his movements become quicker and quicker, he seems with each step to be trying to prevent a fall, and ultimately, unless stopped, he may actually fall forward to the ground. Some patients when gently pushed backwards are unable to stop themselves, and continue to walk backwards until they meet an obstacle or fall. The terms *festination* and *propulsion* have been used to describe the forward tendency; *retropulsion* and *retrogression*, the backward movement. It has been noticed as an early symptom that the toes are curled under the foot when the patient begins to walk (P. Stewart). The face has a characteristic and fixed expression, due to the rigid condition of the facial muscles (Parkinsonian mask), and this may be a comparatively early symptom of the disease. Speech may be similarly slow at first, and afterwards rapid; it is often high-pitched or thick and feeble. Otherwise all movements tend to be slow on account of the rigidity; for instance, the patients turn with difficulty. The muscles are not hypertrophied by their excessive action, and only in late cases with long-continued rigidity do they present some atrophy. The reflexes and electrical reactions are generally normal. Some subjective sensations are often experienced, such as dull aching pains, sense of fatigue, or a feeling of restlessness, and especially a sense of great heat, which is often accompanied by free perspiration; while a flush on the cheek of the patient shows vasomotor weakness.

The disease is chronic and progressive, yet its course may be very slow, two or three years perhaps elapsing between the affection of one and another limb. Probably a well-marked case never recovers, but it is fatal only through bedsores or exhaustion in the extreme cases: intercurrent disease—*e.g.* of the lungs—terminates others.

**Diagnosis.**—There is but little difficulty in recognising paralysis agitans when tremor is present. *Senile tremor* is distinguished from it by the constant trembling of the head. The movements also are finer, both arms are often involved together, and there is no rigidity; it comes on later in life. In *disseminated sclerosis*, which occurs at an earlier age, the movements are wider, more irregular, and brought on by voluntary efforts only, the head is implicated, nystagmus is present, and the speech is scanning or staccato. In the cases which begin with rigidity and weakness the diagnosis may be difficult in the early stages. Especially if the symptoms are confined to the limbs of one side, there is a resemblance to a hemiplegia of pyramidal origin, but this may be excluded by the absence of alterations in the reflexes, and by the presence of the mask-like expression, which even when present in slight degree is often characteristic. Cases of slight double hemiplegia may be distinguished by the bulbar symptoms and mental changes, as well as by minor alterations in the reflexes.

**Treatment.**—This is most unsatisfactory. Business worry and excitement should, of course, be avoided. Various sedative drugs may be employed; and of these the most useful is hyoscyamine hydrobromide, which may be given by the mouth in combination with a bromide mixture, beginning with $\frac{1}{200}$ grain three times daily, and being increased until improvement is observed, with due regard to the possibility of poisoning. Or a subcutaneous injection of $\frac{1}{200}$ grain to $\frac{1}{50}$ grain may be given at bed-time to induce sleep. The patient is nearly always at his best in the early morning, and may even at this time be free from tremors. Every effort should be made to spare him all unnecessary fatigue from the commencement of the day, especially in the business of dressing. These patients also frequently need help in changing their position, and especially is this the case when

they are in bed at night; attention to this point may be of assistance in securing sleep.

Massage and passive movements may be of some use for the rigidity, especially if given after a hot bath.

## MENINGITIS

In the cerebral as in the spinal meninges, we have to distinguish an inflammation of the dura mater, or *pachymeningitis*, and inflammation of the pia mater, or *leptomeningitis*.

The pia mater appears to be much more subject than the dura mater to the influence of micro-organisms, and to these bodies nearly all forms of lepto-meningitis can be traced.

The organisms most often concerned are—

1. The *pyogenic organisms* (streptococci, staphylococci) which may invade the meninges in pyæmia, septicæmia, erysipelas, perhaps small-pox, after injury to, and operations on, the cranial bones, and in diseases of the ear, nose, and frontal sinuses; these cause a *suppurative* meningitis.

2. *Tubercle bacilli*, secondary to a tuberculous focus, either in the brain itself or in some other part of the body.

3. The *pneumococcus*, often in association with pneumonia, as part of malignant endocarditis, or as a primary affection.

4. The *meningococcus*, or *Diplococcus intracellularis* of Weichselbaum, which causes cerebro-spinal fever, or epidemic cerebro-spinal meningitis, including its posterior basal variety.

5. *Syphilis* is a frequent cause of meningitis, which is, however, generally subacute or chronic in its course.

6. The bacilli of *influenza*, *typhoid fever*, the *gonococcus*, the *Bacillus coli communis*, and other organisms have been found in some cases.

The results of bacterial invasion are seen in the effusion of lymph or of pus on the surface of the brain, often with an increase in quantity of the cerebro-spinal fluid.

There is a general resemblance to what has already been described under the head of cerebro-spinal fever and tuberculous meningitis.

### SUPPURATIVE MENINGITIS

**Ætiology.**—If we exclude cases of cerebro-spinal fever, a purulent meningitis is in the majority of cases the result of invasion by the pyogenic or septic organisms, and arises in circumstances similar to those which lead to abscess of the brain; that is, its common cause is a focus of disease in the immediate neighbourhood. Thus it may follow injuries to the head, or be set up by extension of inflammation in neighbouring parts, such as otitis media, mastoid suppuration, disease of the nasal cavities, syphilitic caries or necrosis of the skull, suppurative phlebitis, or abscess of the brain. But it occurs also as a complication in some general diseases of an acute, febrile, or infective nature—pyæmia, septicæmia, malignant endocarditis, enteric fever, small-pox, and scarlet fever. A pneumococcal meningitis is also purulent (*see* p. 845).

**Pathology.**—The inflammation chiefly affects the pia mater and arachnoid (*leptomeningitis*). When it has spread from a diseased bone of the skull, the dura mater itself may show localised inflammation, but the extension of the disease over the brain is by means of the other membranes. Commonly the convex surface of the brain presents a more or less extensive layer of bright yellow or green pus, which may be on both sides, or confined to one side, the side of the lesion in secondary cases. The pus frequently follows the course of the larger vessels, and dips down with the pia mater into the sulci. Though mostly affecting the upper surface of the hemispheres (meningitis of the convexity), it may extend

to the base, or into the spinal canal. The brain tissue beneath it is commonly softened, and may present ecchymoses or minute abscesses.

**Symptoms.**—While there is a general resemblance to the symptoms of tuberculous meningitis, the course of acute meningitis is usually much more rapid, and there is much diversity as to the prominence of particular symptoms. Where meningitis supervenes upon other acute illnesses, its features may be more or less masked. In cases without apparent cause, and in cases caused by chronic inflammatory lesions, like otitis, the symptoms often commence acutely with chill or rigor, and acute pain in the head. This is generally very severe and constant and aggravated from time to time. The patient becomes feverish, shuns light and noises, and may lie curled up in bed, resenting interference, as in the tuberculous cases. Vomiting often occurs at the commencement. There may be rigidity of the muscles at the back of the neck, and the head is drawn back. The pupils are often contracted. Convulsions also may occur quite early, and may be followed by active delirium, or by drowsiness accompanied by delirium; and in later stages there is often paralysis, with repeated attacks of convulsions, generally bilateral. The paralysis is very variable, corresponding to the situation of the effusion; from its frequent occurrence at the vertex it less often affects the cranial nerves than does tuberculous meningitis, though there may be squint; but an arm or leg is often paralysed, or there may be complete hemiplegia. Sometimes there is rigidity of the paralysed limbs, or of their fellows. The pupils become dilated, and the ophthalmoscope sometimes reveals optic neuritis, which may develop rapidly under observation. The temperature is high, varying from 102° to 104°, the pulse is mostly rapid, respiration is sighing, irregular, or of Cheyne-Stokes type, *tache cérébrale* may be well marked, and in some cases the abdomen is retracted. The drowsiness passes into deep coma, and, finally, the evacuations are passed involuntarily, the breathing and circulation fail, mucus accumulates in the chest, and death terminates the scene. The disease is often fatal within two or three days of the first symptom, and sometimes even less; exceptionally the illness lasts longer, as in a patient under my care, who died on the twentieth day.

**Diagnosis.**—This presents the same difficulties as in tuberculous meningitis, but, the course being much more rapid, it is less often the stage of headache than the stage of coma, or delirium, that may be misunderstood. Sometimes a diagnosis has to be made when severe headache and a quickly following coma are the only important features of the case, or from a convulsion occurring quite unexpectedly in the course of some septic or infective disease. Where a primary source for the meningitis, such as otitis, exists, one may be easily led to a right opinion; in the absence of this, one must look for fever, or any indication of paralysis or rigidity of a limb. As compared with ordinary *apoplexy*, the hemiplegia of meningitis is often much less complete; it may, however, be a typical hemiplegia, so far as the distribution is concerned, from the meningitis involving the cortical motor area. Meningitis has to be recognised as one of the results of *chronic otitis*, and the difficulties in determining its presence in that disease have been already pointed out (*see* p. 838). The symptoms of meningitis may also be confounded with those of *abscess*, and all the more as either may occur from disease of the ear, or of the cranial bones. The complete clinical course of fever, headache, delirium, coma, convulsions, and paralysis or rigidity, all within three or four days, is in favour of meningitis; in abscess there is more likely to be severe headache for some days before the coma—the temperature is either lower or oscillating, with chills, rigors, or sweating. The diagnosis from *tuberculous meningitis* has been already discussed. By relying too much upon the mental condition in cases of meningitis (and abscess) in young women an unjustified suspicion of *hysteria* may for a time be entertained. The absolute diagnosis may be as a rule made by lumbar puncture. The cerebro-spinal fluid in the early stages may be clear, but microscopical examination reveals polymorphonuclear

leucocytes and the presence of bacteria. Cultivation will usually determine the nature of the infecting organism. In the later stages the fluid becomes turbid, or even yellow from the presence of pus. The acute syphilitic cases usually occur in the secondary stage of the disease and show other signs of syphilis. The cerebro-spinal fluid in such cases shows an excess of lymphocytes, and gives a positive Wassermann reaction.

**Prognosis.**—The majority of cases of suppurative meningitis are fatal. What proportion, or if any at all, recover is still much debated, because post-mortem evidence is not forthcoming to prove conclusively the actual occurrence of meningitis; but the prognosis must be unfavourable in proportion to the rapidity and severity of the symptoms.

**Treatment.**—Lumbar puncture should be performed daily, and the spinal fluid, which is usually under considerable pressure, should be allowed to drain until the rate of flow appears normal. This procedure is frequently successful in relieving headache and neck stiffness. Morphia is often necessary to relieve pain and allay irritability. In syphilitic cases treatment with neo-salvarsan should be instituted at once. If the streptococcus be identified as the causal agent antistreptococcal serum should be given subcutaneously.

### PNEUMOCOCCAL MENINGITIS

The cerebral meninges may be the first part of the body to be invaded from the bloodstreams by the pneumococcus, when the disease may be called *primary*; but the meningitis is more often secondary to a pneumococcal infection of the lung, pleura, or other part. It is sometimes associated with malignant endocarditis, and pneumonia in a common infection. Out of twenty-three cases at the Children's Hospital, Great Ormond Street, seven followed empyema and purulent pericarditis, seven ear disease, and others followed pneumonia (F. E. Batten).

In symptoms and morbid anatomy it closely resembles other forms of suppurative meningitis, and in former times some of the descriptions of the latter must have been drawn from pneumococcal cases. In these latter the exudation is generally a thick, viscid, greenish pus; which mostly occupies the vertex, and is either in the meshes of the pia arachnoid or in the subdural space. It is sometimes found at the base, and sometimes in both situations, and very generally there is some pus on the posterior surface of the cervical spinal cord.

The symptoms—namely, intense headache, vomiting, high temperature, paralysis, convulsions, and coma—are very rapidly developed, and may be fatal within twenty-four hours. Thus in a case of malignant endocarditis with pneumonia the patient was taken with convulsions and died twelve hours later. Sometimes, however, in secondary cases the meningitis has been found *post mortem*, without having given rise to definite local symptoms.

The diagnosis may be inferred from the clinical associations of the case, or from the rapidity of its course. It can generally be decided by a lumbar puncture, when the pneumococcus may be identified in the cerebro-spinal fluid.

The **Prognosis** is very bad, and **Treatment** can only be conducted on the same lines as in other suppurative cases. Anti-pneumococcal serum may be tried (*see* p. 242).

### SYPHILITIC MENINGITIS

The syphilitic virus may in rare instances give rise to the symptoms of an acute leptomeningitis (*see* p. 843). These cases occur shortly after the primary infection during the stages of general dissemination of the spirochætes. Much more common than this is a chronic inflammatory process of syphilitic origin occurring at a later period, from one to five years or even later, after the initial infection,

which affects both pia-arachnoid and dural membranes in a widespread manner, and gives rise to symptoms both by strangulation of the cranial nerve roots and by causing an increase of intracranial tension.

**Morbid Anatomy.**—The appearances are very similar to those already described in connection with syphilitic meningomyelitis (*q.v.*). As a rule pia arachnoid and dural membranes are affected together and become welded into a dense fibrous mass, which may be as much as $\frac{1}{8}$ inch in thickness and envelop the whole of one or both hemispheres. At the base of the brain the lepto-meninges alone may be affected, but are usually adherent to the dura. The interpeduncular space is a common situation for this form of meningitis, from which may result compression of the upper cranial nerves, particularly the third and sixth pairs, and also interference with the arterial branches of the circle of Willis. As in the case of syphilitic involvement of the spinal meninges, there is always a certain amount of vascular disease accompanying the meningitis. This takes the form of endarteritis and perivascular sheathing with lymphocytes. With this is associated some degeneration of the underlying nervous substance itself. Thrombotic occlusion of the narrowed vessels may lead to the additional feature of patches of cerebral softening.

**Symptoms.**—The symptoms of the rare form of acute syphilitic meningitis occurring in the early stages of the disease are similar to those met with in other acute inflammatory affections of the leptomeninges, and have already been described.

The main clinical features of the chronic variety are intense headaches, together with vomiting and other signs of increased intracranial pressure, and multiple paralyses of the cranial nerves. In the early stages the symptoms are very frequently intermittent. Thus the cranial nerve palsies—and it is the third and sixth nerves which are most often affected—are commonly transient; and the headache may come on in paroxysmal attacks, lasting a few hours or a few days, which are followed by relatively long intervals of quiescence. Papillœdema may be present, or a gummatous mass pressing upon the optic chiasma may lead to optic atrophy with alterations in the visual fields similar to those met with in pituitary tumour.

The cranial nerves are picked out in a random manner indicative of the widespread distribution of the inflammatory process. Localised patches of thickening upon the surface of the hemispheres may give rise to attacks of Jacksonia epilepsy (*q.v.*), which may be followed in turn by paralyses of monoplegic or hemiplegic distribution. As the disease progresses the headaches become more continuous, and the cranial nerve palsies more constant.

Mental deterioration is a frequent symptom resulting from the combination of increased intracranial pressure with syphilitic disease of the blood vessels. The syphilitic endarteritis also may lead to additional symptoms of cerebral thrombosis with sudden onset of paralysis of one or more limbs.

**Diagnosis.**—The chronic form of syphilitic meningitis has to be distinguished from other conditions which give rise to signs of increased intracranial pressure with paralysis of the cranial nerves. Of these the most important is cerebral tumour. Points in favour of a syphilitic origin are a relapsing tendency and scattered cranial nerve palsies. A strongly positive Wassermann in the blood is the rule in cerebral syphilitic meningitis, and the reaction may also be positive in the cerebro-spinal fluid. In cases of cerebral tumour the reaction is negative in both blood and fluid.

Some cases of syphilitic meningitis with mental deterioration, headaches, and epileptic attacks may closely simulate general paralysis of the insane, especially if the Wassermann reaction is positive in the cerebro-spinal fluid. The diagnosis in such an instance may usually be arrived at by observing the results of anti-syphilitic treatment. When the disease is mainly confined to the meninges the symptoms as a rule disappear, and the Wassermann reaction often becomes

negative under treatment, whereas neither of these results is obtained in general paralysis.

The **Prognosis** is good in cases which are diagnosed and effectively treated in the early stages, but, as in the case of syphilitic disease elsewhere in the body, there is a tendency to relapse, and it is well, therefore, for the patient to submit to a short course of treatment annually as a preventive measure against recurrence.

**Treatment.**—This is the same as for syphilis elsewhere, with the provision, however, that it is best not to commence with intravenous injections of arsenical compounds, such treatment being liable in some cases to provoke an initial reaction in the inflammatory tissue with increased symptoms of intracranial tension. It is, therefore, wise to begin with potassium iodide, which should be given in large doses (120 to 180 grains daily) for the first fortnight, after which treatment should be continued on the lines already indicated (*see* p. 776).

## CEREBRAL PACHYMENINGITIS

The dura mater becomes inflamed on its surface (*pachymeningitis externa*) in consequence of injuries, or the extension of inflammation from diseased bone, otitis, or any of the causes already mentioned as leading to suppurative meningitis. The inner surface of the dura mater is also inflamed in many cases of suppurative meningitis (*pachymeningitis interna purulenta*) and in *syphilitic meningitis* (*see* p. 845). The symptoms in these cases, due to the inflamed dura mater, are not distinguishable from those referable to the other membranes.

### PACHYMENINGITIS INTERNA HÆMORRHAGICA

This is a condition in which a mass of blood and partially organised blood clot is found upon the surface of the brain between the dura and the arachnoid membrane. This is usually surrounded by adhesions by which it is encapsuled.

**Ætiology.**—The condition is found most commonly in association with chronic alcoholism and chronic insanity and in old persons. Two views have been put forward as to its causation, (1) that the initial process is inflammatory (of toxic origin), with subsequent extravasation of blood from engorged capillaries; (2) that the effusion of blood is of traumatic origin and that the subsequent encystment results from the reaction of the meninges to the mass of blood clot. In favour of the second explanation Trotter has put forward the arguments that the condition in no way differs from that known to occur as a delayed result of head injury, and that the insane person or alcoholic is quite likely to sustain a head injury and subsequently forget its occurrence. According to Trotter, then, the effusion is due to oozing from the veins passing from the surface of the brain to the longitudinal sinus, and the laceration of these vessels is due to an unrecognised head injury. Sudden displacement of the brain, especially in a forward or backward direction, may be supposed quite readily to cause such laceration, and may possibly result from a trivial blow. If this view is correct the condition is identical with that described in surgical text-books as *chronic subdural hæmatoma* (7).

**Morbid Anatomy.**—The inner surface of the dura mater is covered with one or more layers of membrane, soft and friable when recent, tougher and more fibrous when old, in colour brownish red, brown, brownish grey, yellow, or even white, and often presenting punctiform ecchymoses; while between the layers may be considerable quantities of blood clot more or less altered by age, or collections of serum containing cholesterin crystals. The deposits are usually situate over the parietal region, near the middle line, and are bilateral in about half the cases. They may be mere membranes, or 2 or 3 mm. in thickness, and if much blood is extravasated, the surface of the brain is depressed. Micro-organisms have been looked for but have not been found.

The **Symptoms** are very variable. Often the condition has been found *post mortem* without any symptoms which would be explained by it; sometimes, on the other hand, a fatal apoplexy is the result of a large hæmorrhage between the membranous layers, which compresses the brain. Such an illness may be extremely difficult to distinguish from other forms of cerebral hæmorrhage. But the attack may be less severe, and recovery takes place, or there are fresh seizures at different intervals. The symptoms are generally headache, giddiness, and somnolence with sudden lapses into coma. With this there may be weakness of the upper neuron type affecting one or both sides of the body. The plantar responses are frequently extensor. Twitching of the limbs may occur, but true epileptiform convulsions are uncommon. Papilloedema is sometimes present. The pulse is often slow or irregular, the pupils are contracted, and there is some degree of fever. In the intervals the patient may return to his former condition, or present some impairment of the cerebral functions, such as diminished intelligence and memory, drowsiness, weakness of the limbs, and headache. The cerebro-spinal fluid is usually normal, but may contain altered blood pigment.

**Diagnosis.**—This has to be made from cerebral hæmorrhage and from causes of increased intracranial pressure, such as tumour and syphilis. A history of symptoms appearing gradually after a head injury is of the greatest value, but in the type of individual most frequently affected is seldom forthcoming. Huguenin enumerated as aids to diagnosis, besides the predisposing conditions, the evidence of sudden and increasing compression, the symptoms showing that the convexity, and especially the cortical motor area, is affected, the spread of the disease from one side to the other, and recovery after apparently severe illness. Cerebral symptoms in the interval, and one or more previous attacks, strengthen the diagnosis. Lumbar puncture by revealing the presence of altered blood pigment may settle it.

**Treatment.**—If the diagnosis can be made correctly the proper treatment is surgical decompression with evacuation of the mass.

## THROMBOSIS OF THE CEREBRAL SINUSES

The blood coagulates in the cerebral sinuses either as a result of some profound cachexia, or in consequence of infection from lesions of adjacent parts.

The former causes mostly an adhesive thrombosis, the sinus being obstructed by laminated clot, without any general infection of the system. It occurs most often in infants, especially those suffering from marasmus or chronic diarrhœa. It mostly affects the *longitudinal sinus*. Coma, stiffness of the back, neck, or limbs, strabismus, nystagmus, and paralysis or spasms of the face, are said to occur. Distension of the veins over the forehead and temple, and epistaxis, have been described as resulting from thrombosis of the longitudinal sinus, and œdema of the skin over the mastoid process when the lateral sinus is affected; but the constancy of the symptoms is doubtful.

The morbid appearances associated with thrombosis of the longitudinal sinus are highly characteristic. The large veins in the pia-arachnoid are thrombosed and stand out like dark cords. There is hæmorrhage. The veins on the inner surface of the dura as they enter the sinus are also distended.

In adults the chlorosis and anæmia of young girls are occasionally causes of thrombosis, but phthisis, carcinoma, and wasting diseases more commonly. The symptoms are similar—namely, vomiting, headache, drowsiness, convulsions, delirium, and coma with nystagmus, strabismus, sometimes optic neuritis, and twitchings or weakness in the extremities. The result depends largely upon the primary ill-health, to which treatment must be directed.

Thrombosis from *local infection* is mainly caused by extension from disease of the ear, but the origin may be in the orbit, nose, mouth, pharynx, or other part

from which the lateral or cavernous sinus can be reached. The *lateral* and the *petrosal sinuses* are more often affected, and it has already been stated that the optic neuritis frequently accompanying the spreading forms of otitis is probably due to this. The symptoms resemble those of cerebral abscess, namely, fever, headache, delirium, stupor, with the addition of rigors as an important feature, and later local cerebral symptoms, such as paralysis or convulsions. Thrombosis of the lateral sinus may extend into the *jugular vein*, and produce a hard swelling, with more or less tenderness, or even redness, œdema, and finally suppuration, in the upper part or whole vertical extent of the neck. Thrombosis of the *cavernous sinus* causes proptosis of the eye, chemosis or swelling and vascularity of the conjunctiva, and œdema of the eyelids and root of the nose. The eyeball becomes fixed, and the optic nerve inflames.

In these infective cases, as a rule, septic particles are conveyed into the right heart, and thence into the lungs, so that a fatal pyæmia is the result.

**Treatment** In thrombosis dependent on general ill-health, this condition must be treated. If local lesions are the cause, they must be dealt with surgically. The spread of an infective thrombus down the jugular vein can be prevented by tying the vein below the clot and clearing out its contents, as well as those of the lateral sinus, if necessary. Citric acid in doses of 30 or 40 grains every four hours has been given with the view of diminishing the coagulability of the blood.

## TUMOURS OF THE BRAIN

Intracranial neoplasms (including the infective granulomas) are of comparatively frequent occurrence among diseases of the brain. In the early stages of their growth they may give rise either to no symptoms at all, or to signs which require the most careful investigation by the physician before a successful diagnosis can be made. It is in these early stages, however, that surgical operation offers most chance of alleviation or cure. In the later stages the symptoms produced are as a rule easy of recognition, but the outlook from the point of view of surgical treatment is proportionately less hopeful.

**Ætiology.**—Syphilis and tubercle account for the granulomas. In a certain number of cases of tumour there is a history of preceding head injury, but the causal connection is doubtful. Hydatid cysts and cysticercus occasionally play the part of cerebral tumours.

**Morbid Anatomy.**—*Tuberculomas* account for a high proportion of all cases of intracranial neoplasm met with in children. About 18 per cent. of cases in persons under twenty years of age are of this nature. After this age they are progressively less common, and are rarely found after thirty. They most usually occur in the cerebellum, pons, or mid-brain, but are found also in other situations, and may be multiple. They consist of globular masses in the cerebral substance, ranging from $\frac{1}{3}$ inch to 2 or 3 inches in diameter. These masses contain an inner core of yellow caseous material bounded by an outer narrow zone of a pinkish-grey colour and dense consistency. They can be easily detached from their bed.

Microscopical examination reveals the presence of tubercle bacilli in sections through the wall of the granuloma, but the characteristic giant cell systems are frequently lacking.

Circumscribed *gummas* are relatively uncommon—about 3 per cent. of all intracranial neoplasms. They always grow in connection with the membranes, and are therefore commonly situated upon the surface of the brain. They may, however, be found in one of the central parts growing from a deep fold of the pia mater. They tend either to break down and become caseous in the centre, or to take the form of firm fibrous masses.

*Hydatid cysts* and the *cysticercus cellulosæ* are rarities which need not be described in detail. Hydatid cysts may attain to a considerable size and produce

the signs of a large brain tumour. Those of the cysticercus cellulosæ are usually small and multiple, and, besides being found in the cerebral substance, may lie free in the ventricles, or, situated upon the surface of the brain, may give rise to a chronic diffuse meningitis.

Of true intracranial neoplasms the commonest are the *gliomas*, which amount to more than 50 per cent. of the total. They occur with equal frequency at all ages, and in all parts of the brain. They are commonly deep seated in the cerebral substance. Histologically the gliomas are of the nature, as their name implies, of an overgrowth of the neuroglia cells. They vary considerably in microscopic appearance according to the activity of cell proliferation, but in this respect they are characteristically malignant tumours, that their cells tend insidiously to invade the nervous substance without the formation of a surrounding capsule. They do not, however, give rise to metastases either in the brain or elsewhere. They are in the first place vascular growths, but in the course of their development the vessels leading to the central parts of the tumour frequently become occluded with resultant necrosis. The necrotic material commonly undergoes liquefaction with the formation of cysts, which may reach a considerable size, containing in extreme instances as much as 50 c.c. of greenish-yellow fluid which is highly albuminous, and may coagulate into a solid mass on standing in a test-tube.

The so-called *dural endothelioma* comes next in order of frequency among brain tumours, comprising from 15 to 20 per cent. These tumours may occur at any age, but are most commonly discovered giving rise to symptoms between the ages of thirty and forty. They are more common in the anterior and middle fossæ than in the posterior fossa of the skull. They arise apparently from arachnoid cells on the inner surface of the dura mater, and are essentially slow-growing encapsulated tumours of innocent type. They vary in size from that of a pea to a tangerine orange, are roughly spherical in shape, nodular, and of moderately firm consistency. They may have a wide connection with the dura mater or retain their attachment to it by a comparatively narrow stalk. They occur naturally upon the surface of the brain, which is compressed by the tumour, but a growth arising from the falx cerebri may so bury itself in the hemisphere from its mesial aspect as to appear as a subcortical mass beneath the lateral surface. At the point of attachment to the dura these tumours may become calcified or even ossified, or they may grow outwards eroding the bones of the skull. They are occasionally multiple, and may be associated with multiple tumours of the nerve roots. Microscopically they consist of densely packed cells of a spindle shape, with large oval nuclei, which tend to be arranged in whorls and are supported by a connective tissue stroma. The centres of the whorls frequently undergo hyaline degeneration and calcification, and are then called psammoma bodies. Tumours of similar type may arise from the arachnoid membrane.

The remaining types of brain tumour are all relatively rare in comparison with glioma and endothelioma. Perhaps the next in order of frequency is that which grows from the eighth nerve in the cerebello-pontine angle, and has been styled by Cushing the *acoustic tumour*. This commonly attains the size and shape of a small walnut, is a nodular encapsuled growth, and possesses the histological characteristics of a neurofibroma. Cushing also has drawn attention to growths which he describes as simple *adenomas of the pituitary body*.

There are also to be considered *neuroblastomas*, which arise from the nervous tissues themselves; *sarcomas*, which may be primary, growing from the meninges, or secondary to growths in the kidney, suprarenal, eye or elsewhere; *carcinomas*, which are almost always metastatic from various parts of the body, and usually multiple; *papillomas of the choroid plexus*; and *simple cysts*, either of developmental or traumatic origin.

**Pathology.**—The tumour in the course of its growth, by pressing upon the vessels supplying surrounding areas of the brain, causes local anæmia and finally

degeneration of the nerve cells and fibres. This is more likely to be caused in the early stages by rapidly growing tumours in the cerebral substance, such as the gliomas, than by those of slower growth and external origin, of which the endothelioma is the typical example. Tumours of the latter type indeed may produce considerable indentations in the surface of the brain without permanent destruction of the cortex, owing to the fact that the pressure, being so gradually increased, is more evenly transmitted throughout the cranial cavity. Tumours arising from the nerve sheaths (neurofibroma) will naturally commence by destroying the fibres of the nerve affected, and growths arising from the meninges or other structures at the base of the brain cause pressure degeneration of the cranial nerves, and sometimes also erosion of the bone.

The cranial cavity may be considered as a rigid box completely enclosed save for one comparatively large aperture, the foramen magnum, and several smaller ones for the vessels and for the exit of the cranial nerves, of which the most important are those through which pass the optic nerves. Along these nerves the arachnoid and dura are reflected in the form of sheaths extending as far as the optic discs.

The growth of a tumour within the cranium will therefore cause an increase of intracranial pressure, the degree of which depends, as will be shown, not only upon the size, but also upon the position, of the tumour. It will be remembered that the cranial contents are very definitely separated into two divisions by the tentorium cerebelli, and that of these two the anterior division is again subdivided by the falx cerebri, which, however, does not fit tightly over the anterior part of the corpus callosum, so that the partition here is incomplete.

Let us consider first the mechanical sequence of events caused by a tumour of one cerebral hemisphere (*supra-tentorial tumour*). In the beginning this makes room for itself by compression of the blood vessels, by obliteration of the cerebral sulci and flattening of the gyri, by occlusion of the lateral ventricle of the same side, which ultimately becomes reduced to a mere slit, and by enlargement of the hemisphere affected and dislocation of part of it across the mid-line beneath the falx cerebri, thus causing pressure upon the opposite hemisphere. With still further increase in the size of the tumour the pressure becomes generally raised in the whole anterior division. This increase of pressure is transmitted through the cerebro-spinal fluid contained in the sheaths surrounding the optic nerves to the nerve heads, where it gives rise to the changes already described as constituting papilloedema (*see* p. 744). Subsequently one of two things may happen: either some of the cerebral substance may be squeezed beneath the edge of the tentorium so as to form a subtentorial herniation, or the still increasing pressure in the anterior division may be transmitted through the yielding membrane of the tentorium to the contents of the posterior fossa. In any case the result is the same—the pressure in the posterior fossa is increased, and this is met by gradual squeezing of a part of the cerebellum into and through the foramen magnum, producing the so-called pressure cone (*see* Fig. 91). This process continues until the pressure cone is sufficiently large to compress the medulla against the foramen magnum. At this juncture the foramen of Magendie is probably blocked, leading to an obstruction between the source of secretion of the cerebro-spinal fluid in the lateral ventricles and the site of its absorption at the base of the brain. The result is a *secondary internal hydrocephalus*, the distension being confined to the ventricle on the side opposite to the tumour, which is probably not occluded. There ensues a rapid further increase of intracranial pressure, wedging the pressure cone and the medulla firmly into the foramen magnum, and death follows from anæmia of the respiratory centres.

As regards their general effects, tumours situated in the posterior division of the cranial cavity—*subtentorial tumours*—may be divided into those growing from *without* the cerebellum, pons and medulla, and those growing *within* the cerebellum, pons and medulla.

*Extra-cerebellar tumours* are usually of slow growth, and have a certain amount of room in the lateral recess in which to expand. It is therefore only in their later stages that they cause a rise of tension in the posterior fossa. This is followed by the formation of a pressure cone from squeezing of the cerebellum into the foramen magnum, and in the manner already described secondary internal hydrocephalus is set up. In this case, however, *both* lateral ventricles are patent and become equally distended from accumulated pressure of cerebro-spinal fluid; the cerebral sulci become obliterated, the gyri flattened, and the pressure being transmitted along the optic nerves causes papilloedema.

*Intra-cerebellar tumours* are usually of more rapid progress, and the increase of pressure caused by them results in the early formation of a pressure cone and the rapid establishment of the conditions leading to secondary internal hydrocephalus, as described above, with general rise of intracranial tension.

Growths of the medulla are rare, and may be fatal from their local effects before distant pressure signs have time to appear.

Growths of the pons are frequently diffuse gliomas which give rise to no symptoms of general pressure, except perhaps in the terminal stages if the Sylvian aqueduct is blocked, when secondary internal hydrocephalus will result.

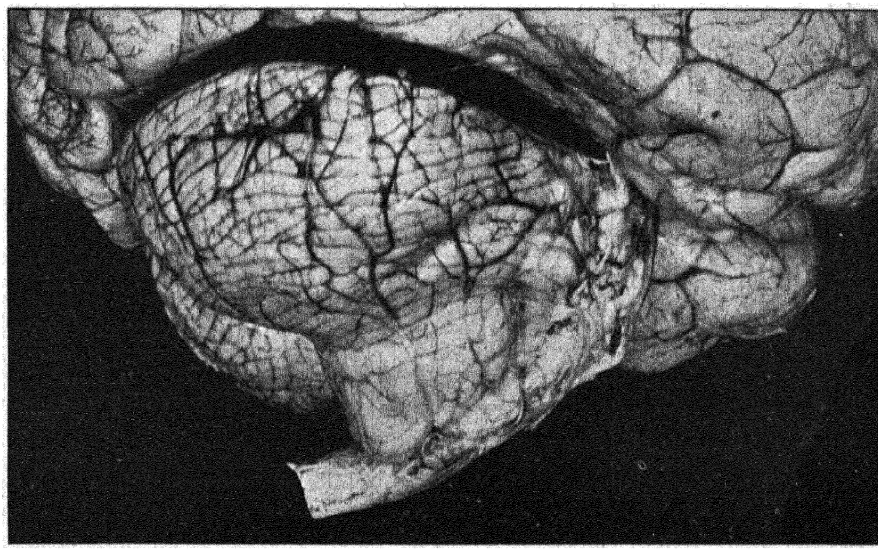

FIG. 91.—Shows the formation of a "pressure cone," photographed from the side. (*After Turner and Stewart.*)

**General Symptoms.**—These depend upon generalised increase of intracranial tension, and are therefore relatively late symptoms of brain tumour. They are—

*Headache.*—This is a more or less constant symptom, and though it may at first be intermittent and of slight degree, in the later stages it is often persistent and attains a severity which is rarely met with in headaches due to any other cause. It may be felt all over or localised mainly to the front, sides, or top of the head. It is frequently at its worst in the early morning, and is often associated with nausea or actual vomiting.

In the early stages the patient may be seized with paroxysmal attacks at considerable intervals, and even later on, when it becomes constant, the headache may at times become greatly intensified without apparent cause. Change of posture, such as stooping, or forcible expiratory movements, as in coughing or

sneezing, often precipitate an attack or intensify pre-existing pain. Pain which is limited to the suboccipital region and radiates down into the back of the neck is strongly suggestive of a tumour in the posterior fossa. Headache which is always referred to the same spot, especially if it be accompanied by tenderness to pressure over this area, is evidence in favour of the growth being situated on the surface of the brain, or growing from the dura mater in this locality.

*Vomiting* is a less constant and a less frequent symptom than headache, but is present in the majority of cases. In its most characteristic form it is associated with headache and is independent of the taking of food. It is most marked in tumours of the posterior fossa, and in these cases is not infrequently associated with nausea and anorexia.

Vomiting is usually a comparatively late feature in the history of the disease.

*Papillœdema* is the most characteristic of all symptoms of cerebral tumour, being due in 90 per cent. of all instances to this cause, while it is entirely absent only in a small number of cases (10 to 20 per cent.). Its appearances and the mechanism of its production have already been described (*see* pp. 725, 741). As a general rule papillœdema occurs earliest and is most intense in intra-cerebellar growths. In tumours of the cerebrum its onset is more gradual, while in the case of extra-cerebellar and intra-pontine growths it may occur only in the terminal stages, or remain absent to the end. The condition is nearly always bilateral, but one optic disc may be affected before the other. The side upon which the papillœdema commences or is more intense bears no relation to the situation of the tumour. A high degree of swelling is compatible with good visual acuity, though the patient may complain of transient attacks of mistiness of vision. Permanent impairment of vision only occurs with the onset of secondary optic atrophy.

*Convulsive attacks* are not so frequent as the symptoms already mentioned, and they are very irregular in their occurrence in the cases attended by them; thus there may be two or three in the whole course of the illness, or they may be very frequent. They may take the form of generalised epileptiform attacks, in which there is an initial stage of tonic rigidity followed by clonic spasms and a temporary condition of stupor, or they may be limited to a momentary loss of consciousness. "Focal seizures" with characteristics distinctive of their origin in definite areas of the brain will be alluded to under the heading of Local Symptoms. They may, however, occur in the late stages of increased intracranial pressure without relation to the site of the tumour, and so be misleading and of false localising value.

*Vertigo* is not an uncommon symptom, and is usually associated with attacks of headache and vomiting.

*Slowness of the pulse* when it occurs is usually a late symptom of increased intracranial pressure.

Among other general symptoms are *mental changes:* the patient may be dull, apathetic, forgetful, sleepy, careless or untidy; in later stages he becomes lethargic, and finally comatose. These, however, occurring as the result of increased tension, are late changes. In the early stages they are more commonly due to widespread destruction of the association fibres, such as is produced by large tumours of the cerebral hemisphere, and as such will be considered under Localising Symptoms.

**Localising or Focal Symptoms.**—It is important to realise from the first that these are of greatest value in determining the site of the tumour during the earlier stages in its growth. In the later stages the mechanical effects of increased intracranial tension may result in damage to parts of the brain, or traction upon cranial nerves, far removed from the site of the original lesion.

*Tumours involving the motor centres and pathways* give rise to symptoms of irritation or paralysis of the parts affected.

*Paralysis* in the form of a slowly progressive hemiplegia, with increase of deep reflexes, loss of the abdominal reflexes, and an extensor plantar response on the

affected side, is produced by deeply seated growths involving the pyramidal fibres. Tumours situated more superficially and at first producing a local anæmia of the cortical centres frequently give rise to attacks of *Jacksonian epilepsy* of motor type. These are localised convulsions, beginning always in one part and unaccompanied at first by loss of consciousness. In these cases, according to the severity of the temporary disturbance in the part, there may be a twitching, say of the hand alone or of the hand and arm, or of all the muscles of one side of the body; if the convulsions spread to the other side of the body, consciousness is usually lost.

In their spread from the point of commencement the movements always show a definite sequence. If they begin in the face, they spread successively to the upper limb, beginning with the thumb and fingers, and then to the lower limb; if they begin in the leg, the arm and face are successively affected; if in the arm, the face and leg in turn. The convulsions are usually followed by some degree of temporary paralysis, especially in the part first affected, and for a short interval after the attack changes in the reflexes may be demonstrated on the affected side in the form of diminished or absent abdominals and an extensor plantar response. With progressive increase in the size of the tumour and destruction of the centres at first irritated, the fits become less frequent and less extensive, giving place to permanent paralysis. If the growth is in the left hemisphere in the case of a right-handed person, some degree of aphasia will accompany the attacks, and remain with the paralysis, especially if the arm and face centres are chiefly involved. The aphasia produced will be at first of the motor type, though later this may be obscured by mental changes.

*Tumours of the post-central regions* in similar fashion produce progressive hemianæsthesia if deeply situated. This is limited as a rule to loss of sense of position and astereognosis in the affected hand, but there may also be a diminution in the subjective intensity of sensations of touch, temperature and pain, which is apt to be of "glove" or "stocking" distribution. Tumours involving the cortex give rise to Jacksonian attacks of sensory character, resembling the motor seizures in the manner of their spread, but consisting of a sensation of numbness or tingling, which is often attended with extreme discomfort.

An attack of this nature frequently passes into a motor seizure of the type already described.

*Tumours of the occipital lobe* give rise to defects in the visual fields of the opposite side, the exact area of the defect depending upon the situation of the lesion. Superficial tumours of this region may cause attacks of subjective sensations of visual disturbance, usually in the form of flashes of light or colours in the opposite visual field. An occipital growth may by downward pressure upon the tentorium give rise to compression of the cerebellum, thus leading to signs of false localising value.

*Tumours involving the uncinate gyrus* give rise to peculiar attacks first described by Hughlings Jackson and known as *uncinate seizures*. These are in the nature of unpleasant sensations of taste or smell, indefinitely described by the patient in various ways as being "like the smell of rotten eggs," a "bitter taste as if a gumboil had burst in my mouth," and so on. This is often preceded by a dreamy state accompanied by visual hallucinations. The attacks may be quite momentary, or may occur in the form of auræ ushering in epileptiform convulsions.

*Tumours of the frontal lobes* frequently give rise to no localising signs of diagnostic importance, and the symptoms are therefore confined to those of general pressure. These may, however, be accompanied or preceded by mental symptoms to a degree unusual in the case of tumours elsewhere. The changes noticed are inattention, inability to keep the mind fixed on any subject, loss of memory for recent events, and incoherence in conversation. Sometimes these patients are childish, casual and irresponsible, and show a tendency to make silly and pointless jests at unexpected moments. In the later stages apathy and stupor supervene.

A high proportion of cases of brain tumour discovered *post mortem* in mental asylums are situated in the frontal lobes. Convulsive attacks may occur either of a generalised nature or beginning with conjugate deviation of head and eyes towards the opposite side from irritation of the centre for this movement in the pre-central area, in which case they are of diagnostic significance. A tumour of the anterior end of the frontal lobe may by pressure upon the olfactory tract produce unilateral anosmia.

*Tumours of the temporal lobe*, apart from those at the anterior end, involving the uncinate gyrus, and those at the posterior end on the left side, involving the speech centres, produce as a rule no signs of localising value, and the symptoms are therefore limited to those of general pressure. Changes in the sphere of intellect and temperament may occur as described in the case of frontal tumours.

*Tumours of the corpus callosum* do not, as a rule, cause very definite symptoms, but may give rise early to mental changes, and are sometimes associated with motor apraxia (*see* p. 719).

*Tumours of the mid-brain* may give rise to localising signs in the form of weakness of upward movement of the eyeballs, defective reaction of the pupils to light, excentricity of the pupils, paresis or paralysis of the third cranial nerves, and defective sensibility over the area supplied by the fifth nerve, due to involvement of its mesencephalic root. Additional symptoms may arise from involvement of the longitudinal tracts in this region.

*Tumours of the interpeduncular space* produce symptoms due to pressure upon the optic chiasma and tracts, the third nerves, crura cerebri and pituitary body, which have already been described under the heading of Pituitary Tumours (*see* p. 593).

In *tumours of the pons* the localising signs depend upon affection of the cranial nerves situated therein, together with symptoms of destruction of the longitudinal tracts. Symptoms of general pressure are conspicuous by their absence until the terminal stages.

*Tumours of the cerebellum* frequently give rise to headache, which is at first localised to the suboccipital region, with a tendency to radiate downwards into the neck. Symptoms of general pressure appear early and are prominent. In addition there are nystagmus, inco-ordination in the performance of all muscular movements, and reeling or staggering gait. In a tumour affecting mainly one lobe of the cerebellum, the headache may be mostly referred to that side, and there may be localised tenderness to pressure.

The tendency of the eyes is to deviate towards a point somewhat to the opposite side, so that nystagmus is coarser and more easily elicited when the patient looks toward the side of the tumour. There is frequently some degree of weakness, tremor, and hypotonia on the affected side of the body, and inco-ordination may be demonstrated in the performance of the finger-nose-finger, diadochokinesis, and heel-knee tests (*see* p. 732).

Characteristic peculiarities of attitude and gait are often present. There is a tendency for the head to be tilted towards the side of the tumour, and for the whole of the body to deviate towards that side. In consequence the ipsilateral shoulder is held higher than the other, and the ipsilateral lower limb is slightly abducted and everted, the former of these being of the nature of a compensatory mechanism for the spontaneous deviation.

In walking there is a spontaneous tendency to deviate or, it may be, to fall towards the side of the lesion, which is corrected by voluntary efforts, so that the patient may appear to stumble towards the sound side. In any doubtful case, the patient may be asked to stand on either leg alternately, when the greater weakness and unsteadiness of the affected side will be apparent. The reflexes as a rule are not altered, but the knee jerk on the affected side may show a pendular character when elicited with the leg hanging free, this being due to loss of muscular tone.

*Tumours of the cerebello-pontine angle.*—These are characterised by progressive symptoms due to pressure upon the cranial nerves, succeeded by signs of a unilateral lesion of the cerebellum when the tumour has grown large enough to compress and destroy that structure. As already mentioned, symptoms of general pressure are of late onset, and usually coincide with cerebellar involvement.

The commonest growth in this situation is the neurofibroma of the eighth nerve known as *acoustic tumour*. This always proclaims its presence first by symptoms referable to the auditory nerve in the form of roaring or whistling noises (tinnitus) and progressive nerve deafness. Next, the sensory root of the fifth nerve is involved, causing first diminution or loss of the corneal reflex on that side, and subsequently numbness and anæsthesia of the trigeminal field. At this stage the vestibular reactions normally obtainable by caloric, rotatory, or galvanic stimulation of the vestibular apparatus are found to be absent on the affected side. The seventh nerve is involved, with resultant facial weakness of peripheral type; nystagmus is present, being most marked when the patient looks towards the side of the lesion; and finally there ensue the signs already described as characterising a unilateral lesion of the cerebellum, together with headache, papilloedema and the other signs of increased intracranial tension.

*False Localising Signs.*—It has already been stated that in the later stages of the growth of a cerebral tumour the mechanical conditions arising from increased intracranial tension may result in symptoms which have a false localising value. These may be produced by traction upon the cranial nerves, by thinning of the cortex from the pressure of internal hydrocephalus, or by the obstruction of some important vessel. The cranial nerves which are most commonly affected in this way are the sixth pair. Therefore a sixth nerve palsy developing late in the course of the symptoms of intracranial tumour is of negligible value as an isolated sign. The other cranial nerves which may be affected in similar fashion are the third, fifth, seventh and eighth. Thinning of the cortex probably accounts for the occasional occurrence of motor Jacksonian attacks and signs of involvement of the upper motor neuron tracts in these late cases. Downward pressure upon the tentorium from a tumour of the occipital lobe may give rise to cerebellar symptoms.

It is also an undoubted fact that in some cases of cerebral tumour with a high degree of intracranial tension the tendon jerks may be lost in both upper and lower limbs. The reasons for this are somewhat obscure.

**Diagnosis.**—When once the signs of increased intracranial tension are established this is comparatively easy. The presence of persistent or constantly recurring headache of paroxysmal nature together with papilloedema is in nearly all cases evidence of a tumour of the brain. A condition which may require careful differentiation is that due to a *gummatous meningitis*; the presence of syphilitic stigmata elsewhere and a positive Wassermann reaction in the blood may help to clear up the diagnosis, but the possibility must not be forgotten of the existence of a cerebral tumour in a syphilitic subject.

*Renal disease* may simulate cerebral tumour in the headache, vomiting and affection of sight, and even the ophthalmoscope may not at once clear up the case, since optic neuritis may occur in renal disease (*see* p. 621), and, on the other hand, in papilloedema from cerebral tumour the exudate may spread to the retina, producing brilliant white spots, especially on the macular side of the disc, resembling those seen in albuminuric retinitis (*see* p. 629). Careful examination of the urine and cardio-vascular system will probably lead to a correct diagnosis. Optic neuritis in association with giddiness and vomiting may also occur in some cases of *anæmia*.

The possibility of *cerebral abscess* should always be considered, particularly if there be a history of suppuration in the middle ear or nasal sinuses.

Some cases of *encephalitis lethargica* presenting a picture of stupor with altera-

tions in the reflexes, cranial nerve palsies, and occasionally optic neuritis may require careful differentiation, which may usually be achieved on the history of subacute onset with pyrexia, and other distinctive symptoms.

*In the early stages* the diagnosis is usually a matter of great difficulty. Cases commencing with Jacksonian seizures, *petit mal*, or generalised epileptiform attacks are difficult to distinguish from idiopathic epilepsy. A definite local aura as the mode of onset of the fit and the presence of a transient post-convulsive monoplegia or hemiplegia with altered reflexes are signs favouring an organic basis, especially if consciousness be retained throughout; and in such a case, after careful observation, an exploratory operation may be justifiable, or the patient may be watched for the earliest signs of papilloedema to complete the diagnosis.

Jacksonian seizures, motor or sensory, may also usher in *general paralysis of the insane*, which may be distinguished by the pupillary and mental changes and the positive Wassermann reaction in blood and cerebro-spinal fluid.

The symptoms of tumours of the cerebellum sometimes closely resemble those of *disseminated sclerosis*, but the latter may be distinguished by the remittent history and the absence of general pressure symptoms.

Certain tumours by reason of their character and site cause a train of symptoms so definite as to be comparatively easy of recognition in the earlier stages, and of these tumours of the pituitary body and acoustic tumours are important examples.

*The X-rays* may occasionally be of service in the localisation of a cerebral tumour. It is only in rare instances that the plate reveals the actual position of a tumour which has undergone calcareous changes, yet the evidence afforded by stereoscopic plates in such cases is so conclusive that a lateral photograph of the skull should be obtained in every case before operation. Alterations in the shape of the sella turcica have already been referred to under Pituitary Tumours (*q.v.*), and the posterior clinoid processes may also be atrophied from the pressure of a distended third ventricle in cases of subtentorial tumour with internal hydrocephalus.

*Lumbar puncture* is a procedure which is attended with considerable danger in cases of cerebral tumour with advanced pressure symptoms, especially in the case of subtentorial growths. The sudden drainage of the cisterna magna may allow the cerebellar cone described above to descend still further through the foramen magnum, and this may lead rapidly to death from compression of the medulla. In cases in which there is reason to suspect syphilis it may be necessary to obtain a specimen of cerebro-spinal fluid for the Wassermann reaction, but a few cubic centimetres only should be withdrawn.

**Prognosis.**—In cases which are not subjected to operation the average survival period in a series of cases verified *post mortem* was from one to two years (Tooth), being longer in the tumours of innocent type than in the gliomas and sarcomas. The prognosis as regards life also varies with the situation of the tumour, being bad as a rule in those of the posterior fossa. In certain cases, however, the symptoms may regress, and there may be remissions lasting for months or even years. The expectation of life is perhaps longest in pituitary tumours of simple type, which by reason of their situation within the sella turcica are precluded from causing increase of intracranial pressure until a very late stage in their development.

As regards other symptoms apart from headache, the pain of which may make life unbearable sooner or later, vision is lost from secondary optic atrophy. In the later stages also mental symptoms are likely to supervene.

In the great majority of cases alleviation is all that can be obtained by surgical treatment, but the prognosis as regards duration of life is materially improved by decompression. In cases in which it is possible completely to remove a growth of innocent type recovery may be complete.

**Treatment.**—The treatment of syphilitic gumma is the same as the treatment

of syphilitic meningitis (*see* p. 845). The treatment of other cerebral tumours is almost entirely surgical, and may be considered under two heads: *palliative*, with a view to relieving pressure symptoms, and *radical*, with a view to exposing and removing the growth.

Palliative treatment consists in removing an area of bone and freely incising the dura before replacing the flap of soft tissues, thus allowing additional room for expansion. It is necessary first to decide whether the tumour is above or below the tentorium. For a tumour above the tentorium the operation of decompression is performed over the right temporal lobe, since, in the event of possible herniation of the brain through the opening, destruction of this part is less likely to be attended by symptoms of serious import than any other. For a subtentorial tumour the bone is removed in the suboccipital region from the superior curved line down to and including the foramen magnum. The operation of decompression, properly performed, relieves headache, and prevents or delays the loss of eyesight which otherwise occurs. Indeed, in a case in which commencing optic atrophy has already led to visual failure decompression may be followed by some degree of recovery of the visual acuity. The mortality of such an operation performed by one expert in the technique is less than 5 per cent

When a deliberate attempt is made to extirpate the tumour the operative mortality even in the most skilled hands is much higher. Cushing's figures for 1920 give a mortality of 15·5 per cent. for seventy-seven cases in which removal was attempted. In the case of a supra-tentorial tumour a flap including the bone is reflected over the site where the tumour is presumed to be, so that if the growth can be removed *in toto* the cranial cavity can be closed. There is always a hope that such an operation may disclose a completely removable endothelioma; if the growth should turn out to be of a more serious nature, the operation can be turned into a decompression by removal of the bone. As an exploratory measure this osteoplastic flap operation is justifiable in all cases in which a localisation can be made.

Gliomas are, as a rule, not completely removable; but much relief may sometimes be gained by the discovery and tapping of a cyst, whose walls may subsequently be in part destroyed by the cautious injection of formalin into the cavity.

Tumours of certain types and in certain positions may be partially extirpated, and when they recur a further operation of the same kind may be performed. In this way Cushing has done much to relieve symptoms and prolong life in the case of the acoustic tumours of the cerebello-pontine angle. The surgical treatment of pituitary tumours has already been considered (*see* p. 595).

Recent experience with deep X-ray therapy suggests that this may be of considerable value in the treatment of pituitary tumours.

Medicinal treatment is limited to partial relief of symptoms. Complete rest, ice-bags to the head, caffein, phenacetin and aspirin in combination, and, lastly, opium are the means available.

## CHRONIC HYDROCEPHALUS

By hydrocephalus is meant the accumulation of fluid within the cranial cavity. An acute effusion is mostly determined by meningitis, either tuberculous or posterior basal, and the former disease was once known as acute hydrocephalus.

A division has been made of chronic hydrocephalus into *internal* and *external* forms, according as the fluid is contained entirely in the ventricles of the brain or is formed outside between the brain and the skull, in the subarachnoid space. It is true that in old age, and from other conditions, the convolutions of the brain diminish in size, the sulci widen, and the space in the skull thus left by the disappearance of brain substance is filled up by fluid. Similarly a loss by local

shrinking of the brain is replaced by fluid on the surface. But this compensatory secretion has none of the effects of true hydrocephalus. True external hydrocephalus is an extremely rare condition, the nature of which is not at present understood, but it is thought to arise as the result of chronic inflammatory changes in the meninges, and as such has also been termed serous meningitis.

Internal hydrocephalus, in which the distension is confined to the ventricles of the brain, may be divided into the congenital and the acquired types.

## CONGENITAL INTERNAL HYDROCEPHALUS

**Ætiology.**—The condition is either noticed at birth or shortly afterwards, and must presumably depend upon some congenital abnormality in the secretion, circulation or reabsorption of the cerebro-spinal fluid.

There is no evidence in favour of the supposition that over-secretion on the part of the choroid plexus is the cause. Cases have been found in which a congenital block has been discovered in the Sylvian aqueduct or elsewhere, in the path between the fount of origin of the cerebro-spinal fluid, which is chiefly in the lateral ventricles, and the venous channels at the base of the brain into which it is reabsorbed (*see* p. 736). There remain, however, a large majority in which no such cause can be found, and the suggestion has therefore been made that the failure is on the part of the channels through which the fluid should normally be reabsorbed, and that the arachnoid villi in these cases are absent or defective.

This hypothesis remains unproven, but some support is lent to the idea by experiments showing that in animals, a small quantity of a suspension of finely divided particles of carbon (lampblack) being introduced into the subarachnoid space, these particles find their way to the arachnoid villi in the walls of the venous sinuses, and by thus blocking the channels for reabsorption of the cerebro-spinal fluid give rise to a picture of internal hydrocephalus in every way resembling that seen in man.

**Morbid Anatomy.**—In this form of hydrocephalus, the ventricles of the brain contain an excess of fluid, sometimes amounting to a quart or more. It has the characters of cerebro-spinal fluid—that is, it has a specific gravity of 1,006–1,009, contains a small quantity of chloride of sodium, only a trace of albumin, and sometimes urea or cholesterin. The liquid may occupy all the ventricles, or all except the fourth, or the two lateral ventricles alone. By its increasing quantity the substance of the brain is enormously distended, the convolutions are flattened, sometimes reduced to a few lines in thickness, and the basal ganglia are correspondingly thinned out. The aqueductus Sylvii may be distended to the size of the finger when the fluid is in the fourth ventricle; it is often closed when the fourth ventricle is not dilated. In extreme cases the distinction between grey and white matter is lost in the parts exposed to most pressure; the ependyma is often thickened, and contains amyloid bodies, while its surface is covered with fine granulations.

Hydrocephalus is sometimes associated with other lesions of the central nervous system, *e.g.* spina bifida or syringomyelia.

**Symptoms.**—The most obvious, and it may be for a time the only, symptom of the disease is the condition of the child's head which results from it. The pressure on the brain is transmitted to the skull, and as this expands outward the head becomes enlarged. The enlargement is often extreme. In congenital cases it may form a serious obstruction to delivery; in others it appears in the first few months of life, and the circumference may amount to 24 or even 32 inches, instead of 16 or 18 inches, which are the usual measurements up to the age of one year. The head is at the same time globular, and the skull projects over the face and neck almost uniformly all round. The face looks small and shrunken in proportion, and has a distressed, anxious, or senile expression in severe cases.

The distension from within drives the orbital plates outwards, and the eyeballs are turned down so that the lower part of the iris and of the cornea is lost under the lower eyelid, and the upper part of the sclerotic is exposed. The increased size of the head is due to a separation of the cranial bones from one another, so that the fontanelles are much enlarged and the sutures widened. In these spaces fluctuation can sometimes be felt. In cases of long standing it is found that ossification has gone on at the margins of the bones, advancing into the sutures, so that ultimately, if the patient lives, by this means and by the formation, from independent centres in the membrane, of fresh plates of bone (*ossa triquetra*, Wormian bones), the deficiencies of the skull may be completely filled in. In the early stages, however, the bones are thin, wanting in diploe, and transparent. The skin of the scalp is tightly stretched, and excessively thin, and large blue veins ramify over the surface. The hair is generally scanty. In some cases, where the fluid is not very abundant, the bones may yield sufficiently to obviate any considerable pressure upon the brain substance. The symptoms may not then go much beyond the enlargement of the head; at most there is some general weakness and loss of flesh, from which after a time the child recovers. But in most cases there are other symptoms. The muscular power is deficient; especially the large head cannot be held upright, and falls from side to side, or has to be supported by the hands when the child sits up in bed. The child cannot walk, or acquires the art in moderate cases only after a long time. Vision is often defective or lost; and in extreme instances there is atrophy of the optic nerves, which has been preceded in some cases, it appears, by optic neuritis. The other senses may be, to a certain extent, impaired. The intellectual functions are often defective. The child slowly learns to talk, continues childish out of proportion to its growth, and is fretful, irritable, or vicious in temper. Nystagmus, rigidity and spasms of the weakened limbs, convulsions, and vomiting occur often in severe cases. Many of these patients die young, relapsing into a condition of apathy or semi-coma, lying in bed with eyes closed or twitching, with rigid limbs and incontinence of urine and faeces, constantly moaning or whining, and refusing food or else eating voraciously.

Finally, convulsions, or coma, or some intercurrent disease, such as bronchitis, pneumonia, or measles, may end the scene.

In some cases the fluid has escaped by rupture of the integuments or by bursting through the nose or eyes.

**Prognosis.** The duration is variable. Few cases survive beyond the third year. In the mildest cases recovery may take place, or rather the disease is arrested. Sir Frederick Taylor has recorded a case ("Clin. Trans.," 1897) where a lad reached the age of sixteen with perfect mental development and physical capacity, and died then with rapid cerebro-spinal symptoms, the ventricles containing 30 ounces of fluid. A few cases have lived to sixty or seventy years of age.

**Diagnosis.** – Confusion is most likely to take place between this and rickets. The rickety head is cubical in form rather than spherical, the vertex being flattened; the downward displacement of the eyeballs is absent; the limbs may be feeble, but the mental powers are not deficient; and the other evidences of rickets – beaded ribs, thickened wrists, sweating of the head, and general tenderness – are present at one time or another.

**Treatment.** – In cases in which the Sylvian aqueduct and foramen of Magendie are not obstructed the distended ventricles may be drained by lumbar puncture. In the other cases in which there is an obstruction between the ventricles and the spinal subarachnoid space, drainage may be effected by ventricular puncture. But the effect is only temporary. Many attempts have been made to effect permanent drainage, but none so far have proved successful. Great care should be taken to prevent the development of bedsores from pressure of the tightly distended scalp upon the pillow.

### ACQUIRED INTERNAL HYDROCEPHALUS

**Ætiology.**—This may occur either in children or in adults, but more commonly in the former. It is most commonly caused as a sequel of basal meningitis, particularly in cerebro-spinal fever (*see* p. 93), by inflammatory adhesions which block the pathway of the cerebro-spinal fluid at its points of exit from the fourth ventricle. A similar blockage also occurs as the result of the mechanical conditions produced by tumours of the brain (*see* p. 851). It is especially likely to be caused by tumours directly blocking the Sylvian aqueduct even in their early stages. In addition there is a small group of cases of acquired hydrocephalus in which no satisfactory cause can be found for the condition.

**Morbid Anatomy.**—The appearances in cases due to cerebral tumour have already been described. In the post-meningitic cases the foramina of Magendie and Luschka are obliterated by inflammatory material.

**Symptoms.** In the post-meningitic cases the condition of hydrocephalus closely follows the infective disease. In infants the enlargement of the head, together with the other symptoms described in the congenital form of the disease, are obvious signs of the condition. In older children and in adults the chief sign may be violent and persistent headache. The symptoms of increased tension accompanying the condition when caused by cerebral tumour have already been described (*see* p. 852).

The rare cases for which no cause can be found when occurring in adults present symptoms of increased tension, which can hardly be diagnosed from those of cerebral tumour. The distension of the third ventricle may give rise to optic atrophy from pressure upon the chiasma, and to paralysis of upward movement of the eyes from pressure upon the superior corpora quadrigemina.

The **Prognosis** depends upon the cause. In cases due to cerebral tumour it has already been discussed. Of the post-meningitic cases some children may eventually recover, but are often epileptics or mentally deficient; in adults the condition may continue for some months and then terminate suddenly in a fatal issue with failure of respiration. In the cases of obscure origin (primary acquired hydrocephalus), the outlook is more hopeful: there may be remissions or intermissions lasting in some cases for years.

**Treatment.** In the post-meningitic cases ventricular puncture and drainage have been recommended with subsequent introduction of anti-meningococcal serum. In those due to cerebral tumour the treatment has already been considered. In primary hydrocephalus a decompression may be necessary to save vision or life.

## GENERAL PARALYSIS OF THE INSANE

*(Encephalitis Syphilitica)*

Although mental diseases do not come within the scope of this work, it is desirable to describe this complaint, because it depends upon actual structural changes in the central nervous system, and because the paralytic symptoms are often the prominent features of the case for long periods of time, and may give rise to a difficulty in diagnosis from other purely physical conditions.

Shortly stated, the disease consists in progressive symptoms, partly of a paralytic, partly of a mental, character, terminating in dementia and complete loss of power, and dependent upon widely spread anatomical changes in the brain, spinal cord, and nerves.

Dementia paralytica is in every case caused by syphilitic infection. A history of syphilis is obtained in about three-quarters of the cases, and when the disease occurs in children or quite young persons, there has been syphilis in a parent. In the later stages of the disease the Wassermann reaction can be obtained from the blood and cerebro-spinal fluid in every case, and careful search with suitable

staining methods has now in many cases revealed the presence of the *Spironema pallidum* in the brain. Predisposing factors appear to be alcoholic excess and mental or physical stress, but syphilis is the *conditio sine quâ non.*

The disease is much more frequent in men than in women, and occurs mostly between the ages of thirty and fifty, five to fifteen years after infection.

**Morbid Anatomy.**—The lesions are very variable, but the following are found in different cases: thickening of the calvarium, which is deeply pitted by the Pacchionian bodies; thickening of the dura mater, with false membranes (pachymeningitis); abundant subarachnoid fluid, with thickened or adherent membranes, the adhesion when present being more over the frontal, parietal, and temporo-sphenoidal lobes, and more on the upper than the lower surface; wasting of the convolutions, especially the ascending parietal, paracentral, and first frontal at its base; a violet-red colour of the cortex of the brain; in some cases much fluid in the lateral ventricles, with softening of the brain tissue; in a larger number, a general hardening of the brain. Beneath the lining walls of the ventricles there is frequently an irregular overgrowth of neuroglia which gives the surface a granular or frosted appearance; this is often best marked in the floor of the fourth ventricle. In the spinal canal the same changes may be found: pachymeningitis, or adhesion of membranes, or effusion of blood within the dura mater. The spinal cord is wasted, or presents the lesions of posterior or of lateral sclerosis. Microscopic examination shows the following changes: The pia mater is infiltrated with lymphocytes, plasma cells and mast cells. The brain shows proliferation of the neuroglia especially in the superficial layers of the cortex, with large glia cells, new-formed blood vessels, thickening of the intima and adventitia of the vessels and infiltration of their lymph sheaths with lymphocytes and plasma cells, destruction of the myelin sheaths of the nerve fibres, and degeneration and sclerosis of the ganglion cells, especially of the pyramidal cells of the third layer. The sympathetic ganglia are, according to Savage, not appreciably affected. The disease is thus a chronic meningo-encephalitis with increase of connective tissue and degeneration of the neurons; the origin in syphilis, the association with the spinal sclerosis, and other facts make it highly probable that the degeneration of the nerve elements is primary (Mott). The spirochæte of syphilis has been found in the cerebral cortex.

**Symptoms.**—Considerable difference is seen in the grouping of the symptoms; the nature of the mental changes in the early stages is variable, and there is no parallelism between physical and mental symptoms, nor between individual physical signs.

The disease is frequently ushered in by epileptiform or apoplectic attacks. The former may take the form of transient phases of unconsciousness like those of *petit mal*, of which the patient has no knowledge, and which may pass unnoticed for some time. The latter may leave in their wake incomplete palsies of monoplegic or hemiplegic type.

More frequently the earliest symptoms appear in insidious changes in the personality. The man becomes careless or neglectful, tends to intemperance in drink, or spends more money than has been his wont without justification, or he is irritable or restless, changing in his affection towards his wife or family, or jealous without a cause. On the other hand, the early picture may be simply that of neurasthenia, with complaint of abnormal fatigue, sleeplessness and irritability. Headaches may be a prominent symptom, depending, no doubt, upon the meningeal involvement. The intellectual faculties are as a rule involved moderately early. There are defects in memory for the remote as well as the recent past, with consequent discrepancies in the dating of events in the patient's history. As a rule he possesses no insight into these defects, and will maintain that his memory is as good as ever. Disturbances of mood are frequent, the commoner type being that in which the patient is extremely elated, active and voluble, with delusions of grandeur. These concern himself alone; they express

what he is, what he possesses, or what he can do. He is the Almighty, the King of England, or the Prime Minister, the most handsome or the most powerful man in the world. He has invented the most ingenious devices for making money, and is already possessed of boundless wealth. On the other hand, the dominant mood may be one of extreme depression, characterised by an extravagance of delusion which helps to differentiate it from a simple melancholia. In this condition the patient may attempt suicide. Yet another type of onset is that which shows itself in progressive apathy and deterioration of interests without any underlying disorder of mood.

On the physical side the facial muscles are smoothed out and expressionless, and there is commonly a tremor of the lips and cheeks in speaking. Speech is slovenly and slurred, with a tendency to miss syllables in the pronunciation of difficult phrases (such as "Methodist Episcopal"). There is a fine tremor of the protruded tongue, and there may also be atrophy from involvement of the hypoglossal nuclei. Pupils of the Argyll-Robertson type may be seen early in the illness, and are seldom absent in the terminal stages. Tremor of the outstretched hands is commonly seen, and shown also in the handwriting. The condition of the tendon reflexes in the early stages varies with the degree of involvement of pyramidal and posterior column fibres. Extensor plantar responses may be found either with exaggerated or absent knee and ankle jerks.

In the later stages there is wholesale deterioration, both mental and physical, with loss of sphincter control, and the patient dies as a rule from cystitis, pneumonia or other intercurrent infection.

Dementia paralytica is found, though rarely, in children the subjects of congenital syphilis, either manifesting itself from the first in mental deficiency or giving rise to progressive deterioration at or after puberty.

**Diagnosis.**—The mental condition in *cerebral arterio-sclerosis* may simulate that of dementia paralytica, but in the former disease the memory defect is more usually confined to recent events, and is recognised by the patient. *Alcoholism* may be mistaken for general paralysis, the tremor of the lips, tongue, and hands largely contributing to this; commencing *peripheral neuritis* might further complicate the case. But the close association of the symptoms with continued drinking, the absence of inequality of the pupils, and the improvement on prolonged abstinence, would point to alcoholism. Mental failure with definite cerebral lesions such as *tumours*, or the dementia resulting from bilateral vascular lesions (*pseudo-bulbar palsy*), may give rise to difficulties. Certain cases also of *disseminated sclerosis* with early cerebral involvement may require careful differentiation.

From the general physician's point of view, it is important to recognise that various anomalous paralytic symptoms may be the first symptoms of general paralysis. If a case is typically tabes dorsalis there is no special reason to anticipate mental trouble; but if the symptoms develop very rapidly or present unusual groupings, or if there are mixed symptoms not conforming to the ordinary types of the spinal cord diseases, the mental condition should be closely scrutinised, and the possibility of general paralysis of the insane should be kept in view. The diagnosis may be made with certainty on the basis of laboratory findings. The cerebro-spinal fluid in every case shows a positive Wassermann reaction, though that in the blood is frequently negative in the early stages. The fluid also contains usually an excess of lymphocytes and a moderate increase in albumin, and with the colloidal benzoin test gives the so-called "paretic curve" (*see* p. 738).

**Prognosis.**—This in an undoubted case is hopeless; the fatal termination may be looked for in from two to five years from the time when the diagnosis has been made. The disease, however, even when untreated, is not always steadily progressive, and there may be remissions during which the patient is able to return to some of his former activities.

**Treatment.**—Although there is no case on record of the cure of a case of dementia paralytica, there is some evidence to show that the disease process may be retarded by energetic antisyphilitic treatment, which should be commenced immediately on the lines laid down for syphilitic myelitis (*see* p. 776). In some cases treated in this manner remissions have occurred of a duration quite unusual in untreated cases, in which for a time the patient has been able to fill a useful place in society.

More recently attempts have been made to treat the disease by inoculating patients with malarial parasites, allowing them to experience several bouts of fever and finally curing the malaria by means of quinine. It is hoped that the reaction provoked by the malaria, which is like syphilis a protozoal infection, may have a beneficial effect upon the disease. This line of treatment is still in the experimental stage, and although some favourable results have been reported, in view of the long remissions which may spontaneously occur in the disease, it is yet too early to pronounce judgment upon its worth. Its dangers are by no means negligible. A number of deaths from malaria have been reported.

From the broader standpoint of the patient's friends and relations and the general community early diagnosis is most important, that all may be warned and the patient be committed to a proper institution before he bring ruin upon his own fortunes and reputation, and endanger the welfare of others.

## CHOREA

(*Sydenham's Chorea, Chorea Minor*)

Chorea (χορεία, a dancing) is characterised by irregular involuntary movements of different parts of the body. The popular equivalent, St. Vitus's dance, has reference to the occurrence in the Middle Ages of epidemics of dancing mania, when patients were cured by a pilgrimage to the shrine of St. Vitus Chorea Sancti Viti. But the complaint in those epidemics partook rather of the nature of hysteria, and though the name *chorea* is still sometimes used to indicate some other forms of abnormal movement, it is, as a rule, reserved for the disorder now to be described.

**Ætiology.**—It is mostly a disease of childhood: nearly half the cases occur between the ages of five and ten, and another third between ten and fifteen. It is more frequent in girls than in boys, in the proportion of two or three to one, and it is more common among the poorer classes of society. It is not strongly hereditary in its ordinary form. Among antecedent diseases acute rheumatism is the most important. About one-third of choreics have had rheumatic fever; choreic movements sometimes occur in the course of rheumatism, or rheumatic pains during chorea. In some other cases of chorea the attack has been preceded by one of the infectious disorders, such as scarlet fever or measles, or some other septic disorder. Among adult patients pregnancy is a common antecedent; some of them have had rheumatism, and others chorea in childhood. Fright or mental shock of some kind appears to be an aggravating cause of the disease in many cases, though parents are often too ready to account for the attack in this way. It may arise after injury, perhaps also as a result of emotion.

**Morbid Anatomy.**—The nervous system after death does not present to the naked eye any morbid appearances; but among the microscopic changes may be noted obstructions of minute vessels in the brain, small foci of softening, swelling and degeneration of nerve cells in the corpus striatum and other parts, enlargements of the perivascular spaces, and hæmorrhage around minute vessels. An excess of lymphocytes in the cerebro-spinal fluid has been found on lumbar puncture. Of the other organs of the body the heart is the only one that is generally involved, and in fatal cases of chorea this nearly always presents evidence of endocarditis (seventy-five out of eighty cases, Sturges; seventeen out of

eighteen cases, Fagge). Fine granulations are found along the edge of the mitral valve on the auricular face, and sometimes on the aortic valves. These are present even when there has been no antecedent rheumatism. Occasionally the valvular lesion is older and more extensive.

**Pathology.**—Chorea certainly has a toxic origin, or is dependent on infectious disease. The facts in favour of this are—the frequent occurrence of endocarditis and its almost universal presence in the fatal cases; the association with rheumatic fever, and possibly other diseases of infectious nature; and the mode of death, which is by no means always explained by simple muscular exhaustion, for the patient may lie for some hours before death perfectly tranquil, and give the impression that convalescence has begun. The same thing happens in tetanus and hydrophobia, in which also muscular movements are determined by the presence of poisons, but the nerve lesions, if demonstrable at all, are only microscopic. Choreic movements have been produced in rabbits by the injection of Poynton and Paine's rheumatic diplococcus into the veins. Other observers have isolated pyogenic organisms from choreic cases. The localisation of the disease in the brain is shown by its frequent connection with emotional disturbance, the influence of the will, of emotion, and of distraction of the attention upon the movements, their cessation during sleep, their frequent limitation to one side in the limbs, while they affect both sides of the face and trunk, and the coincident disturbance of the mental faculties.

This is confirmed by the clinical evidences of organic disease above recorded, and by the scattered histological changes hitherto looked upon as accidental or secondary. It is clear that chorea can now scarcely be included amongst functional disorders, and it may be possible to regard it as a form of infective encephalitis. Additional support to this view has been lent not only by the microscopical researches described above, but by the occurrence of chorea as a part of the clinical picture in some cases of encephalitis lethargica.

**Symptoms.**—The most prominent feature of the disease is the action of the muscles: they are in a condition of (1) involuntary movement, (2) ataxy or incoordination, and (3) slight degree of actual weakness or paresis. The patient is in a constant state of movement, whether lying, sitting, or standing; and the movements, which affect nearly all the muscles of the body, are jerky, irregular, and devoid of purpose. The fingers are opened and shut, the wrist suddenly extended or flexed, or the shoulder lifted. The facial muscles are twitched, the eyebrows suddenly elevated, the head or the eyes rotated to one side, and the chin elevated or depressed. In the lower extremities the movements are often less; the toes are twitched, or one knee gives way. In the muscles of the trunk, one notices half-rotation of the body to one or other side, sudden retraction of the abdomen, or jerky action of the respiratory muscles.

The irregularity is more marked on voluntary movements. If the hands are stretched out in front, the child is quite unable to hold them steady; on protruding the tongue, it is put out with a jerk, and perhaps withdrawn suddenly, and the muscles of the jaws act capriciously at the same time; in walking the legs are thrown about, the body is jerked round, and the shoulders are lifted. In the same way it may be seen that the muscles relax with great readiness; after grasping an object, one or two fingers quickly yield, and soon the hand and arm will drop. The movements are increased when the patient is watched, or if she becomes excited; they cease during sleep.

The vocal cords have been seen to quiver, and a low-pitched, monotonous voice is attributed to their want of tension. Speech is irregular, and the patient is unable to sing a long note; these defects may be due to the irregularity of the respiratory movements.

In some cases there are one or more of the numerous signs which are accepted as indications of disease of the pyramidal tracts or of the cerebellum. These are especially demonstrable on the weaker side in cases of marked hemichorea.

Sensation is but little disturbed; there may be some formication or tingling, but very rarely any definite hyperæsthesia, or anæsthesia. Both nerves and muscles show increased irritability to faradic and galvanic currents.

It is not always easy to say what the condition of the mind is: often a child with chorea looks silly or idiotic from the purposeless contractions of the facial muscles, which in this case are not a true index of the mind. Apart from this, however, the child's disposition is apt to be altered; she becomes fretful, irritable, capricious, or excited, while intellectually she has a weak memory and is unable to fix the attention.

In about half the cases a murmur over the heart's area may be recognised. In children irregularity of the heart-beat is mostly due to sinus arrhythmia; but other irregularities, and partial heart-block in particular, may be present (*see* p. 304). The murmur is commonly heard at the apex of the heart, and is systolic in time. Mostly it is limited to this area; occasionally it is audible in the axilla and behind, and is obviously due to mitral regurgitation. Sometimes a hæmic basic murmur is present. The origin of the apex murmur has been much discussed, but since endocarditis has been frequently found in fatal cases, and some of the murmurs of chorea are undoubtedly due to a valvular lesion, it is fair to suppose that in other cases they arise from endocarditis or myocarditis. Some, indeed, may be the result of preceding rheumatism; but this will not account for the majority, which appear to develop in the course of the chorea itself.

**Varieties.**—Sometimes the symptoms are very slight, and remain so for some time; the fingers are only twitched a little, irregular movements are scarcely noticed, but the child drops things that she attempts to carry. In some cases the movements are limited to the arm and leg of one side only (*hemichorea*). In others there is decided paralysis, with only slight choreic movements; the arm hangs by the side, and can with difficulty be raised; the fingers are twitched occasionally, and the grasp is extremely feeble (*paralytic chorea*).

Exceptionally the movements are very violent; standing or sitting is impossible, and the patient is confined to bed, where she throws herself about in the wildest contortions, striking the hands and arms against the sides or head of the bed, and rubbing the elbows, shoulders, buttocks, hips, knees and heels, so as to produce serious abrasions of the skin. Feeding becomes difficult or impossible, as everything placed to the mouth of the patient is jerked aside or spilt; and even if it gets into the mouth it may be rejected by the want of co-ordination for deglutition. These cases (*chorea gravis*) sometimes progress with great rapidity: the patient appears to be exhausted by the constant movement and the want of sufficient nutriment; rapid emaciation takes place, the face is flushed, the eyes sunken but bright, the lips and tongue dry, the pulse rapid, and ultimately death may occur, being preceded often by some rise of temperature and by cessation of the movements. In some the mind is severely affected, and the patient becomes delirious, or even wildly maniacal. Such violent cases are much more frequent in adults between the ages of fifteen and twenty-five, and a large proportion are in pregnant females.

**Duration.**—The duration of chorea is very variable. The majority of cases last from six weeks to three months; not infrequently slight twitching may occur for many weeks or months after the severer manifestations have subsided, and the symptoms may again after a time become aggravated. In the end most cases recover. The violent cases are usually of short duration; if death takes place, it is often within two or three weeks from the first symptom, or from the time when the movements become violent; if recovery ensues, the movements become quieter after a few weeks, though complete cure may be delayed some time. Chorea is very apt to recur even after its entire subsidence; second and third attacks are frequent. These may be of shorter duration than the primary attack, but are not different in other respects.

**Sequelæ.**—The disease sometimes leaves behind it a liability to sudden starts, which in the course of months subside. In some cases towards the end of the attack paralysis of the limbs occurs. This may be only on one side (*choreic hemiplegia*); but rarely all four limbs are affected, the child lying quite helpless, and each limb dropping like a log on being raised from the bed. Speechlessness, mental weakness, maniacal and melancholic conditions, also occasionally occur, and are generally temporary. Epilepsy has also been observed as a sequel of chorea. The endocarditis may terminate in chronic valvular disease.

**Diagnosis.**—This rarely presents any difficulty. Movements closely resembling those of chorea may occur as a part of *hysteria;* they are generally more rhythmical, more localised, and may recover quickly. *Habit spasm* may be present in children, and is closely allied to the above; the movements are localised, voluntary in character, more under control and less constant than those of chorea. There are jerky movements in *Friedreich's ataxia;* but the gait is different, the history is a very long one, and other signs are present.

**Prognosis.**—In children it is favourable, apart from the condition of the heart; in young adults it is much more uncertain.

**Treatment.**—The child should be kept quiet in bed, and everything tending to worry or annoy should be kept from her. She should not be subject to the ridicule of companions, nor to much study of lessons. The diet should be plain, nutritious, and abundant. Antipyrin (5 to 7 grains), aspirin (7 to 10 grains), and chloretone (5 grains) three times a day are beneficial. Arsenic is usually given, but there is no evidence that it is of any value. In the very violent cases the patient must be protected from injury by padded boards at the side of the bed, the nutrition must be maintained, and food may have to be given through a nasal tube. To procure rest and sleep, chloral is probably the best drug, but it must be given with caution; morphia is undesirable. Trional in 10 or 16-grain doses has been used with success in both these and the milder cases. Quiet may be obtained for a time by inhalations of chloroform, but the movements return as the anæsthetic effect passes off. In cases of paralysis after chorea Sir Frederick Taylor has seen good results from strychnia. When the movements are slight in mild cases, or in recovering stages, exercises may be found useful.

### HUNTINGTON'S CHOREA

This is a rare disease *sui generis* to be sharply distinguished from the form of chorea already described, though in some ways it resembles it. It is a hereditary affection of middle life, of progressive nature, leading to dementia.

**Ætiology.**—Heredity plays the most important part in the disease. It is transmitted from one generation to another, though occasionally the members of one generation may be spared. Men and women are equally affected, and the disease usually begins between the ages of thirty and forty. As the result of a genealogical research conducted in the United States of America 900 cases of the disease were traced back to three brothers who landed in America from England early in the seventeenth century.

**Pathology.**—In certain cases carefully examined chronic diffuse or disseminated inflammatory changes have been found in the brain and meninges, but the origin of the process is not clear, and the exact pathology remains uncertain.

**Symptoms.**—The cardinal symptom is motor restlessness of a type similar to that seen in the chorea of children. There is a constant and rapid succession of purposeless inco-ordinated movements of all kinds, affecting all parts of the body. These movements show a tendency to affect the proximal groups of the limb musculature to a greater extent than occurs in the chorea of children. The limbs are consequently thrown about with considerable force, and the gait is grossly affected. The movements may be temporarily inhibited by voluntary effort.

Mental changes are constantly associated with the disease, at first in the form of irritability and restlessness, sometimes leading to suicidal depression, and finally terminating in progressive dementia of organic type, so that these patients, as a rule, end their lives in mental institutions.

The **Prognosis** is hopeless, the duration of life being from ten to thirty years.

The **Treatment** is symptomatic only.

# NERVOUS DISEASES OF UNCERTAIN PATHOLOGY

Under this heading will be described certain diseases in which the signs and symptoms point to disturbances of function in the nervous system, for which, however, no structural basis can be found *post mortem* either with the naked eye or by microscopical examination. These diseases are sometimes termed *functional nervous diseases*, to distinguish them from those in which a definite organic basis can be found. Some of them probably depend upon minute structural changes in various areas which have so far escaped recognition; others (the psychoneuroses) result from a temporary failure of the organism as a whole to react normally to environmental conditions.

## EPILEPSY

(*Idiopathic Epilepsy, Genuine Epilepsy*)

Epilepsy has already been spoken of as a *symptom* of certain diseases of the brain, particularly those which affect the cortex (*e.g.* cerebral tumour, encephalitis, syphilis of the brain), and also of some toxic states of which uræmia is the chief example. The attacks provoked by such causes differ in no way from those presently to be described. Epilepsy in all cases probably depends upon disordered function of the cerebral cortex, whether this is due to congenital defect, to local disease or injury, or to the effect of circulating toxins with a selective incidence. But in the very large majority of cases we are unable by present methods to discover any such cause. The attacks continue indefinitely without the appearance of any further signs of disease, and if an autopsy be obtained microscopic examination reveals no abnormality of the brain. It is this group of cases which, in our present ignorance of their pathology, we are obliged to group together under the symptomatic title of epilepsy.

*Epilepsy, therefore, may be defined as a disease in which there are attacks of sudden loss or impairment of consciousness, with or without convulsions, independent as far as our present knowledge goes of any demonstrable lesion of the brain, or state of toxæmia.*

**Ætiology.**—The disease is rather more common in females than in males. In about one-third of all cases there is a history either of epilepsy or of insanity in the family. Three-quarters of the cases begin under the age of twenty. After this the tendency to develop the disease gradually diminishes with advancing years. Yet epilepsy, being on the whole a common disorder, it is by no means rare to meet with cases commencing in adult life or even in old age.

In a small percentage of cases the seizures appear to be directly related to previous injury to the head which may or may not have been associated with symptoms of "concussion." The interval between the injury and the first attack may cover several years. The clearest proofs of such relationship are cases in which the attacks commence with symptoms directly referable to the site of the injury, as in the case of a gunshot wound over the left motor cortex, and subsequent

fits beginning with convulsive movements in the right side of the body. Even in these cases, however, the hereditary influence is sometimes apparent. Of 18,000 men who sustained serious head injuries during the Great War only 800, or less than 5 per cent., had developed epilepsy three years after its conclusion, and among these the proportion with an epileptic family history was higher than in the remainder. Occasional causes for the attacks are sometimes to be found. Chief among these are mental fatigue, excitement, or anxiety. Lack of sleep, physical fatigue, and fasting may also precipitate attacks. In some cases there is a definite relation to menstruation—the attacks occurring as a rule at the onset of the period.

**Pathology.**—By the definition given above all cases are excluded in which a pathological cause for the epileptic attacks can be found. We are limited, therefore, to speculation. Arguing from the known causes to the unknown, we have reason to believe that epilepsy depends either (1) upon defective function of the cerebral cortex, congenital or acquired, or (2) upon the presence in the body of poisons with a selective action upon the cortical nerve cells

**Symptoms.**—The essential feature of epilepsy is a disturbance of consciousness, which usually, but not always, amounts to complete loss. With this in the majority of attacks there are associated convulsive movements.

It is convenient to distinguish between "major" and "minor" forms, according to the severity of the convulsive movements, but there is no absolute line of distinction.

In *Major Epilepsy* or "*Grand Mal*" there are usually to be discerned several stages: (1) aura; (2) unconsciousness and tonic contraction; (3) clonic convulsion; (4) recovery. The *aura* (or breath, from the sensation of air passing up the limb to the head, which is one form of this symptom) is any sensation or motion experienced by the patient while he is yet conscious, mostly of very short duration, and terminating abruptly in loss of consciousness and convulsion.

1. The aura is absent in about one-half of the cases. In the others it may assume a variety of forms, of which the following may be mentioned as of common occurrence: tingling and numbness in the arm, leg, face, or tongue; twitchings or spasms in the same parts; loss of vision, or visual hallucinations, such as flashes of light, or colours (generally red or blue) or definite objects or enlargement of surrounding objects (*megalopsia*); hallucinations of sound, noises, etc.; unpleasant odours or tastes; sensation of choking, nausea, vertigo, epigastric pain; flushes of heat, coldness, perspiration, palpitation of the heart; an indefinite sense of fear or anxiety; a *dreamy state*, or sense of unreality, or the feeling that what is happening has occurred before; running or jumping, or other co-ordinated movement. Auræ of sensation and motion are mostly unilateral, but may be bilateral; the arm is more often affected than the leg, and facial auræ mostly consist of spasm. Visual auræ are much more common than auræ of the other special senses. Sometimes a vague sense of fear may last some time before the occurrence of the actual fit; but, as a rule, the aura is of momentary duration.

2. The fit itself commences with sudden unconsciousness; if standing or walking, the patient often falls suddenly forwards, or seems to be thrown violently to the ground, sometimes with an involuntary cry, shriek, or low tremulous groan—the epileptic cry. He is then found to be in a state of tonic convulsion, the back rigid and slightly arched, the legs extended, and the head drawn backwards or rotated to one side. The face is often pale at first; the pulse is quick, but sometimes it cannot be felt, and this has been attributed to compression of the artery by muscular contraction; the pulse has also been observed to cease at the moment of unconsciousness. The general tonic contraction fixes the chest, and respiration is stopped, so that the face becomes more and more dusky, and eventually is quite cyanosed. The tonic stage lasts from three to thirty or forty seconds, and then passes into the stage of clonic convulsions.

3. Twitchings begin in the face, the eyelids, and the side of the neck, and quickly extend to all the muscles of the body and limbs. There is a rapid succession of to-and-fro movements, of alternate flexion and extension in the limbs, of opening and shutting of the eyelids and of the jaws, lateral deviation of the eyeballs, and perhaps of the head; the tongue is pushed forward, and may be caught between the teeth; saliva is freely secreted, frothed in the mouth, and escapes from the lips mixed with blood from the bitten tongue. The face becomes livid, or almost black, and the lips and features are swollen. Urine, fæces, and semen may escape during this stage, and the violent contraction of the muscles may even cause dislocation of the shoulder. The patient is, of course, quite insensible; the conjunctivæ do not respond to a touch, and the pupils are dilated or oscillate.

4. The clonic stage lasts a few minutes, rarely more than five or six, and then the convulsions gradually subside—they become less frequent, and are interrupted by pauses of some seconds; the breathing becomes easier, the frothing at the mouth ceases, and the face gradually assumes a more normal colour. Finally, the patient remains simply comatose, and the coma passes into natural sleep, or consciousness is recovered rather suddenly soon after the cessation of the convulsions. When consciousness is finally regained the patient is as a rule dazed and complains of headache.

The reflexes are mostly absent for a short time after the attack, and then for a time the deep reflexes may be increased. The plantar reflex after a temporary absence is at first extensor, then again normally flexor. The urine may contain a trace of albumin or sugar; petechiæ may be seen under the skin from rupture of blood-vessels during the stage of venous congestion; sometimes there are vomiting, or serious mental disturbances, such as delirium, which is often of a maniacal kind.

The mechanical injuries from which the epileptic suffers will, of course, remain after the fit, and may give valuable indications in cases where the fit has not been seen - for instance, in nocturnal epilepsy. These are the bitten tongue, petechiæ on the skin, a dislocated shoulder, and, in other cases, various cuts, wounds, or bruises, from the falling of the patient upon the ground or against unyielding objects.

*Minor Epilepsy* or "*Petit Mal.*"—This consists, in a large number of instances, of little more than a sudden unconsciousness; in the midst of talking, perhaps, the eyes become fixed, the pupils dilated, the speech incoherent, and the patient is obviously unconscious of what is going on around him; he may, if at meals, put his fingers in his plate or his cup, or commit some other irregularity that he would not do if conscious. The condition lasts a few seconds, and then he becomes conscious, and goes on with what he was doing, or perhaps recognises that there has been a blank, or feels giddy, or has headache, and is glad to lie down for some time. Sometimes giddiness is the most marked feature of the attack, and in other cases a sensation in one or other part of the body, or a spasmodic movement, which may be quickly followed by temporary unconsciousness, though the former will seem to the patient the chief feature of the attack. These have a close resemblance to the auræ of the major attacks, and include sensations in the epigastrium, hands, head, nose, eyeballs and cardiac region; olfactory and visual sensations; jerks in the limbs, head, or trunk; sudden tremor, screaming, or dyspnœa; mental conditions, such as a sudden state of fear, etc.

In many instances there is no complete loss of consciousness. The patient is aware of his surroundings and often of what is being said by those around him, but everything for the moment appears distant, he fails to comprehend the full significance of events and is incapable of formulating his thoughts clearly.

**Post-epileptic Conditions.**—The attack is generally followed by a period of drowsiness and often by severe headache. A serious psychical disturbance is a

not uncommon sequel to an epileptic fit, and follows the minor attack even more frequently than the major. It may take the form of stupor or amentia lasting some days. In another case various *automatic* actions occur of which the patient is then and afterwards entirely unconscious. Perhaps the commonest of these is the act of undressing, which in a public place may lead to a charge of indecent exposure, or the patient may put in his pocket any object which happens to be near, irrespective of its ownership. Occasionally he may be violent and aggressive, and while still in the automatic state cause bodily injury to others. These cases have great medico-legal importance, since the occurrence of epileptic fits may be quite unknown, and the criminal behaviour may be attributed to wilful and conscious action. Sometimes these attacks are maniacal in their character (*epileptic mania*), and the automatic actions are accompanied with much mental disturbance, such as terror, violent passion, delusions, and hallucinations.

Equally important, on account of the difficulty in diagnosis which they occasion, are minor psychical sequelæ. The patient recovering from an attack of true epilepsy frequently behaves in a childish, emotional manner, weeping and laughing alternately, and will sometimes pass into a hysterical "fit."

**Varieties.**—Though we can generally distinguish between the major and minor attacks, there are attacks which present intermediate characters. The two forms are often only different phases of the disease in the same person; thus it is not uncommon for children to suffer first from minor epilepsy, and as they grow older to develop the major attacks. They may both occur in the same patient alternately, or more or less irregularly; and in patients in whom the fit is preceded by an aura the aura may occur alone on some occasions, and on others it may be followed by some only of the features of the attack, which stops short of its complete development.

**Course of the Disease.**—The frequency of epileptic attacks varies considerably in different cases, and at different periods in the same case. Thus there is generally an interval of one or more months between the first and second attacks, but with the progress of the disease the intervals often become shorter, and the fits may be as frequent as two or three in a week, or even several in a day. In some cases two or three fits occur in quick succession, or at short intervals, and the patient is then spared for a long time. A severe fit is much more likely than a slight one to be followed by a long interval. Probably alcoholic indulgence, injudicious feeding, and mental or physical over-exertion, increase the frequency of the fits. In some female epileptics the attacks come on with each menstrual period.

**Status Epilepticus.**—In rare instances the patient has a series of fits, extending over some hours, or one or two days, and never recovers consciousness in the intervals between them. The heart beats violently and rapidly, the respirations are quick, twitchings occur in the intervals of the convulsions, the temperature often rises to 105° or 107°, and the patient may die collapsed, or may become delirious.

**Health between the Attacks.**—This depends a good deal upon the frequency of the fits. Where these are not numerous, the individual may enjoy excellent health. Many epileptics are strong, hearty, and vigorous, never ailing at all except at the time of the attack. When, however, the fits are very frequent, or the disease has lasted a long time, the mind generally suffers, the patient becomes dull and irritable, the memory is deficient, and intellectual processes are slower, until eventually a condition of *dementia* is reached. In children, sometimes, even after a few fits, permanent imbecility or mania may be developed.

**Death from epilepsy** is by no means common, and, except in the case of the rare status epilepticus above described, it is mostly the result of some injury to which the patient is exposed during the fit. Thus during a fit he may be thrown from a height, or fall into water and be drowned, or be choked by food, or he may be

smothered in bed by his face being buried in the pillow, or he may die later from injuries received by a fall into the fire, or from a carriage or bicycle.

**Diagnosis.**—Epilepsy is with no great difficulty recognised when actually seen, but one is often called upon to prescribe for fits which only occur at times when the physician cannot witness them; and it is not always easy to come to a right conclusion from the descriptions of friends. The major attacks have to be distinguished from attacks of hysteria, and from simulated fits, minor epilepsy from attacks of syncope or vertigo. Hysterical attacks seldom, if ever, occur when the patient is alone or unlikely to attract an audience. The onset is seldom so dramatically sudden as in epilepsy, and the attack is usually related to an emotional stimulus. The movements in the hysterical fit never consist of simple tonic or clonic jerkings, but are of a more purposive character. Thus the patient will clutch at those around her, dash her head against the wall or floor, clench and unclench the hands, or bite at those who seek to restrain her. The convulsive movements of epilepsy are over in a few minutes. The motor phenomena of hysteria last as a rule much longer. The hysterical patient frequently falls during the attack; the epileptic never. The hysterical patient never falls so as to hurt herself severely, nor does she bite her tongue or pass water in the attack. In the hysterical fit the eyelids are often tightly closed, and attempts at opening them are resisted. The occurrence after the fit of mental disturbances (*see* p. 870) and the exaggeration of deep reflexes with loss of abdominal reflex are in favour of epilepsy, while an extensor plantar response obtained after the attack is conclusive evidence of its epileptic nature.

It must not be forgotten that an attack of epilepsy may be immediately followed by one of hysteria, and that the same patient may at different times exhibit epileptic and hysterical fits.

The *malingerer*, who attempts to excite sympathy as a sufferer from epilepsy, can, with a little care, generally be detected. He is careful to fall so as not to hurt himself, whereas the epileptic is thrown down suddenly, and if in the street will probably strike his head or face, or will fall in the road, not making any effort to save himself. The malingerer is red in the face rather than pale or livid; his skin perspires from the exertion; his pupils are not dilated and are sensible to light. The fact that he has not lost consciousness may be tested in various ways: by touching the conjunctiva, when the eyelids will close, though he will probably resist attempts to raise the upper eyelid; by applying snuff to the nostrils; by producing some very painful impression, as by forcing one's thumb-nail under that of the malingerer.

Minor epilepsy, or *petit mal*, is distinguished from *cardiac syncope*, or simple fainting, by its occurrence under circumstances not conducive to fainting, by its suddenness, and by its rapid recovery, followed by mental confusion rather than physical prostration. The occurrence of spasm or of any warning sensation other than the feeling of faintness is in favour of epilepsy. Syncope comes on more slowly, and is recognised as a gradually increasing faintness by the patient. Still this may happen as a warning sensation of *petit mal*. To distinguish other forms of *giddiness* from the vertiginous form of minor epilepsy, one must remember that ordinary vertigo is not accompanied by loss of consciousness, and that in the aural form, or Menière's syndrome, a history of tinnitus and deafness may be obtained.

When it has become certain that, in any case, the convulsions are really epileptic, it has yet to be determined that they are not due to tumour of the brain or cerebral syphilis, or the uræmia of Bright's disease, before one can pronounce the disease to be epilepsy. In a great number of cases of idiopathic epilepsy, the long history of recurring convulsions with no associated symptoms will serve to distinguish it, whereas in cerebral tumour there will probably be other indications, such as headache, vomiting, papilloedema, or local paralysis. Nevertheless epileptic attacks may for many months be the only symptom of this disease.

Attacks of epilepsy frequently usher in encephalitis syphilitica (general paralysis of the insane), and in the absence of all other signs may be the first warning of the disease. For this reason epilepsy commencing in a man of middle age must always be suspect. The pupils and mental state must be carefully examined and the Wassermann reaction obtained if necessary in the spinal fluid as well as in the blood. In *Bright's disease* the convulsions are epileptic, but the patients, as a rule, show good evidence of their state of health in albuminuria, high tension of pulse, hypertrophy of heart, raised blood urea preceding uræmia; the fits are often ushered in by drowsiness and muscular twitchings, are of much longer duration, and recur frequently in the same day with intervals of drowsiness or semi-coma.

It remains to be mentioned that nocturnal attacks of epilepsy may be for a long time unrecognised, if they are not actually witnessed by any one. They may be suspected if a boy or girl not suffering from nocturnal enuresis, and beyond the age at which that is usual, unexpectedly wets the bed, or if there are petechiæ on the face or body, or a sore tongue which the patient cannot account for, or headache or dulness, and a feeling of being unrefreshed.

**Prognosis.**—Epilepsy rarely recovers without treatment, and the hope, so often entertained by the patient's friends, that attacks beginning in youth will cease with the development of puberty, or with the appearance of the menses, is very unlikely to be realised. They can, however, be very markedly controlled by treatment, and are generally the more amenable the later in life they have begun. According to Gowers, the prognosis is better if the fits occur only during waking, or only during sleeping hours, and not under both circumstances; if there is no considerable mental change; if the attacks are only of the major kind, and not both major and minor; and it is better if there is an aura than if there is none.

The effect of the fits upon the mental condition of the patient is, as a rule, directly in proportion to the duration of the illness and the frequency of the attacks.

A cure is estimated to take place in from 10 to 12 per cent. of the cases; and Aldren Turner has found the results of treatment by bromides to be—arrest for two and a half to twenty-two years in 23·5 per cent., lessened severity of the fits in 28 per cent., and no influence at all in 47·8 per cent. Although no reliable statistics are yet available, the number of cases successfully treated will probably show an increase since the introduction of luminal.

**Treatment.**—The object of treatment is to reduce the frequency or prevent the recurrence of the fits. The management of a patient during a fit has also to be considered

*In the Interval.*—Something may be done in many cases by careful attention to physical and mental hygiene. Peripheral sources of irritation, such as bad teeth and errors of refraction, should be treated. Food should be light and digestible, with a minimum of meat; large meals should be avoided, and especially heavy suppers just before going to bed. Stimulants, including tea and coffee, should be prohibited in excess. Regular, but not exhausting, exercise is to be recommended, and regular hours of sleep. As regards occupation, something should be found, if possible, which does not make too great a demand on the patient's capabilities, and which does not involve danger to life if a fit occurs.

The epileptic personality is frequently lacking in balance and stability, and the patient may have difficulty in adapting himself to situations of ordinary difficulty in his environment. Epileptic children should, if possible, be taught at home rather than forced into the discipline and competitive stress of school life, and need especial assistance in dealing with the emotional difficulties of puberty and adolescence.

In chronic cases treatment in so-called epileptic colonies where the habits, diet, exercise and recreation are systematically ordered, has been found to present many advantages.

Of medicines the most useful is bromide of potassium. The bromides of sodium and ammonium have also been employed on the ground that the potassium salt had a depressing effect, but the results of recent pharmacological researches are opposed to this view. Potassium bromide has a decided influence upon the fits in a majority of cases. The average dose for an adult is 15 grains three times a day, but this may be increased to 2 drachms in the day to obtain the necessary effect. In some cases it may be possible to time the dosage so as to obtain the maximum sedative effect when most needed: a woman, for instance whose fits occur only with her menstrual periods, may be given a small daily dose during the rest of the month, with instructions to take double or treble the amount at the time when the attacks may be expected; or in cases of nocturnal epilepsy large doses taken at bed-time may take the place of smaller quantities prescribed three times daily. In any case the remedy must be continued for several months or years. Its effect is generally to diminish the frequency or the severity of the fits, so that they occur at intervals of months, instead of every week; if the bromide is left off, the fits again become more frequent, and consequently the patient is glad to continue the use of the drug as a part of the daily routine. Even if the fits cease entirely for several months or a year, the bromide should be continued for two years after the last fit, and then in gradually diminishing doses for another year.

Very large doses of bromide cause dulness and lethargy, with muscular weakness and cold extremities. This condition is known as *bromism*, and is liable to be induced by doses larger than ½ drachm three times a day. If it occurs the drug must be diminished in quantity, or stopped altogether for a time. The bromide is best taken in plenty of water, and the eruption of acne which sometimes occurs from its use may be prevented by adding 2 or 3 minims of liquor arsenicalis, but if this is prescribed signs of cumulative poisoning from the arsenic must be watched for.

If bromide fails other remedies may be tried. Of these the best is luminal which may be given in doses of from 2 to 5 grains in the day to an adult, either in tablet form or as luminal sodium in a mixture. In the latter form it may usefully be combined with bromide. In some cases which are uninfluenced by bromide luminal has a remarkable effect. There are some persons, however, who readily develop toxic symptoms from the drug, such as drowsiness, giddiness and erythematous rashes. Another drug which occasionally proves successful when others fail is the tincture of belladonna in doses of 10 to 15 minims twice or three times a day if it is tolerated. Sodium biborate in doses of 10 to 15 grains two or three times a day has also been recommended by experienced observers and has no harmful effects. Whatever the drug used, the need of continuity in treatment must be impressed upon the patient from the first.

*During the Attack.*—In cases where there is a definite aura the attack can sometimes be arrested. If the aura consists of a sensation in the hand, which gradually proceeds up the arm, the fit may possibly be checked by vigorously rubbing the part, or by tightly constricting the arm above the seat of the sensation, thus preventing, as it were, its progress to the centres. Where this is successful the patient may wear a cord looped round the upper arm, with one end conducted down his sleeve to the wrist, so that by pulling upon this end he can at once constrict the arm. Other patients may be able to ward off fits by turning the attention forcibly to some mental or physical effort as soon as the aura is perceived, and attempts of this kind should be diligently encouraged. Occasionally the inhalation of nitrite of amyl will prevent the further development of an attack. When the fit has really begun little can be done in the way of treatment, but the patient can be protected from some of the results of the convulsions. As a rule he must lie where he falls, unless this is in itself a position of danger (a pool of water or the fire), but he can be prevented from injuring himself against surrounding objects; his collar, necktie, cuffs, and

other tight bands should be at once loosened; and a piece of cork, gutta-percha or firewood should be held between the teeth to prevent the tongue being bitten. False teeth worn by an epileptic should always be removed at night, as they may be loosened in a fit and become impacted in the pharynx.

For the *status epilepticus* the dose of the bromides should be doubled. Frequently, however, rectal medication alone is possible. One drachm of chloral hydrate and 2 drachms of potassium bromide should be given in this way as a single dose. Lumbar puncture and withdrawal of 10 to 15 c.c. of spinal fluid appears sometimes to have a good effect. Failing these remedies, chloroform inhalations must be resorted to.

## INFANTILE CONVULSIONS

Convulsions occur with much greater readiness in infancy than in later periods of life, and under somewhat different circumstances. The higher centres are less developed and exert less controlling power over the lower. The circumstances under which convulsions generally occur are the following: (1) The onset of acute diseases, such as scarlatina, measles, and pneumonia; the convulsions here seem to take the place of the rigor of adults. (2) Local diseases of the brain, of which acute meningitis and encephalitis are the most frequent; but tuberculous tumours, chronic hydrocephalus, and lesions following otitis are occasional causes. (3) Great exhaustion, as after prolonged diarrhœa, or diarrhœa and vomiting; the resemblance of this condition to acute meningitis has been already mentioned (*see* p. 132). (4) Venous congestion of the brain, such as may be caused by an attack of whooping-cough, which sometimes terminates in general convulsions. A child has been known to cry itself into convulsions at once if its mother left it, holding its breath, and becoming more and more livid, until the fit began. Convulsions which not infrequently occur at the end of pneumonia may sometimes belong to this group. (5) Rickets is now held to be responsible for the majority of cases of infantile convulsions not included in the above groups. Often the fit is induced by some peripheral irritation, such as indigestible food; intestinal worms, especially *Ascaris lumbricoides;* cutaneous irritation, such as pins in the clothing, instanced by Trousseau; rarely, perhaps, the process of dentition. (6) Some infantile convulsions must be regarded as really epileptic, since epilepsy may begin in infancy; especially those must be so regarded which commence in early childhood *e.g.* at two or three years, when the influence of rickets is beginning to wane.

Of these six groups it is especially the last two that are usually considered as infantile convulsions proper, the convulsions in the other cases being more definitely symptomatic.

Convulsions in children may closely resemble the epileptic fit of adults, but in a large number of instances they are less complete. They often begin with a short tonic stage: the eyes are turned to one or other side, the pupils are dilated, the head is drawn back, and the arms and legs are rigidly extended. The face may be at first pale, but the lips soon become livid. Twitching then begins in the lips or eyelids, and extends to the whole body, which may be thrown into violent clonic convulsion. The fit lasts a few minutes, and is followed by recovery, or there is a succession of fits, alternating with coma, during which slight twitching of the facial muscles or extremities may take place. Often the convulsion is very much slighter, and consists of little more than deviation of the eyes, or squinting, or fixation of the chest with commencing lividity of the lips, or the convulsive closure of the glottis, known as *laryngismus stridulus* (*see* Diseases of the Larynx), or the hands are extended and rigid, with the thumbs turned into the palms, or the hands and feet are disposed in the manner characteristic of tetany (*see* p. 584). Convulsion may be followed by temporary

hemiplegia in children, as in adults, and strabismus is an occasional result. The convulsions which mark the onset of encephalitis may persist into childhood or adult life, accompanying the paralytic or mental defects of which also such a lesion is the cause (*see* Encephalitis, Infantile Diplegia). Finally, convulsions in children are not infrequently fatal.

**Diagnosis.**—The recognition of infantile convulsions is not itself difficult. It is necessary, however, to determine upon what they depend. If it is a first fit the possibility that it is the onset of an *exanthem* or *pneumonia* must be remembered: the temperature and respirations should be watched, the chest frequently examined, and eruptions should be looked for. Fits due to *cerebral disease* are more likely to be unilateral, and may be accompanied with other symptoms, such as headache, vomiting, retracted abdomen, or optic neuritis. In other cases the indication of rickets must be sought for in the beaded ribs, the enlarged epiphyses, open fontanelle, and delayed dentition; and careful inquiry should be made after some source of irritation, such as unsuitable food, and others above mentioned.

**Treatment.**—This, as in epilepsy, consists of the treatment of the fits, and the means to be taken to prevent recurrence.

When a fit occurs it is usual to place the child at once in a warm bath. If the bowels have not been recently opened, or if there is reason to suppose the ingesta are causing irritation, a grain of calomel may be placed on the tongue. If the fits are very violent and continuous, chloroform may be cautiously administered. It will promptly check the convulsions; but they will probably return soon after it is withdrawn, when it may be again given for a few minutes. When the child recovers sufficiently, 5 grains of bromide of potassium may be given, or if the fits are continuous, it may be given in somewhat larger doses (7 or 10 grains) by the rectum. Chloral may be combined with it to the extent of 3 to 5 grains.

To prevent the recurrence of the fits, one must deal with the predisposing condition, and with any special susceptibility to convulsions. If an exanthem, meningitis, encephalitis, or whooping-cough is the cause of the fit, the disease must be dealt with as advised elsewhere; the fits accompanying such illness are very little amenable to special treatment. The frequency of convulsions in rachitic children can be much influenced by treatment suitable to this disease, such as regulation of the food, administration of cod liver oil, and general hygienic improvement, together with the use of potassium bromide in doses of 2 or 3 grains three times daily. Similarly the cases that are more allied to epilepsy should be treated by the regular use of the bromides

## MIGRAINE

(*Megrim, Sick Headache, Hemicrania*)

This complaint consists of recurring attacks of headache, sometimes preceded by certain abnormal sensations, and often associated with nausea and sickness.

**Ætiology.**—The prime cause appears to be a constitutional defect in the function of the nervous system. In a large proportion of cases there is a family history of the disease. The headaches commonly begin about the age of puberty, but inquiry will often elicit a previous history of "bilious attacks" in childhood, and it appears that migraine and the so-called cyclical vomiting of children are closely related. The complaint more rarely develops in later years. Having once appeared, it usually persists throughout adult life, with a tendency for the attacks to diminish in frequency or disappear after the age of fifty. It is rather more frequent in women than in men, and the attacks are sometimes closely related to menstruation. As occasional causes of the attacks many different agencies have been held responsible. Mental exhaustion, especially that arising from anxiety, appears the commonest, and this probably explains the relative frequency of

migraine among intellectual workers and persons of an excitable disposition. In a certain number of cases eye-strain, especially that resulting from uncorrected errors of refraction or of muscle balance, is an important factor. Constipation or dietetic indiscretions may initiate paroxysms. But, as in the case of epilepsy, we must confess that the majority of the attacks occur without apparent cause.

**Pathology.**—No anatomical changes have been found to account for the disease, either in the brain or elsewhere. The nature of the attacks, especially the association of headache, vomiting, slowing of the pulse rate and visual disturbance, suggests that they are caused by a temporary increase of intracranial pressure, and this, in the absence of any gross cause, must be due either (1) to a mechanical obstruction to the outflow of cerebrospinal fluid from the ventricles

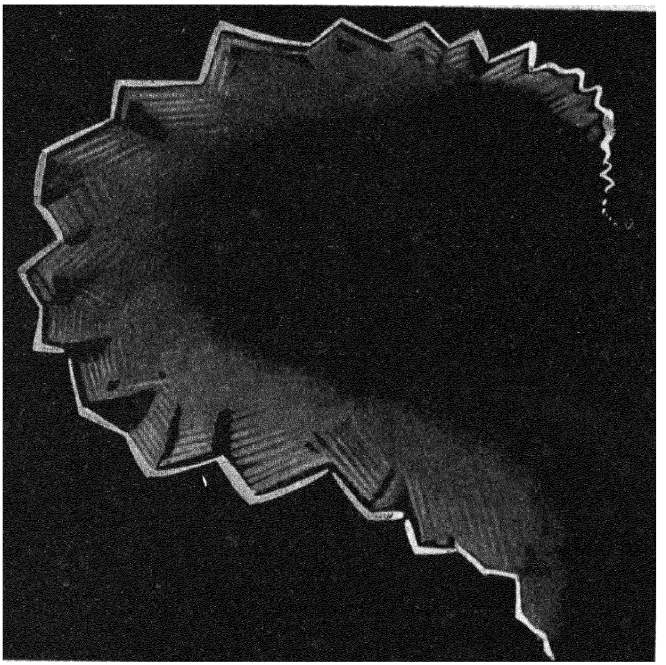

FIG. 92.—The spectrum seen in some cases of migraine, and known as *teichopsia*, or fortification spectrum.

producing for the time being an internal hydrocephalus, or (2) to a transient paralysis of vasomotor control, with consequent capillary engorgement and œdema of the brain.

**Symptoms.**—A complete attack of migraine consists of (1) an aura; (2) headache; (3) visceral disturbance in the shape of nausea, vomiting or sometimes diarrhœa. Incomplete attacks are of common occurrence; any one symptom or pair of symptoms *may* occur without the others, the association of headache and nausea being the most frequent.

The aura when present frequently takes the form of a visual disturbance.

The patient may be apparently in perfect health when he notices that he is unable to see what is directly in the centre of the field of vision, but has to shift his head a little for the purpose, or he sees at once that the centre is occupied by a bright spot. In a few minutes the area of blindness enlarges, and if he turns towards a dark part of the room, or to a clear surface, like the ceiling, he will see a small circle of sparkling colours, having a zigzag or vandyked pattern. From

minute to minute the circle grows larger and larger to one or other side and away from the centre, opening out in form of a horseshoe, which obscures the field of vision as it spreads outwards while vision returns in the centre and progressively improves. The outer margin of the horseshoe consists of a zigzag line of colours; within this is the blind area, full of movement as of a boiling fluid; within this, again, the recovered area of vision. Generally in half an hour from the beginning the horseshoe has reached the periphery of the field; every object is now visible, though, perhaps, with a sensation of oscillation or quivering. It is obviously a subjective sensation, due to a disturbance of the brain which must be situate behind the optic chiasma on the side opposite to the blind part of the field. This curious spectrum has been described as *teichopsia*, or fortification spectrum (*see* Fig. 92). Owing to its remarkable character, the fortification spectrum has attracted much attention. In its complete form, however, it is not common. More usually the sensation is limited to vague flashes of light, followed by darkness in the half-field, or a simple onset of hemianopic dimness. The part of the visual field varies from one attack to the other, being sometimes the right halves, sometimes the left, sometimes both outer halves, or the upper or lower parts of both fields may be affected (transverse hemianopia). In some instances the hemianopic character is absent, and there is a general dimness of vision. The visual aura is sometimes followed or replaced by sensory or motor phenomena with the same distribution as that of a hemiplegia. These usually begin with a sense of tingling in the tongue and lips of both sides, which then appears in the face and hand, and gradually spreads over the whole side of the body, the part first attacked recovering as the others are invaded. The whole process occupies twenty to thirty minutes. The tingling is occasionally accompanied by motor weakness, and if the right side be affected there is transient disturbance of speech also.

In some cases the only disturbance preceding the headache is of a *mental* kind; there is depression, languor, or fear of impending evil, or there may be an unusual sense of well-being; and these feelings may last from half an hour to two or three hours.

Following the aura as a rule immediately, comes the headache. This is of all degrees of intensity, so slight as not to interfere with mental work, or so severe as entirely to unfit the sufferer for any action whatever. It often lasts a whole day, sometimes two or three, and in the severer forms it increases gradually, until at its height nausea and vomiting occur, and a certain amount of relief follows. After this it quickly disappears, or it subsides more gradually, or it continues till the patient seeks his bed at night; and he awakes, perhaps after a prolonged sleep, cured, but with some sense of weakness or fatigue. The pain is often unilateral (*hemicrania*), but it may begin on one side and change over to the other, and even return again to the side first affected, or it may affect both sides at once; the side first affected is generally that which is opposite to the visual spectrum. The pain may begin in the frontal, temporal, or parietal region, or behind the eye, often at a very limited spot, whence it may spread in different directions, or become general. It is often boring in character, and aggravated by movement, light, or sound; and the patients are only comfortable in the recumbent posture. The pain may be so bad that the patient passes into a stupor or becomes delirious. With this the face is pale and drawn, the hands and feet cold, the pulse feeble, small, and slow. Only in some cases it appears that as the headache continues these conditions are reversed, the face flushes, and sweating occurs.

The attacks of migraine recur at intervals of a few days or two or three months, an interval of three or four weeks being more common. Particular attacks may be determined by the special causes enumerated, but sometimes it is impossible to find out what has induced the disturbance. Apart from certain clinical features of resemblance, there is an association between migraine and epilepsy. They are sometimes found occurring in the same patient. In other instances a patient

suffering from migraine may later develop epilepsy and thenceforward remain free from the migrainous attacks, or the exchange may be in the other direction.

*Ophthalmoplegic migraine* is the name which has been given to a group of rare cases in which recurrent attacks of headache are associated with temporary paralysis of one of the oculomotor nerves. As a rule the paralysis clears up between attacks, but may gradually become permanent. No satisfactory explanation of these cases is forthcoming. The fact, however, remains that attacks of this nature may continue for twenty to thirty years without the development of any signs of serious organic disease.

**Diagnosis.**—The periodicity of the attacks with intervals of perfect freedom, the age of onset, and the association with nausea are as a rule sufficient for a diagnosis. But in every case other causes must be excluded. The recurrent headaches of cerebral tumour are sooner or later associated with papilloedema or signs of local damage to the brain. Other causes of paroxysmal headache that must be considered are those due to infection of the nasal sinuses, syphilitic meningitis, cerebral arteriosclerosis, and toxic causes such as uraemia.

The aura may be confused with that of epilepsy. But in the latter disease the aura is of momentary duration, while in migraine it occupies about twenty minutes. Nor in migraine is there any disturbance of consciousness. Some patients may be troubled more by the nausea or vomiting than by the headache, and seek relief for these symptoms only.

**Prognosis.**—Under treatment much improvement may be obtained, both in the frequency and in the severity of the attacks, though the disease commonly continues for years. It is, however, not dangerous to life, and there is no evidence that sufferers from migraine are more liable than others to hæmorrhage, thrombosis, and other diseases of the brain. The disease often becomes less frequent or disappears altogether after the age of fifty, especially in women after the climacteric.

**Treatment.**—*In the interval.*—The patient should be placed under the most favourable hygienic conditions, including a carefully regulated diet, the avoidance of constipation, exercise without exhaustion, pure and bracing air, and exemption from excessive brain study or mental worry. Hurst maintains that many sufferers may be cured of their attacks by the correction with glasses of errors of ocular refraction or muscle balance.

Of drugs the most valuable is luminal. This may be given in tablet form or in a mixture as luminal sodium. Half a gram thrice daily is usually adequate and insufficient to cause drowsiness. It must be taken regularly for many months, and as a rule diminishes both the frequency and severity of the attacks. In some cases Gowers' mixture containing liq. trinitrin ℥i, liq. strychninae ℥v, tr. gelsemii ℥x, sodium bromide gr. x, dilute hydrochloric acid ℥x, taken regularly as a preventive, has a good effect.

*During the attack.* The sufferer usually learns from experience that the less severe attacks can be cut short. Ten or fifteen grains of aspirin and an hour's rest on a sofa in a quiet and darkened room often suffice, especially if during this period sleep can be procured. If the symptoms appear at the end of an exhausting day a good meal and a glass of wine will sometimes clear the air, provided the nausea has not set in. In the more severe attacks there is total cessation of gastric peristalsis (as shown by X-rays), and nothing can be taken by mouth except sips of water. Rest in a darkened room is then the only means of relief. If vomiting is severe and prolonged, rectal infusions of 6 per cent. glucose should be given; they help to cut short the attack and lessen the subsequent exhaustion. Cold applications to the head are usually welcomed. If the pain is unusually severe, morphia should be given hypodermically, and the dose may need to be repeated. If this should be necessary, it must be administered either by the medical attendant or by a trained nurse. The morphia should never be entrusted to the patient or her friends.

## MYOCLONUS

(*Myoclonia*)

Under this title are grouped a large number of conditions, of which the essential feature is short, quick contraction of muscles, not forming part of epilepsy, hysteria, chorea, athetosis, etc. Myoclonic movements occurring in encephalitis lethargica have already been referred to (*see* p. 634).

### PARAMYOCLONUS MULTIPLEX

Friedreich described under this name the case of a man who had sudden lightning-like contractions of the large muscles of the arms, forearms, and thighs. The contractions ceased when he walked, and were worse when he was quiet in bed; if one arm was used the contractions ceased in it, but continued in the other. The contractions, though violent, produced no movement of the limb as a whole. The knee jerks were increased; the skin sensibility, muscle sense, and vasomotor and secretory phenomena were normal. The condition disappeared under treatment with galvanism, but it relapsed and persisted till the man's death. Numerous other cases have been recorded of clonic muscular contractions which differ in many points from Friedreich's case. The essential seems to be the occurrence of spontaneous rapid contractions of isolated muscles or parts of muscles in different parts of the body, in some cases the limbs, in others the face and trunk, generally bilateral, but not of necessity equally, or at the same time, on the two sides; sometimes, but not generally, causing locomotive effect in the parts involved; occurring at the rate of 60 to 100 in the minute, but quite irregularly; varying in frequency and force on different days; and often increased when the patient is under observation, or exposed to external stimuli (sound, touch). The mechanical excitability of the muscles is increased; their electrical excitability is unchanged. Some cases have been associated with epilepsy (*myoclonus epilepsy*), others have shown close resemblances to hysteria, and it has occurred in two or more members of the same family; rarely these movements have been associated with definite disease of the central nervous system. The pathology is unknown. Galvanism to the spine, hydropathic treatment, arsenic and chloral have been used to combat it, and recoveries have occurred; but the prognosis is not good.

## THE TICS

(*Habit Spasms*)

A tic has been defined as a co-ordinated, systematised, purposive act, reproducing in an involuntary manner the co-ordinated movements of every-day life (Meige).

The movements usually commence in childhood, between five and twelve years of age, affecting especially the facial muscles, and being frequently repeated at irregular intervals. Such movements are blinking the eyes, twitching of the angle of the nose or mouth, shrugging the shoulders, twitching the fingers, uttering noises or words, kicking out the legs, or other simple movements. They have probably been voluntary, in the first instance, for the purpose of relieving an irritation, or in response to some local sensation, and then have been repeated reflexly, or stimulated by a thought, and finally have become automatic—a bad habit, of which indeed the child may be almost unconscious. The movements may become less under observation, and in the same child one kind of movement may be cured, and after a time another will take its place. It is a question whether certain visceral disorders should not be considered as belonging to the tics, although they are more usually referred to as symptoms of hysteria, and will therefore be described under that heading. Such are certain types of functional

vomiting and aerophagy—a condition in which the individual in attempts at retching is continually taking down gulps of air into his stomach.

Movements of a reflex nature arising, in the first place, from peripheral irritation are not perpetuated as tics in persons whose mental adjustment is normal. Thus in children the disorder is frequently associated with bed-wetting, night terrors, and other signs of a neurotic constitution, and the habit probably always originates at a psychical level as an expression of faulty adjustment of the personality as a whole. In favour of this also is the fact, ascertainable from the patient in most cases, that the movement is preceded by an imperative desire to perform it, and that if this is inhibited great mental distress is occasioned.

The distinction usually has to be made from chorea, in which the movements are, as a rule, more widespread, and are purposeless and irregular.

In the causation two sets of factors have to be considered: on the one hand, neglected sources of peripheral irritation, such as a chronic conjunctivitis or a frayed collar, and difficulties of an emotional nature, such as unhappy domestic surroundings or imperfect adaptation to school life; these are the environmental factors, and, in the phraseology of the old parable, may be called the seed. On the other hand is the neurotic personality which furnishes the soil, and it is upon this latter that the prognosis largely depends. In some cases removal of all sources of irritation, a complete change of environment, and assistance in the solution of personal difficulties may effect a cure, especially in children. In adults and in cases of long standing the disease is likely to be permanent, taking the form in some cases of a mere eccentricity of behaviour. It may be a symptom of deeply rooted mental instability leading to further manifestations, such as those described below under General Convulsive Tic. In the treatment bromides are useful for their sedative effect, together with exercises in which the patient is taught to relax the affected muscles, and to use them for other movements. These exercises may be practised daily in front of a looking-glass, and should be continued as a routine for some time after the abnormal movements have ceased. In addition, attention should be paid to the underlying psychological factors, as already indicated.

### GENERAL CONVULSIVE TIC

A form of clonic muscular contraction, which is probably only a more severe degree of the above, has been described as *maladie des tics convulsifs* by Guinon and Gilles de la Tourette. It is characterised by contractions of the facial muscles, by systematic movements of different parts of the body which always repeat themselves in the same way, by the utterance of strange noises, by repetition of words heard (*echolalia*), by frequent utterance of obscene words and expressions (*coprolalia*), and by imitation of other movements (*echokinesis*). It often begins in childhood; the facial clonus occurs first, and the extension to other parts of the body, and to other kinds of movement, takes place from time to time in the course of subsequent years. It is a very obstinate disease, but improvement has been obtained by the help of bromides, chloral, hydropathic treatment, isolation, and gymnastics, and psychotherapy.

### SPASMODIC TORTICOLLIS

Besides the temporary affection known as stiff neck or rheumatic torticollis (*see* p. 910), there are two more lasting conditions known by the name of torticollis: *fixed torticollis*, or congenital wryneck, and *spasmodic torticollis*. The former is due to a permanent shortening of the sterno-mastoid muscle, which is attributed in some cases to injuries during birth, is observed first during childhood, if not in early infancy, and causes asymmetry in the bones of the face.

*Spasmodic torticollis*, or spasmodic wryneck, is a disorder of function characterised by tonic and clonic contractions of the muscles of the neck, whereby

the head is forced into an abnormal position, and it is in many ways allied to the tics.

**Ætiology.**—The disease is rarely seen before the age of thirty; it affects both sexes, but females more often than men. The cause cannot always be ascertained; neurotic inheritance, inflammation of the cervical glands or of the muscles themselves, exposure to cold, falls and injuries, and overstrain of the muscles of the shoulder, arm, or neck in particular occupations, have been recorded as antecedents. Cases have been reported in which the symptom has occurred as a sequel of encephalitis lethargica.

**Pathology.**—Of this little is known. No pathological lesion has been found to account for it, but it presumably depends upon disturbed function of motor centres in the brain or brain stem.

**Symptoms.**—It begins gradually, being first felt as a mere discomfort in the neck; then distinct jerking movements of the affected muscles are felt, by which the head is rotated or displaced. If, for instance, the right sterno-mastoid is affected, the head is constantly being jerked in the direction of the action of this muscle, the chin is thrust forwards or upwards to the opposite side, and the occiput is drawn down towards the clavicle. The contractions are sudden, irregular, and frequent; for a few moments there is a remission, during which the patient slowly and cautiously tries to bring the head straight, when the muscle again contracts, and the face is gradually forced round to the left. The movements are for a time checked by supporting the head, and they cease during sleep, but immediately the patient awakes the movements recommence, and continue with but little rest throughout the day; they are generally increased when attention is directed to them. The muscle most frequently affected is the sterno-mastoid, and next to that the upper part of the trapezius and the splenius capitis; the complexus and trachelo-mastoid, the deep rotators of the head, and the platysma myoides are also sometimes concerned. The position of the head is of course determined by the muscles which contract. The sterno-mastoid produces the results already described; the trapezius draws the head backwards and downwards towards its own side, with slight rotation of the face towards the opposite side, while the splenius draws the head downwards with slight rotation towards its own side. Two or more of these muscles may be affected at the same time, most often the sterno-mastoid with the upper part of the trapezius of the same side, or the splenius of the opposite side. An intermediate position of the head will of course be the result. Or corresponding muscles on the two sides may act together, drawing the head backwards during their contraction. In such cases there is generally an associated contraction of the frontal muscles which normally contract when one throws back the head to look upwards. Occasionally the disease itself spreads to the muscles of the face, or to those of the shoulder or arm, especially at the height of the paroxysm; and in rare cases the lumbar or spinal muscles may be affected so that the body is drawn down to one side by the frequent contractions. In slighter cases, or in early stages, there may be no pain, but in severer forms there is neuralgic pain in the contracting muscles. From their excessive action the muscles often hypertrophy; they at least retain their natural bulk. The electrical irritability is normal or increased.

**Diagnosis.**—The constant movements distinguish this disease from congenital wryneck, with its early history and facial asymmetry; from the temporary disorder "stiff neck"; and from spasm of the muscles in caries of the cervical vertebræ.

The **Prognosis** is unfavourable; the spasms may subside after a few months, but far more often persist in a more or less severe form for the rest of life.

**Treatment.**—The disease is most intractable, and numbers of drugs have been tried with but a small amount of success. Those which have been most

useful are the bromides, asafœtida, zinc valerianate, belladonna, and hyoscine hydrobromide by subcutaneous injection. Opiates and chloroform inhalation check the spasm for a time, but it returns in a few hours unless the dose is repeated In some cases exercises are of value as described in the treatment of tics, and especially education in the relaxation of the affected muscles.

In severe or obstinate cases the nerves or the muscles may be divided. If the former are selected, the deep cervical nerves must be resected as well as the spinal accessory. The movements will cease, but some incapacity must result from the accompanying paralysis and atrophy; and the disease may reappear in adjacent muscles. Kocher, of Berne, claims success for his method of dividing in two, three or four operations the sterno-mastoid, trapezius, splenius capitis, complexus, trachelo-mastoid, and even the obliquus inferior. Relief has been obtained by the use of a light steel spring clamp to the back and sides of the neck (Hall).

The symptoms are invariably worse under the influence of mental fatigue and anxiety, and may therefore be improved by means of psychotherapy.

## WRITERS' CRAMP AND ALLIED NEUROSES

Those persons whose occupations necessitate complicated movements for long periods of time, such as clerks, pianists, violinists, telegraph operators, cigar-makers, and others, may be subject, when engaged at work, to spasmodic and irregular contraction of the muscles concerned, so that the movement is badly performed and ultimately cannot be effected at all. A large number of those who thus suffer have previously had some organic or functional nervous disorder, or may be referred to the class of neuropathies by heredity, or of neurasthenics. The exciting cause is some depressing mental condition, mental anxiety, or business worry; an injury, or local disease of the hand or fingers; but more than all an excessive use of the hand in the occupation concerned.

The disease is most common in those who have a great deal of writing as their daily occupation, such as lawyers' clerks, secretaries, etc. It is hence called *writers' cramp* and *scriveners' palsy; graphospasm* and *mogigraphia* have been used as technical terms. This form is naturally more frequent in men than in women, and occurs mostly between the ages of twenty and forty. Gowers pointed out that in the act of writing the pen may be moved across the paper in four different ways: (1) The little finger is fixed on the paper, and the fingers carrying the pen work upon the little finger as a pivot; (2) the wrist is fixed and acts as the pivot; (3) the pivot is at the centre of the forearm, resting perhaps on the edge of the table or desk; (4) all the movements take place from the shoulder. In the first method the movements of the fingers are most complicated and strained, and in the last there may be no finger movements at all. He stated that writers' cramp scarcely ever affects those who employ the last two methods of writing.

**Pathology.**—This affection has been thought to arise from the weakness of certain muscles and the over-action of antagonist muscles, or from weakness of one muscle being supplemented by another muscle, which in its turn gets fatigued and is followed by another, until all are worn out or as the result of reflex action, stimulated through the sensory nerves. A probable explanation is that it is due to a defect in the centres associated for the act of writing by a morbid lowering of resistance in the commissural connections between the centres, so that there is a radiation of impulses, and so over-action of muscles not necessarily engaged in the act.

**Symptoms.**—The affection generally comes on gradually; it may be felt at first as some degree of aching or strain, which is relieved by ceasing to write. After a time the act of writing is accompanied by a spasmodic tonic contraction of the finger or thumb holding the pen; the finger is pressed firmly on the pen, or it is flexed so as to move up the pen, or it slips off the pen so that the latter is

grasped between the fore and middle fingers. The thumb may be similarly affected, or the fingers may be extended or lifted from the paper, or the pen may be driven into the paper, or the hand stops its movements entirely. The attempt to continue writing under these conditions produces a cramped, irregular, angular writing, with thick down-strokes; and after a time the spasm becomes so pronounced as to render the act impossible. This is the *spasmodic* or *spastic* form of Benedikt, which is by far the most common; but sometimes there is tremor of the fingers—*tremulous* form; and a *paralytic* form with fatigue alone has been described, but is quite rare. The spastic form often leads, by the frequent contraction of the muscles, to pains in the hands and wrist, which may after a time become distinctly neuralgic in character; and there is often some tingling or sense of numbness. The spasm may be limited entirely to the act of writing, and other movements, even of a delicate nature, can be performed without difficulty. Sometimes writers' cramp is associated in the same person with spasm on playing the piano or violin, and not infrequently in severe cases some other operation may be at the same time imperfectly performed.

The muscular power is for the most part preserved, or there may be a little weakness of grasp, or slight but definite weakness of certain muscles of the hand. The electrical reactions may be quite normal, or they show a slight increase or diminution of irritability in some old cases.

The course of the disease is variable. In slight cases treated at once by perfect rest from writing the patient may recover completely; but if he has persevered, forcing himself to write by steadying his hand with the other, or by mechanical contrivances, and has ignored all treatment, the disease is often quite obstinate, and may never be thoroughly cured.

The **Diagnosis** is not generally difficult; writers' cramp at least is not likely to be mistaken for anything else, but it must be remembered that some nervous diseases, such as chorea, hemiplegia, and other paralyses involving power in the hand, may be first detected in the attempt to write, and may be regarded wrongly as writers' cramp. Nervous people, too, who have obtained some acquaintance with the disease, may easily fancy that a little fatigue is the commencement of it.

**Treatment.**—The first essential is complete rest from writing. In mild cases this is sometimes sufficient to effect a cure in one or two months. Gowers insisted that, on again beginning to write, the patient should learn to write from the shoulder entirely. In more severe cases a much longer rest is required, and if writing is necessary to the patient, he may learn to write with the left hand or use a typewriter. Occasionally, but by no means always, the newly educated left hand also becomes affected. Various devices have been invented, or are improvised by the patients themselves, to save the strain on the muscles of the fingers, such as running the pen through a cork, which gives a larger grasp; or holding a wooden ball in the hand, upon which the pen is fixed at the required angle. Nussbaum's "bracelet" carries the pen, and surrounds the fingers, so that they hold it by muscles (abductors) different from those commonly employed in writing. But, as a rule, these instruments only postpone the time at which complete rest must be taken. A return to the normal action of nerve and muscle may be sought in the use of general tonics, such as iron, quinine, arsenic, and strychnia, and in local treatment, gymnastic exercises, passive manipulations, and massage.

The treatment of the other occupation neuroses must be the same in principle as that already described for writers' cramp.

## NEURALGIA

This term is sometimes used for pain, felt in the course of a particular nerve and its branches, for which no organic cause can be found. It therefore describes

a symptom rather than a disease. Pain of such distribution may be due (1) to disease of some part of the body supplied by the nerve in question, the pain being, as it were, reflected throughout the whole distribution of the nerve; (2) to disease affecting the nerve fibres either in the peripheral nerves, the posterior nerve roots, or occasionally the grey matter of the spinal cord; (3) to disease of some internal organ whose sensory nerve supply comes from that segment of the spinal cord which corresponds with the superficial distribution of the pain.

As examples of the first cause may be mentioned pain of trigeminal distribution caused by an inflamed tooth; of the second, pain of intercostal distribution due to pressure upon a posterior nerve root from tuberculous caries of the spine; and of the third, the pain along the ulnar border of the left arm experienced in angina.

A common cause of neuralgic pains is a local neuritis the result of trauma, intoxication or infection. In these cases there may be no positive indication, such as muscular wasting or numbness, of actual disease of the nerve concerned. There is, however, usually tenderness to pressure over points where the nerve lies against bone, and the occurrence in some cases of definite signs, such as the loss of ankle jerk in sciatica, makes it highly probable that the others are also due to a localised neuritis. They have therefore been described under that heading (*see* p. 738). There is, however, one form of neuralgia which, on account of its distinctive features, merits separate description.

## TRIGEMINAL NEURALGIA

### (*Trifacial Neuralgia, Tic Douloureux*)

In this affection there are recurrent attacks of paroxysmal pain, referred to the distribution of one or more branches of the fifth nerve. The attacks begin most frequently in the area supplied by the second division, less often in that of the third; the first, or ophthalmic, division is rarely involved, and then only when there is neuralgia of the second division also. In a few cases all three divisions of the nerve may be affected at the same time. The disease may occur at any period of adult life, but is most common at about the age of fifty. Women are attacked more frequently than men. The ætiology and pathology of true trigeminal neuralgia are at present obscure. It cannot be traced directly to dental sepsis, and may occur in persons who have been edentulous for years. No constant changes are found in the branches of the fifth nerve or in the Gasserian ganglion. Nevertheless the frequency with which the second and third divisions are primarily involved suggests that the disease may be due to the action of bacterial toxins originating from septic foci in the teeth or air sinuses, and reaching the Gasserian ganglion by way of the perineural lymphatics.

As a rule the initial paroxysm is sudden in its onset, and there may be an interval of weeks or months before the next. The intervals, however, gradually become shorter, and the paroxysms more intense, until the patient's life is made intolerable by attacks occurring every few minutes of the day. The pain is extremely severe, and may completely incapacitate the patient while it lasts; it is described as burning, darting or quivering, or as a sensation of red-hot needles being thrust into the cheek. It is often accompanied by a flow of saliva or tears, and there may be flushing of the cheek. The paroxysm is as a rule of brief duration, lasting only a few minutes. During the attack the skin and mucous membranes of the affected area are extremely sensitive, and a breath of cold air upon the cheek, the contact of food with the buccal mucous membrane, or even the movement of talking may suffice to initiate a further paroxysm. In the quiescent intervals, however, and even in the brief remissions between a succession of paroxysms, there is a complete absence of pain or tenderness.

Apart from the paroxysms of pain there are no signs of impairment of the functions of the fifth nerve. The corneal reflex is normal; cutaneous sensibility

is preserved; there is no weakness or wasting of the muscles of mastication. By these facts true trigeminal neuralgia may be distinguished from cases of gross organic disease (syphilis or tumour) involving the Gasserian ganglion or its branches.

**Treatment.**—Drugs are of little or no avail; tincture of gelsemium in large doses, combined with bromide, may be tried. The patient will usually discover for herself the advantage of protecting the affected side of the face from cold by means of a shawl or other covering, and may ward off paroxysms by employing writing instead of speech, and taking nourishment through a straw inserted into the opposite corner of the mouth. In skilled hands injection of the nerve roots with alcohol has given good results. The second and third divisions may both be dealt with in this fashion, and relief may often be obtained for as much as a year before the injection has to be repeated. In time, however, this method fails to give relief, and some more drastic procedure is necessary. The most satisfactory is division of the sensory root of the fifth nerve between the Gasserian ganglion and the pons; this involves a surgical operation of some magnitude, but is more certain than the alternative, which is to inject the Gasserian ganglion with alcohol through the foramen ovale. The disadvantage of either of these procedures is the risk of trophic changes occurring in the eye with subsequent blindness, which is considerable. Most patients, however, will cheerfully take the risk rather than continue to endure the pain.

## NEURASTHENIA

(*Nervous Exhaustion*)

This is a condition of disordered health resulting from exhaustion of the nervous system and probably also of certain endocrine glands, in which there is a diminished capacity for physical and mental exertion combined with various subjective sensations and vasomotor phenomena, the whole being independent of any organic disease of the nervous system. The disease is relatively uncommon, except as a complication of anxiety neurosis (*q.v.*)

**Ætiology.**—Neurasthenia is a disease of adult life, being most frequently met with between the ages of twenty-five and fifty, and is rather more common among men than women. Heredity plays a part in the causation in so far as a special liability to the illness appears to run in certain families. The main causal factors appear to be prolonged physical or mental fatigue, the latter being chiefly of importance when accompanied by insomnia. To these may be added intoxications of various kinds both chemical and bacterial. As examples may be mentioned repeated pregnancies and prolonged lactation in women; hard physical work in tropical climates; the exhausting effects of certain bacterial infections, especially typhoid fever and influenza; chronic alcoholism; and toxic absorption from sources of focal sepsis. In the absence of any obvious cause of this kind, the disorder is usually due to the mental strain of an underlying anxiety neurosis.

**Pathology.**—The assumption that the symptoms to be described are due to exhaustion of the nervous system is based upon their general resemblance to those observed in the case of healthy individuals who have been subjected to prolonged physical or mental strain, especially if they have gone without a proper allowance of sleep. Similar symptoms may occasionally be observed in persons suffering from chronic intoxications of bacterial or chemical origin. Instances have been described of human beings who have died apparently as the direct result of being without sleep for a period of several days, in which no adequate cause of death has been discovered upon post-mortem examination.

It is clear that the organism as a whole is capable of fatigue, and that the functions of the central nervous system are affected under these conditions. Yet

we know so little of the biological processes associated with nervous activity that we are ignorant of what underlying changes may be responsible for the fatigue of the neuronic systems. It has been shown experimentally that a nerve fibre shows signs of fatigue in the complete absence of oxygen, and that the ability of a nerve to conduct an impulse may be abolished by the action upon it of certain substances, of which alcohol and the so-called anæsthetics are examples. There is some post-mortem evidence from observations made upon human beings who have died as the result of prolonged physical strain and insomnia, and upon animals deprived of sleep for experimental purposes, to show that pathological changes take place in the nerve cells under such conditions; and in certain of these cases also changes have been described in the adrenal glands.

**Symptoms.**—The symptoms in the earlier stages may be considered analogous to those experienced by a healthy person on the morning after an unexpected night's work without sleep, or an outburst of alcoholic over-indulgence. The patient complains of heaviness in the head, difficulty in concentration, irritability and a general feeling of lassitude and inadequacy. If he seeks medical advice and is properly treated at this stage the symptoms as a rule disappear. Not infrequently, however, they develop with the addition of insomnia. This is the most troublesome of all symptoms, and leads to aggravation of the others. The subjective sensations of mental and physical fatigue become more pronounced, and the patient commonly complains of headache, backache, muscular weakness and indigestion.

The headache is usually described in terms of discomfort rather than pain, and varies from a sense of great heaviness to a feeling as if the head were encased in a leaden cap. The patient may also complain of throbbing in the head and buzzing in the ears. The backache is, as a rule, associated with a tired feeling similar to that experienced by a person who has been on his feet all day. The muscular weakness also is accompanied by a sense of great fatigue, so that the patient finds that he cannot undertake any physical effort without becoming exhausted. With this he may complain of aching in the limbs. Loss of appetite and constipation are frequent symptoms, and in his anxiety to remedy these troubles the patient is apt to increase them by the constant use of purgatives and injudicious dieting.

Mentally he becomes depressed about his condition, and tends to worry about his physical symptoms; in some cases this leads to a hypochondriac state in which the attention is constantly turned towards the stomach, heart or bowels, which are blamed as the causes of the illness. In this state also the patient often becomes anxious about his sexual powers, the activity of which is diminished together with that of the other bodily systems. He may then complain of spermatorrhœa, or attribute his trouble to masturbation practised in his boyhood. The extent to which mental symptoms of this nature develop depends upon individual peculiarities. Thus a person of neurotic tendencies is especially likely to develop feelings of inferiority and morbid anxiety under circumstances leading to a state of nervous exhaustion.

In addition to the depression, there is difficulty in concentrating the attention upon any subject for more than a short time, together with a sense of great mental fatigue on attempting any intellectual task. This, in its turn, is apt to lead to restlessness and irritability as the patient seeks distraction in various directions without relief.

On examination no signs of organic disease will be discovered. In some cases the pupils are large, and it may be demonstrated that the ciliary muscles are readily fatigued on accommodation. On lateral deviation the extra-ocular muscles also are easily tired, which leads to nystagmoid jerkings of the eyeballs, but there is no true nystagmus.

Sensory examination reveals no abnormality. As regards the motor system, although there is no definite weakness in the performance of single movements, the capacity for sustained effort is markedly impaired. The reflexes are normal

Signs of disturbances of the sympathetic system are often present in the form of blueness and coldness of the extremities and excessive sweating.

**Diagnosis.**—This can be made only when organic disease has been excluded by careful examination. Of the conditions which in their early stages are liable to be confused with neurasthenia, phthisis, general paralysis of the insane, pernicious anæmia, chronic Bright's disease, and Addison's disease may be mentioned as the commonest. They may, however, be distinguished by means of careful clinical observations and the appropriate tests.

The symptoms of neurasthenia must also be distinguished from those of anxiety neurosis. This distinction is not always easy, since the latter condition, if prolonged, often results in true nervous exhaustion. Careful analysis of the history as a rule will decide the point. In the absence of any apparent cause, neurasthenia is generally found to depend upon an underlying anxiety neurosis.

**Prognosis.**—The prognosis is good in those cases for which it is possible to secure adequate treatment before the trouble has progressed too far. When the condition is of long standing much depends upon the insomnia, which completes the vicious circle of disease by perpetuating fatigue; provided that the normal habit of sleep can be regained, the chance of recovery is good.

In cases where the neurasthenic state has developed upon the basis of submerged emotional conflicts, such as occur in anxiety conditions, the patient may be temporarily benefited by treatment of the nature to be described, but is liable to relapse, and the ultimate prognosis will depend upon that of the underlying neurosis.

Finally, there appears to be a small group of cases developing after a sudden emotional shock, or a prolonged illness such as typhoid fever, in which the capacity of the nervous system to react adequately to mental or physical strain seems to be permanently impaired.

**Prevention.**—The condition of neurasthenia is to be regarded as an exaggerated degree of that experienced by many persons at the end of an arduous week's work, or more especially at the end of a year's labour, when the annual holiday is due. "No man can do hard mental or physical work for more than six days in the week, or for more than eleven months in the year, without breaking down sooner or later. Adequate holidays, real holidays, in which a man gets completely away from his work, are the best preventives of neurasthenia" (Hurst).

**Treatment.** In the first place, any obvious source of septic absorption should be attended to, and all drugs should be stopped. The main feature of the treatment is rest. Whenever possible this should be carried out away from the patient's own home, but if this cannot be managed he should be secluded as far as possible, especially from relatives and friends, who are likely to make demands upon his emotional resources. In the first place, he should be put to bed in a quiet room, and should be kept in bed for one to two weeks. He should, however, be allowed to get up in order to go to the lavatory and bath, for this at the same time is less irksome and prevents him from losing the use of his legs to the extent occasioned by unmitigated rest. During this period he may be allowed to read light literature, but should receive no letters nor visitors. This rule, however, should not be made so rigid as to deprive the patient of news upon matters which may be the subject of anxiety. No special restrictions should be enjoined as to diet, which should be that to which the patient is accustomed in normal health. Over-feeding with milk is likely to do more harm than good, unless there is wasting from refusal to take food (see p. 414). Alcohol may be allowed in small quantities if the patient has been accustomed to take it, and the same rule applies to tobacco, the quantity, however, being strictly limited. If necessary, the bowels should be opened daily with enemas rather than purgatives. General massage of the muscles should be performed once a day while the patient is in bed, and after the first few days physical exercises may be com-

menced, at first of a very mild nature, but gradually increasing in severity. Such exercises can easily be carried out in bed, and the patient is thus encouraged at an early stage of the treatment to become an interested observer of his own progress towards health.

Meanwhile every attempt should be made to combat the insomnia. In a mild case quiet and change of surroundings may suffice. A hot bath just before settling down for the night, followed by exercises in muscular relaxation, is often useful. If such measures are not effective, there should be no hesitation in having recourse to hypnotics in order to promote the habit of normal sleep. Sufficient doses should be employed in the beginning to ensure six or seven hours' sleep. Medinal, paraldehyde, or a combination of chloral and bromide, are the most useful drugs. When the patient has had a sequence of two or three good nights the dose should gradually be reduced (*e.g.*, an initial dose of medinal, grs. x, may be reduced by gr. i nightly). If the neurasthenic condition is secondary to an anxiety neurosis, psychotherapy should be used in association with these measures (*see* Anxiety Neurosis Treatment).

At the end of the second week the massage should be discontinued, and the patient should begin to get up, at first for half an hour in the morning and again in the evening, the time allowed being increased daily by half an hour. As soon as possible he should commence to take gentle exercise out of doors, the amount being gradually increased daily. It is of advantage in this connection to prescribe a regular plan of activities. At this stage he should be allowed selected visitors and should be encouraged to seek diversion in mild amusements.

When he gets up enemas should be discontinued, and if his bowels are not open regularly senna pods should be given, beginning with a full dose and decreasing by one pod each night. At the same time he should be instructed to go to the closet after breakfast each morning at the same time and to stay there until he gets his bowels open or for twenty minutes.

At the end of a month from the commencement of treatment the patient should be fit to lead an ordinary life, and should then have a fortnight's clear holiday, if possible away from home, before returning to work. During this holiday it may be well to prescribe a rest for one hour each day after the midday meal, during which he should lie down on a couch or bed in a darkened room and endeavour to go to sleep.

For the future he should take good care to follow the rules laid down under Preventive Treatment.

# THE PSYCHONEUROSES

We have now to consider certain illnesses whose symptoms are due to disturbances at the highest level of bodily integration, that is in the mental processes of the individual. These disorders are sometimes called *nervous* because it is the nervous system which is chiefly responsible for the integration of the personality; they are called *functional* to distinguish them from illnesses which are due to structural lesions of the nervous system.

In order to understand the manner in which these disturbances arise it is necessary to know something of the laws governing the mental activities under normal conditions.

## PSYCHOLOGY

Underlying all those processes of feeling, thinking and doing which constitute the mental activities of man are certain driving forces whose general trend is to impel the individual to secure, not only for himself, but for his species, dominance over the environment. Of the source and nature of this store of primitive energy we know little. It is the same which we see in all lower forms of life as the essential feature of their existence. In the behaviour of the

plant or of the insect we are able to observe the workings of these forces in their simplest form; the organism inherits certain modes of response to its environment, relatively few in number and invariable, through which they are expressed. In the mammalia, the development of the cerebrum provides opportunity for a great variety of modes of expression, which renders the study of behaviour more difficult, and in man this difficulty is vastly increased by the existence of conscious memory and association of ideas, and by the development of speech and writing.

We can, however, still discern underlying the mental activities certain primitive trends known as the instincts. These may be considered as innate, reflex modes of response of the individual as a whole to bodily needs or external stimuli.

Psychologists are not in complete agreement as to the proper classification of the instincts, but two at any rate—the instinct of self-preservation and the reproductive or sexual instinct—are universally accepted as fundamental, and to these probably may be added a third, which Trotter has termed the "*herd*" *instinct*, meaning by this the instinctive desire of every normal human being to be one of a group and to conform more or less in feeling, thought and action to the habits of that group.

With these instincts there are associated certain states of feeling which take the form of a desire for the fulfilment of the instinctive need, and are called the emotions. To take an example from the instinct of self-preservation, for instance, in the face of an enemy, there may be a feeling of fear or a feeling of rage representing a desire to be removed from, or to remove, the potential source of injury. The emotions lead by way of the sympathetic nervous system to important bodily changes, which appear in most cases to be preparatory for action, and in themselves lend new force to the instinctive impulse.

In the mental activities and behaviour of the adult, these fundamental forces of instinct and emotion are revealed in the form of numerous trends in the personality, which are not infrequently in conflict one with another, but tend in a well-balanced individual to be co-ordinated towards a common goal. Thus in the life history of a normal person the instincts of self-preservation and sex find their expression in the competitive struggle for wealth and position, and in marriage; while the instinctive desire to do that which is generally approved by the group is satisfied at the same time. Other normal outlets may take the form of competition in sport, the formation of hobbies, intimate friendships, social activities of various kinds, and so on. The efficiency and happiness, or, as we may term it, the mental health, of the individual, depends upon the degree to which he is able to find expression for the instinctive forces of his personality in ways which are consistent with one another, and which lead towards the goal of personal and social progress. This involves from the first a continuous process of adaptation of the instincts to one another, and their adjustment to changes both in the body and in the environment.

The nature of this adaptation and its difficulties depend upon a number of variable factors. Thus the degree to which an individual is endowed at birth with each of the instinctive forces is in itself variable, and this is clearly a matter of fundamental importance in seeking to understand his behaviour, or the power of one set of instinctive reactions may be directly impaired by disease or injury, as in the case of sexual regression in the male following disease of the pituitary body, or loss of the testicles. Again, bodily illness of any kind leads to impaired efficiency as a whole of the mechanism by means of which the process of adaptation is effected, and will, therefore, call for readjustments in the general plan of the mental activities. A man who has lost his eyesight or a limb, or whose range of activities is limited by cardiac disease, is required to find satisfaction in life in new ways, to direct his energies into other channels. Finally, environmental factors of all kinds provide an almost infinite variety of obstacles and pathways. Inherited circumstances of poverty or wealth, the conditions of

home and school, early friendships, disappointments and bereavements, all play their part in calling for adaptation and adjustment.

Under the conditions of modern civilisation, the progress of mankind has come to depend more and more upon the merging of individual interests into those of the community, and a successful adaptation to these conditions makes considerable demands upon the powers of the individual, who, being at birth a comparatively primitive creature, has to undergo a somewhat rapid process of evolution in the course of development to adult life. Much depends upon the proper development of the herd instinct, to which therefore all systems of education are especially directed. The ethical and moral standards acquired during adolescence are among the most important assets of the fully developed personality.

## PSYCHOPATHOLOGY

The diseases to be described as the psychoneuroses arise from failures of adaptation and adjustment in the sense in which these words have been used already. The result of such a failure is what is popularly called a "nervous breakdown." The symptoms are variable, but depend upon the following principles which govern the mental processes and their bodily accompaniments:—

**1. The Emotional Reaction.**—Whenever the emotions are strongly aroused by any external or internal stimulus there results a state of mental excitement which has an unpleasant quality and is termed *anxiety*. This normally persists until the situation has been met, and is then followed by a sense of relief.

In proportion to the amount of anxiety, reflex bodily changes occur. These are effected chiefly through the autonomic nervous system and are beyond voluntary control. They may be conveniently classified as follows:

*Circulatory System.*—A sudden and severe emotional stimulus may cause temporary cessation of the heart-beat through vagal inhibition. This results in loss of consciousness, and is responsible for the common faint or swoon of emotional origin.

Less severe stimulation causes acceleration of the pulse with forcible thumping action of the heart and throbbing of the large vessels.

The peripheral arterioles and capillaries, under the influence of severe emotion, may contract, with resultant pallor, but more commonly dilate, the phenomenon of blushing being due to this cause.

Except in the cases of severe emotion with vagal inhibition, the blood pressure is raised.

*Alimentary System.*—The secretion of saliva is diminished. There may be spasm of the pharynx and œsophagus. The movements and tone of the stomach and intestines are diminished, there is spasm of the pyloric and ileocolic sphincters. In acute emotion vomiting may occur. Gastric secretion is abolished or reduced. The pelvic colon contracts, and the anal sphincter is relaxed, leading sometimes to evacuation of the bowels.

*Respiratory System.*—The respiratory movements are increased in depth and frequency, and with this there may be a sensation of "suffocation."

*Genito-urinary System.*—The secretion of urine is increased, and the bladder contracts. This leads to a call to micturition, or in extreme cases there may be involuntary relaxation of the sphincter with incontinence of urine. In males prostatic secretion and emission may occur.

*Endocrine Glands and Metabolism.*—The output of adrenalin is increased, and this in turn may cause further stimulation of the organs innervated by the sympathetic system. The blood sugar is raised, and glycosuria may occur. Metabolism generally is increased. (*See also* p. 579.)

*Nervous System.*—Besides the mental condition of anxiety, which fills the mind to the exclusion of purposeful thought and of sleep, there may be tremor of the limbs and sensations of numbness. Discomfort or pains in the head are

commonly experienced, and are probably due to changes in the cerebral circulation. The pupils are dilated, and the eyes may be protruded.

*Skin.*- The erectors of the hair follicles may be excited, producing the phenomenon commonly described as "goose flesh." Profuse sweating may occur.

The emotional reaction, if it persists for any length of time, is accompanied by an unusual sense of mental and physical fatigue.

**2. The Conditioned Reflex.**—Pavlow has shown in animals that by means of experience a previously indifferent stimulus may become so closely associated with an effective one as itself to become effective. The experiment in which the sound of a bell was in this way converted into a stimulus to the salivary reflex is familiar to students of physiology. In the same way an indifferent stimulus may through association become capable of producing the emotional reaction.

The association may be produced either by frequent repetition or by the coincidence of particular circumstances with an especially severe emotional reaction. Many examples might be quoted of such an association leading to a conditioned emotional reaction. During the recent war men who had been exposed for long periods to occasional shell-fire would afterwards exhibit tremor and palpitation at any loud and unexpected noise. A woman who was summoned to the telephone to hear that her husband had been killed suffered for several months afterwards from a sensation of extreme anxiety and palpitation whenever she was called to the instrument.

Similar emotional disturbances may be experienced by a man who has met with an accident while driving a motor car when next he takes the wheel, even though he is an experienced driver and the road is clear. Such associations are usually lost with further experience in the same way as the simpler conditioned reflexes established in animals can be broken down by further training.

**3. The Unconscious Mind.**—To any one who has reflected upon the matter it must be evident that one is not fully aware at a given moment of all that is going on in the mind, and further that one may at times act in a manner which implies purposeful thought without being aware of any such thought or purpose. Thus, for instance, a man absorbed in his work may remain seated at his desk for some time after the gong has summoned him to dinner. Given that his auditory apparatus was intact, an observer might reasonably conclude that the man had decided to postpone going in to dinner in order to complete his task. The subject, however, might honestly declare that the reason of his delay was that he had not heard the gong. Or, again, a man immersed in deep thought may put a letter into a post-box, yet remain completely unaware that he has done so. In these instances the rejection of the summons to dinner and the posting of the letter provide examples of unconscious mental processes. Such phenomena become more intelligible when the nature of attention is considered.

Attention may for the moment be compared to a searchlight whose beam is focussed upon a stage on which are represented the mental processes. No part of the stage is ever empty of incident, but its total area is large in comparison with the circle of illumination. Events in the centre of the beam are brightly lit, those in the periphery more dim, while the greater part of the drama is enshrouded in darkness.

The illuminated area may be taken to be the field of consciousness. The remainder of the stage represents the unconscious mind. Returning to the instance in which the dinner gong was ignored, we may say that this event took place upon an unlit part of the stage. It did not come within the circle of illumination or field of consciousness, but remained an unconscious mental process.

We may now carry the analogy a step further and imagine the searchlight to be mounted upon a pivot, so that it may easily swing in this or that direction. Let us assume also that objects and episodes upon the stage have power to swing the searchlight towards them or away according to the emotions with which they

are invested. Events which are associated with the emotions of curiosity or desire tend to attract the beam (positive influence); those which inspire shame or fear repel it (negative influence).

Some objects may have so powerful an attraction that they continuously occupy a central place in the field; others may exert so strong a repelling force as never to come within the area of central illumination, since whenever they appear upon its margin they cause the searchlight to swing away from them.

If at a given moment the centre of the beam be upon episode or object A, with a low positive influence, and upon the margin of the field appears object B, with a more powerful attraction, the tendency of the light will be to swing towards B, unless the attraction of A is in some way increased.

For example we may take the case of a schoolboy on a summer afternoon bent upon his preparation work with the object of completing it as soon as possible. For the moment his book occupies the centre of the field of consciousness. Then, as it were faintly, the sounds of cricket outside appear upon the margin of the field. The beam of attention swings automatically in the direction of cricket, to the exclusion of the work. Then comes the thought that the quicker his task is done the sooner he will get to his play. So the attractive value of the work will be enhanced, and the beam of attention again is focussed upon it

The emotions themselves form a part of the mental activities that proceed upon the stage, and lie to a great extent outside the field of consciousness. Thus a man may be unaware of the motives which direct his attention. This is often apparent in scientific work. The aim of the scientific worker is to discover the truth. He is, however, only too often influenced unwittingly by the desire to prove his own theory correct. Such facts as are consistent with that theory attract attention. Those which are contrary to it escape his notice. The selfish motive which is responsible for this is itself repellent, and remains unseen.

This tendency for a man to remain unconscious of his own emotions if they are of a repellent nature is of great importance in the causation of the psychoneuroses. Thus, for instance, in the recent war a man whose training had taught him to regard the emotion of fear as tantamount to cowardice would sometimes remain oblivious to the presence of this emotion within himself. The emotional reaction would then be present without cognisance of the emotion, and the individual would be driven to explain the phenomena of palpitation, sweating and so on as evidence of bodily disease.

Furthermore, he might be led by his fear to evade his duty without being conscious of the motive which impelled him. Physical disease under such circumstances offered a way of escape, and in this state of mind a man would, in the face of all reasonable considerations, accept the least suggestion of bodily disability.

A blow on the arm with temporary numbness would suggest the idea of paralysis of the limb. This idea, exerting by means of the unconscious motive a strong positive influence, would keep the searchlight of attention fixed upon it; all evidence to the contrary would for a similar reason be excluded from the circle of illumination, so that in a short while the paralysis of the arm became a fixed belief.

Many of the phenomena of daily life depend upon the influence of unconscious motives in directing attention towards certain aspects of self and environment and away from others. Men are notoriously blind to their own faults. They tend to remember their virtues and successes, to forget their failures and peccadilloes. Many acts of forgetting depend upon such a cause.

The man who is being influenced in his actions or his judgments by an unconscious motive is usually ready to defend his position upon logical grounds. His arguments, however plausible, prove on analysis to be fallacious. They are in fact further products of the hidden motive, and to distinguish them from processes of true reasoning they are called by psychologists "rationalisations."

In the causation of every case of psychoneurosis or "nervous breakdown" there are four factors to be considered. These are:—

1. Inherited lack of instinctive balance.
2. Inadequate development of sound mental habits.
3. Bodily illness.
4. Exceptional environmental stress.

## ANXIETY NEUROSIS

is a state of mental distress or anxiety, usually accompanied by physical symptoms, which is out of proportion to the facts of the situation in which it arises.

**Ætiology.**—1. Heredity may play an important part. Some individuals appear to be constitutionally sensitive and therefore prone to emotional over-reaction. In such the illness will usually have developed during childhood.

2. Acquired habits of mind as a rule are the chief cause of the illness. These frequently originate in childhood as conditioned reflexes. The emotional reactions of the child depend largely upon those of the adults who surround him. The child whose mother shows signs of terror at a thunderstorm will himself develop an excessive emotional reaction to this stimulus. So with other and more commonplace happenings. If the mother displays anxiety over the child's health, he himself is likely to grow up with an exaggerated fear of illness. Particular emotional experiences also may have a lasting effect in this way. A girl who has been the victim of an indecent assault may develop a morbid fear of the male sex, and so with other kinds of emotional trauma. In adult life the common fault is that of ignorance. In response to some adequate stimulus, an emotional reaction occurs with its train of physical accompaniments. There is, for instance, a feeling of faintness and palpitation of the heart. The subject takes this to be evidence of cardiac disease, and develops anxiety over his symptoms. This in its turn leads to further emotional reaction and palpitation. So a vicious circle is frequently set up which in time becomes a veritable habit.

The original emotional reaction may simply have been misunderstood. Often, however, the emotion itself may have been unconscious—an episode upon the unlit portion of the stage—and this for reasons such as have already been discussed. A man may, for instance, be seized with an access of panic at having to give an account of his work to his superiors. The emotion is one which is invested with a sense of shame and therefore fails to come within the field of consciousness. The emotional reaction, with its consequent palpitation, anorexia, gastric discomfort or other physical symptoms, then appears as an isolated phenomenon, and actually becomes a source of further anxiety. Unfortunately much harm may result in such cases from erroneous or uncertain medical diagnosis. If the physician accepts the symptoms as having a basis of physical disease, the patient's anxiety is likely to become greater, and the symptoms to be aggravated.

3. Physical disease and disorder may contribute to the development of an anxiety neurosis in two different ways. They may, in the first place, form the starting point for a vicious circle, as has been described, if the patient is already predisposed by past experience to over-anxiety about his health, or if his physician takes an exaggerated view of the gravity of his illness. In the second place, fatigue and toxic illness may by their direct influence upon the mental processes impair judgment and give rise to false perspective, so that molehills in the path of life are regarded with the anxiety due to mountains.

4. The definition given excludes environmental stress from the ætiology of anxiety neurosis. It must be remembered, however, that the facts of the situation may not be apparent save to the patient himself. Symptoms of an anxiety state which appear to the physician to be exaggerated may have their real origin in hidden troubles of a proportionate value.

**Symptoms.** —MENTAL.—At the root of all the symptoms lies morbid anxiety. This, however, is by no means the most frequent complaint. The anxiety itself may be latent, and the secondary symptoms first lead to a medical consultation. Common complaints are of inability to concentrate, loss of memory, indecision and a general sense of inadequacy. Such are the natural results of continuous anxiety, which fills the mind to the exclusion of other subjects. They are, however, often misinterpreted by the patient, who regards them as signs of approaching insanity. *Insomnia* is frequently an important symptom. Two varieties are met with. In the first, which is associated with manifest anxiety, the patient's mind is so full of his cares as to prevent the onset of sleep. The other is met with in cases of latent anxiety, when the patient is to a great extent unconscious of the emotional origins of his illness. In these cases it often happens at bedtime that the mind becomes over full of a stream of thoughts upon neutral subjects, to the exclusion of any view of the hidden worry, and also of sleep. When sleep has eventually been won in such a case it is often broken by terrifying dreams or nightmares, from which the subject wakes with all the sensations of extreme terror. Anxiety about the sleeplessness and its possible effects aggravates the condition.

The anxiety when manifest may be of a general or of a particular nature. General anxiety reveals itself in a tendency to exaggerate difficulties, and exhibit an excessive emotional reaction to all stimuli. Particular anxieties (sometimes called phobias) are numerous and variable.

*Fears of Disease.*—The patient becomes convinced as the result of some abnormal sensation that he is suffering from a serious disease. The common fears are of cancer, syphilis and pulmonary tuberculosis. Although examination and reassurance may for a time dissipate the fear, it usually recurs until the underlying neurosis has been properly treated. Fears of cardiac, renal and gastro-intestinal disease are also of common occurrence.

*Fear of insanity* has already been alluded to. It is of common occurrence, and is based by the patient upon the difficulty in mental concentration, and sense of emotional tension from which he suffers.

*Fears of certain Situations.* These are innumerable. Some of the commonest are fear of open places, of crowds, of closed spaces, of travelling in trains. They may be paralysing in their intensity and seriously interfere with the patient's life.

*Fears in relation to Sexual Matters.*—The commonest of these concerns masturbation. The patient has perhaps practised this habit in the past or continues to practise it at intervals with much emotional conflict. He feels that he has thereby ruined his mental and physical health, and that his illness is due to this cause and is a punishment for his sins. Any chance encounter with sexual topics gives rise to an emotional over-reaction. The subject being one of which he is ashamed, the origin of his anxiety is frequently hidden among the unconscious mental processes. Similar fears may have their origin in anxiety concerning sexual thoughts of which the subject is ashamed. In certain phases of life, and especially in certain persons, these may be of a homosexual nature.

The occurrence of seminal emissions is sometimes the centre of anxiety. Since anxiety itself may result in seminal emission, this is apt to set up a vicious circle.

Impotence in the male is a not uncommon complaint. The symptom may consist of premature ejaculation or of inability to obtain a sustained erection. This is commonly associated with fears concerning past masturbation or seminal emissions.

NERVOUS.—*Extreme liability to fatigue* (mental and physical) is a common symptom. The patient complains that he is incapacitated for his work, is unable to sustain his part in conversation, and becomes exhausted after walking up a flight of stairs. Careful inquiry will usually discover that this fatigue is relative in its action, varying with the extent to which the patient is interested in his

surroundings. Thus a man may be unable to concentrate upon work of a monotonous character for more than half an hour, yet give his undivided attention to a play for two or three hours at a stretch. Despite his inability to meet the obligations of social small talk, he will discuss at great length and with the utmost keenness the symptoms of his illness, and although he may feel unequal to visiting his doctor, will walk a mile to see a football match without undue fatigue.

*Headache* is another common symptom. Sometimes it is described as a pain, which is then as a rule intermittent, coinciding with moments of intense anxiety. More frequently it is referred to in terms of discomfort and is continuously present, as a feeling of pressure upon the top or back of the head, a leaden feeling, or a sensation of tightness, fulness or heat.

*Giddiness* is a frequent complaint. It may be continuous or take the form of attacks which last from a few minutes to several hours. There is no sensation of whirling (as in the vertigo of organic disease), but a subjective feeling of unsteadiness with fear of falling. This fear may be so vivid that the patient dare not move and will cling to others for support. When the symptom is continuous he will sometimes complain that he cannot walk abroad without the arm of a friend to steady him.

*Tremor* of the limbs is an occasional symptom in association with paroxysms of fear.

Alimentary System.—Dysphagia is not uncommon. It may be in part due to lack of saliva, but the complaint is usually that the food sticks in the throat and will not go down. It is often associated with fear of cancer. Gastric discomfort and flatulence are common complaints. The feeling is one of fulness which bears no constant relation to the taking of food, but commonly occurs during or immediately after the meal. The patient seeks to relieve this sensation by belching, and in consequence swallows air, which may produce actual distension of the stomach. A characteristic feature of this form of dyspepsia as a rule is its selective nature. The patient will assure us, for instance, that he invariably has indigestion after eating beef but not after mutton; tea is fatal to his stomach, but coffee innocuous; he cannot tolerate green vegetables, but can eat raw apples with impunity.

Constipation is a common complaint. The patient having for some trivial cause been unable to obtain an evacuation and having had recourse to a purge, becomes anxious upon the next occasion whether his bowels will open without the aid of a drug. The anxiety itself interferes with the reflex process of defæcation, and this in turn leads to a habit of mind in which the consciousness of an aperient taken beforehand is necessary to success.

Diarrhœa is sometimes complained of, but on analysis proves a misnomer, the fact being that the patient feels need to open his bowels under particular circumstances, especially when no convenience is available.

Cardiovascular System.—Præcordial discomfort and palpitation are the prominent complaints under this head. Extreme liability to blushing may be the focus of anxiety.

Respiratory System.—Subjective sensations of choking and dyspnœa may occur with paroxysms of panic and fear of impending death.

Skin.—There may be profuse sweating, commonly associated with fears of consumption. Numbness and tingling is an occasional complaint.

An anxiety neurosis which has persisted for more than a week or two usually leads to a certain degree of true nervous exhaustion, with its attendant symptoms.

**Diagnosis.**—This must depend partly upon the exclusion of organic disease as a direct cause of the symptoms, partly upon the recognition of morbid anxiety as their source. As a rule this is not difficult. It must be remembered, however, that certain chronic intoxications, of which phthisis is the chief example, may be important as causal factors.

When the illness has been recognised as a mental disorder it still has to be distinguished from others, such as dementia præcox and manic depressive insanity.

In some cases of dementia præcox morbid anxieties and fears may occur as early symptoms. These, however, differ from those of the anxiety neurosis in being not only unreasonable, but inexplicable, except in terms of the patients' fantasies if these can be elicited. The patient may, for instance, be anxious about the state of her digestion, but behind this will lie the fixed belief that the lining of her stomach has been corroded away by poisons introduced into her food.

The symptoms of a mild case of manic depressive insanity may resemble in many respects those of an anxiety neurosis In the former illness, however, depression is usually a more prominent feature, and is largely independent of circumstances. The patient cannot so easily be roused to take an interest in his surroundings; his symptoms show little variation with changes in his environment. He gains little or no comfort from the physician's reassurance, whereas the neurotic almost always shows a marked, if temporary, improvement after such an interview. The manic depressive is more consistently apt to blame *himself* for his troubles and will often in giving his story interpose remarks to this effect. His depression also is apt to show a daily rhythm, being at its worst in the early morning He is less likely to be troubled with difficulty in getting off to sleep than by waking in the small hours of the morning with a feeling of profound melancholy. The differential diagnosis may require in some cases a period of observation. It is important in a doubtful case that it should be settled, for the treatment of the two conditions is dissimilar. The manic depressive needs quiet and rest, the neurotic support and encouragement. There is, moreover, a serious risk in the manic depressive of suicide.

As a useful point of general distinction between a person suffering from anxiety neurosis and one who is insane, it may be said that whereas in the former the emotional reaction will be found to be out of proportion to the facts of the situation (when these are discovered), in the latter there is *no* true relation between the emotional reaction and the situation.

**Prognosis.**—So far as serious sequelæ are concerned the prognosis is good. These patients do not become insane; but if untreated they may be incapacitated for work and social intercourse for long periods of time, during which they are miserable themselves and a burden to others. With proper treatment the outlook is good except in those cases in which the hereditary factor is prominent.

**Treatment.**– This consists of analysis of symptoms, explanation and reassurance, the procedure being termed psychotherapy. Rest and drugs are also needed in certain cases.

The taking of the history and the physical examination are all important in the treatment as well as the diagnosis of the condition.

The patient should be encouraged to describe his main symptoms in detail, and further questions should then be directed towards the various bodily systems. Notes should be taken. The patient should then be systematically examined. If analysis of the history and physical examination are sufficient to exclude organic disease, the patient should be told this. If any further investigation is necessary, he should be informed of this, and arrangements should be made as soon as possible for carrying it out. If there are any definite signs of physical disease, this knowledge should not be withheld from the patient, who must be led to feel that he is being treated with absolute frankness. He should further be given some idea of what the signs are and what is their significance. He may thus be led to understand that such organic defects as may be present are insufficient in themselves to explain his symptoms.

The taking of the history and physical examination should occupy at least one hour. Often this is insufficient, and a further interview must be arranged. It is essential that the patient should feel confident that his whole story has been

appreciated, and that no possibility of physical disease has been neglected, for these preliminaries form the basis for further treatment.

Having assured the patient that his symptoms are not due to organic disease, the physician should proceed to explain that they are of course real, not imaginary, symptoms, and are such as may have their origin in emotional disturbance. An outline should be given in suitable language of the psychological principles described above (*see* p. 891). It may be pointed out, for instance, that a cat when frightened by a dog shows many disturbances of its bodily organs, including chemical alterations in the blood, acceleration of pulse rate, and rise of blood pressure, which may be measured, and alterations in the gastric movements, which may be observed with the X-rays, yet these changes do not indicate any disease either of the organs affected or of the nerves controlling them.

Further interviews should then be arranged for analysis of the patient's symptoms in the light of these principles.

The symptom is first traced to the date of its origin, and the patient is cross-examined as to any emotional difficulties which may have existed at the time. Often at first he may fail to call to mind any such troubles. The existence of unconscious mental processes should then be explained, and examples given of the way in which an emotional disturbance outside the field of consciousness may nevertheless give rise to symptoms of an emotional reaction.

In this way it is usually possible to discover adequate causes for the various symptoms, and as these are gradually explained they disappear. An attempt should also be made to discover and explain to the patient the underlying habits of mind which have led up to his illness. In some cases these will be plain to his view; in others a great deal of skill and experience may be necessary to unravel the emotional tangle.

Special methods of mental analysis may be necessary in some cases in order to bring to the light unconscious mental processes of importance. For these the works of Freud and his followers upon psychoanalysis should be consulted. The great majority of cases can be successfully treated by less complicated methods. As the patient begins to realise that he is being treated with sympathy and understanding, and that the treatment offers a real hope of recovery, his desire to co-operate gains strength to turn the light of attention towards aspects of his mind which have before been concealed by motives of shame or dread. Thus he gradually reveals himself to the physician, who is able by collecting the scattered threads of the story to discover the causes of his neurosis. In the presentation of these causes the physician needs to be sympathetic, for it will involve asking the patient to face faults and weaknesses in himself to which he has been blind. There is, however, nothing which these people appreciate more than the quiet assurance that others have passed through similar experiences, and that many of their friends (with whom they are apt to compare themselves unfavourably) have almost certainly suffered, perhaps in secret, from the same miseries and fears.

Since the treatment is chiefly concerned with the elicitation and evaluation of symptoms, those which have been described may be considered again under this heading.

*Neurasthenic Symptoms.*—It has already been explained that an anxiety neurosis, if sufficiently severe and prolonged, is likely to lead to true nervous exhaustion. It is wise therefore whenever symptoms of undue mental or physical fatigability are prominent to commence treatment on the lines advised for nervous exhaustion (*see* p. 888). Insomnia should always be an indication for rest in bed as a preliminary to further treatment. It is important that the reasons for this procedure should be explained to the patient, who should not be allowed to conclude that rest alone will bring about recovery.

A week in bed, with subsequent return to normal activities in a further two or

three weeks, will usually suffice. Psychotherapy should proceed simultaneously. If possible, the patient should be in a nursing home for this part of the treatment.

*Mental Symptoms.—Inability to concentrate* is so often interpreted as a symptom of incipient insanity that inquiry should always be made in this direction. The fear is often concealed by the patient from others, if not from himself. This is especially likely if there is any family history of mental disease—a fact sometimes omitted or denied at the first interview. Complete assurance should be given upon this point. It should be made clear to the patient that, although his illness is a mental disorder, it belongs to a class of minor disorders or psychoneuroses which is absolutely distinct from the major mental disorders or psychoses, and that patients do not pass from one illness into the other. The lack of concentrating power should then be explained as being due to anxiety. A man who has some unsettled worry at the back of his mind cannot give his full attention to his work. The origin of the anxiety and the causes of its perpetuation should then be dealt with in the manner already indicated.

*Insomnia.*—In so far as this is directly dependent upon anxiety, it may be expected to improve with treatment of the cause. The patient often develops undue anxiety over this particular symptom, and will then need to be assured that many persons are in the habit of doing good work with fewer hours of sleep than he himself is getting, and that without any untoward symptoms. It should further be explained that anxiety is a *natural* cause of insomnia, and that as the treatment proceeds he will find himself improving in this respect. The power of hidden anxiety to cause terrifying dreams should also be discussed.

If the insomnia is of recent origin, such simple measures are often sufficient. Sleep, however, is a *reflex* process which depends largely upon habit, and this unfortunately holds good also for insomnia. Therefore, if this symptom has persisted for more than a few days, the physician should not hesitate to treat it by means additional to psycho-therapy. The patient, as always, should be given an explanation of these means and their rationale.

The additional means available are (1) drugs, (2) habit formation, and (3) muscular relaxation.

1. If the insomnia is of comparatively recent duration, and is an object of much anxiety, the following plan is suitable. The patient is instructed the first night to take a sufficient dose of a reliable hypnotic to induce sleep (medinal grs. x, paraldehyde drs. ii, or chloral hydrate grs. xx, with pot. brom. grs. xl). On the following night he is told to have the medicine ready by his bedside and to take it without hesitation if he finds himself unable to sleep without it. A supply of the hypnotic is prescribed sufficient for a week. The patient should be assured that there is no danger of this procedure leading to a "drug habit." After a few nights it will usually be found that he will fall asleep naturally with the drug untasted by his bed, and will then have regained confidence in his ability to sleep unaided. When the insomnia is of longer duration it may be necessary to give sufficient doses of hypnotics to procure sleep over a longer period, the dose being gradually reduced as psychotherapy and other methods of treatment become more effective. Treatment with drugs should always be combined with the other methods referred to.

2. Most persons have some kind of a ritual which, under normal circumstances, precedes and favours sleep. The hour, the room, the order of undressing, the book read in bed, all play a part as conditions facilitating the sleep reflex. This also is true of insomnia. The patient who is being trained to regain the habit of sleep will therefore benefit from a change of surroundings and of bedtime habits. A change of bedroom is often in itself useful.

3. Muscular relaxation is the most important physical condition for the onset of sleep. Patients suffering from anxiety are frequently in a state of excessive muscular tension, which places them at once at a disadvantage. This symptom, of course, depends upon the underlying neurosis, and improves with treatment

of its cause. Nevertheless as a cause of insomnia it may be controlled to some extent by training the patient to relax his limbs. The physician should ask the patient to allow one limb to lie absolutely passive, and test its passivity by raising and handling it. It will usually be found that the patient, so far from letting the limb follow the directions of gravity, is constantly exercising it by his will. With daily practice and concentration, most persons can be taught to relax in turn, and finally simultaneously, all the muscles of neck, trunk and limbs, and this becomes a valuable preliminary to sleep.

*Fears of Disease.*—The preliminary examination must be sufficiently thorough to provide an impregnable position from which the physician can give an assurance that the symptoms are not due to organic disease. This assurance, although it may give temporary relief, is not in itself sufficient treatment. The intelligent patient as a rule will proceed to state that the symptoms exist and must have some cause. The physician then proceeds to explain the manner in which bodily symptoms, often simulating those of organic disease, may result from emotional disturbance. The fear is traced back to the date of its origin, and analysis and explanation are continued. Fears of disease are often associated with primitive ideas of illness as a visitation for past sins, and thus not infrequently have their origin in remorse over sexual misdemeanours.

*Fears of special situations* should be analysed by giving the patient a preliminary explanation in simple terms of the conditioned reflex and unconscious mental processes. When he has been taught that behind his unreasonable terror there may be an intelligible cause he is urged to carry his thoughts back to the time when the trouble began, and to talk freely of his life at that time, especially of any secret hopes, fears, or disappointments. If the physician himself is sufficiently assured that a cause is to be found, the facts will as a rule gradually disclose themselves. The patient is led to face the truth, and encouraged not to avoid the situations which provoke his fears, but to train himself to meet them in the light of the common-sense explanation which he has received.

*Fears in relation to Sexual Matters.*—The commonest of these concern masturbation. Upon this subject the physician, whatever his own prejudices, must be ready to give unbiassed and impersonal advice.

The patient often feels that in practising this habit he has not only fallen from his own ideals, but has proved himself morally degenerate and unworthy to associate with his fellows. He may be honestly assured that among healthy males there are few, if any, who at one time or another have not practised masturbation, and that the friends and acquaintances with whom he compares himself so unfavourably have certainly had experiences similar to his own.

Additional fears are concerned with the ill effects which masturbation has been supposed to have upon the health. Such notions as that the habit leads to insanity and consumption are occasionally inculcated by ignorant parents or schoolmasters, and are sedulously fostered by quacks for their own mercenary ends.

Actually there is no evidence to show that the act of masturbation *per se* has any more harmful effects than natural sexual intercourse. The sequels, such as headache and depression, of which the patient complains, are the results, not of the act itself, but of the mental distress with which it is associated, and this is to a large extent proportionate to the patient's fears of its consequences. The patient will often maintain that the seminal loss is in itself weakening. It may be pointed out that it is no greater than in normal sexual intercourse which occurs regularly in the lives of thousands who are in no way the worse for it. As to the remorse occasioned by falling from the standards which he has set himself, it may be pointed out to the patient that possession of such standards is itself a point in his favour, and that it is in the nature of ideals that a man's reach should exceed his grasp.

Fears concerning frequent seminal emissions should be dealt with in similar

fashion. Such "losses" cannot cause the symptoms which the patient attributes to them, and are in themselves harmless. Anxiety, on the other hand, may be the cause of seminal emissions.

Frank discussion of these subjects will, as a rule, reduce them to their proper place in the mental perspective and leave the way clear to further inquiry into the origin of the neurosis. The patient who suffers from anxieties of this nature is often shy of confessing to them, but will always respond to inquiries if these are made in the proper way. He should be told that all normal persons suffer to some extent from sex difficulties, and he should be questioned directly about his own experiences in regard to seminal emissions and masturbation.

Difficulties of this nature are less frequently encountered among women, and when present are less easy to disclose. Nevertheless worries over masturbation are not uncommon, and must be discussed with similar frankness if there is reason to suspect their existence.

Sexual impotence in the male is commonly associated with worries over past masturbation. Such a man on his marriage proceeds to intercourse in a state of anxiety lest his past misdemeanours may have rendered him incapable of the sexual act. The anxiety is itself sufficient to cause impotence. When it has been explained to the patient that masturbation is a universal habit he will regain confidence. Or a man who has previously been normal in this respect may attempt intercourse at a time when he is suffering from true nervous exhaustion, and having once failed, may develop anxiety which renders him impotent on future occasions. Here again frank discussion and explanation will often suffice to cure the condition. It is sometimes necessary to see the wife separately and clear her mind upon the subject of sex hygiene. There is no more potent factor in causing impotence than a wife who exhibits doubt or anxiety as to her husband's sexual ability.

*Physical Symptoms.*—After the negative physical examination the patient's symptoms should be examined in detail, and explained as being due to emotional reaction. Such an explanation is often new to the patient, and he may need time to comprehend it. Any attempt at forcing it upon him is likely to arouse opposition. Arguments have to be repeated and illustrations given from other cases or from daily life. Every one knows, for instance, that worry may cause a headache. That a man who stands up to address a large audience may experience a sensation of giddiness or unsteadiness on his feet in association with his anxiety to create a good impression is a matter of common experience. Tremor of the limbs is a frequent accompaniment of fear.

Dyspepsia is a well-known symptom of anxiety. A man whose digestive functions are disturbed by a hidden worry is apt to ascribe his discomfort to some particular article of his food. Having once conceived this idea, whenever he partakes of this foodstuff it is with the fear that it will produce symptoms. The fear itself is sufficient to interfere with normal digestion. A vicious circle is thus set up which is at the bottom of the "selective dyspepsia" which is so common a symptom of the anxiety neurosis. Flatulence is due to misinterpretation of the sensation of fulness. In his efforts to belch the patient swallows air, and may produce an actual distension of the stomach. If the patient is simply instructed to restrain the impulse to eructate, he may prove for himself the truth of this explanation.

Præcordial discomfort and palpitation are again well-known accompaniments of anxiety, as also are sensations of respiratory embarrassment and perspiration. When the patient is satisfied that his particular symptoms have no organic basis, and are such as *may* be caused by emotion, he is led along the lines already described to call to memory the origin of his symptom and its emotional contexts. Thus the inner history of the illness may in time be disclosed.

The aim of treatment is to give the patient such insight into the origin of his symptoms as will rob these of the appearance of illness. Even when this is

accomplished his troubles are not necessarily at an end, for he may still be obliged to meet the emotional difficulties which formed the basis of that illness. Often the illness itself will have provided some excuse for avoiding these difficulties. Recovery may bring with it the necessity to return to distasteful surroundings. Hence at this point there is a tendency to relapse. The patient begins again to doubt his mental or physical fitness for ordinary life. This tendency must be met by analysis and explanation of the unconscious motive which is responsible—the desire to find in illness an excuse for evading unpleasant duties. Often the situation may be made easier for the patient by an interview with relatives or friends, whose sympathy may be enlisted on his behalf.

## HYSTERIA

By hysteria is meant a disorder of the mental processes manifesting itself by physical symptoms which can be removed by psychotherapy.

**Ætiology.**—Symptoms of hysteria may appear at any age after infancy. They are more commonly met with in the female sex, but under exceptional conditions, such as those met with in the Great War, may be of frequent occurrence amongst adult males. In the majority of cases heredity plays a relatively unimportant part in the ætiology. Of far greater importance are the habits of mind developed by precept and example during childhood and adolescence. In many instances slight physical illnesses or accidents are exaggerated by the parents, who comfort the child with toys or sweetmeats, or the symptoms form an excuse from the performance of some unpleasant duty, or they may secure for the child the desired position of being the centre of attention. In this way too often are laid the foundations of a habit of mind which tends to make the individual unconsciously rely upon physical symptoms as a refuge from the various difficulties of every-day life. On the other hand the person who is trained in the exercise of his critical faculties, and is able to take a more or less logical view of his life problems, even if he is unable to solve them, is unlikely to develop hysterical symptoms, unless his criticism is disarmed by peculiarly appropriate suggestion. The responsibility of education as an ætiological factor was clearly demonstrated during the Great War, when, although functional nervous disorders were probably equally common among officers and private soldiers, hysterical symptoms were relatively of far more frequent occurrence among the latter.

According to the modern conception of hysteria, the essential point in the ætiology of a particular hysterical manifestation is the presence of an *unconscious motive* for its existence. This has already been illustrated in the instance quoted above of the soldier developing a hysterical paralysis under stress of warfare when the unconscious motive was the desire to escape from danger. In other cases, the motive may be much more difficult to understand, but it is probable that in all cases it exists. Thus fits of a hysterical nature occurring in a young woman may be shown to result from a repressed wish to attract to her side an inattentive lover, a paralysis or tremor of the right hand may occur under circumstances which demand the writing of an unpleasant letter, or the symptoms may have as their motive in a shy individual the desire to assert himself and to become the centre of attention.

An additional cause for the symptoms is usually to be found in the shape of a *suggestion*. This may come from within. For instance, a woman has a twinge of pain in the back; she remembers a friend who had the same symptom and afterwards developed paralysis of the legs, and is convinced that she is suffering from a similar disease. Or the suggestion may come from without, from the chance remarks of friends or the erroneous opinion of a medical man.

Whatever the suggestion of disease, it is accepted without logical grounds by reason of the unconscious motive in the background. There is no more satisfactory excuse for evading emotional problems than the assumption of illness.

This, of course, is well recognised by the malingerer. But there is a real difference between the malingerer and the patient who is suffering from hysterical symptoms. The business of the former is to deceive others; the patient with hysteria deceives himself, and to provide a satisfactory solution of his difficulties this self-deception must be complete. The malingerer can, and will, discard his symptoms whenever it may be convenient; the hysterical disability, however inconvenient it may be in some ways, remains constantly present until the patient's belief in its reality is removed.

The **Symptoms** of hysteria may be of the most diverse nature, and may closely simulate many forms of organic disease, from which they are to be distinguished only by careful examination. By certain writers in the past a good deal of stress has been laid upon the value of certain physical signs said to be pathognomonic of the condition and therefore known as the *stigmata* of hysteria. More recently, however, it has been shown that these signs have probably in all cases been due to suggestions made by the physician in the course of physical examination. It is to be remembered that the hysterical patient is lacking in the critical faculty, and therefore his judgments upon the nature of his own bodily sensations are likely to be influenced by the least suggestion made by another, especially if this falls in with his own fixed idea of serious physical illness and is made by a person in whose knowledge of disease he has implicit belief. Therefore, for instance, in the course of our examination of cutaneous sensibility, the tone in which an inquiry is addressed to the patient may suffice to determine his answer, even if this be at variance with all considerations of common sense. Thus may be developed areas of cutaneous analgesia in which the patient states that he is unable to feel the pricking of a pin, even if this is pressed in deeply enough to draw blood. Or in the examination of the visual fields abnormalities may be discovered which vary with, and depend upon, the method of examination (Hurst).

It is important to remember that in a large number of cases of hysteria there are to be found signs of underlying organic disease either of the nervous or other bodily systems, which has formed the starting point for the development of a neurosis. The physician must, therefore, be prepared on occasions to find signs of hysteria and of organic disease in the same patient, and must, in summing up the case, decide what proportion of the symptoms is functional and is, therefore, amenable to cure by psychotherapy. For instance, a patient may be seen complaining of paralysis of the whole arm from the shoulder downwards starting from an injury received at work; examination reveals the scar of the injury to be below the elbow; the signs of division of the ulnar nerve at this level are present in the form of the appropriate wasting of the small muscles of the hand; sensory examination, however, shows the anæsthesia to extend up to the level of the axilla, and there is total loss of power of the muscles without any wasting or alteration in the tendon jerks. In such a case the greater part of the symptoms may be removed by the methods of psychotherapy to be described later, leaving a small organic residue which needs surgical treatment. Or a woman may be seen who has for some months been bedridden with total paralysis of the legs. Examination of the nervous system reveals definite signs of organic disease in the form of extensor plantar responses and absent abdominal reflexes, and a careful analysis of the history is sufficient for the diagnosis of disseminated sclerosis. Yet the defects of nervous function in the shape of paralysis, loss of sensibility, spasticity, or inco-ordination, are insufficient to account for the inability to walk. In such a case psychotherapeutic measures, though, of course, without any effect upon the underlying organic disease, may serve to restore to the patient the power of walking. Some of the commoner manifestations of hysteria will now be described under the headings Mental, Nervous and Visceral.

**Mental Condition in Hysteria.**—It has already been stated in the definition of hysteria that the disorder is primarily one of the mental processes, and

although the outward manifestations of this disorder usually take the form of physical symptoms, the mental condition may sometimes be recognised in the general behaviour of the individual. The essential feature in the mental state of hysteria is the process of dissociation whereby a group, or complex, of ideas and feelings belonging to the personality is shut out from the field of consciousness. In certain cases there may occur temporarily a complete reversal of this state, so that the whole field of consciousness is filled for the time being by the ideas and feelings previously excluded, while those which formerly constituted the conscious mental activities of the individual are, in their turn, shut off. Thus arise the cases of dual personality in which a person may for a period of minutes, hours, or even days, speak and behave in a manner wholly foreign to her usual demeanour, and on returning to her usual self have no memory whatever of what has occurred. The same mental process of dissociation is responsible for the condition of so-called hysterical amnesia, in which the individual loses all memory of some phase of his life which is associated with painful and unpleasant experiences.

The patient with physical symptoms of hysterical origin does not, as a rule, show signs of emotional stress, and is, therefore, not commonly "hysterical" in the lay sense of the term. On the other hand, it is frequently to be observed that these patients, in spite of a superficial anxiety to be relieved of their symptoms, show no real depth of feeling in this respect, and are, in fact, abnormally content with their condition. This is easily understood if it be admitted that the symptom itself is providing satisfaction for certain underlying wishes in the patient's mind of which he himself is not clearly aware.

**Nervous Symptoms.** —*The Hysterical Fit.* -The attacks known as hysterical convulsions and "fits of hysteria" are commonly caused by emotional disturbance. They occur as a rule in the presence of others, or in circumstances where they are likely to attract attention. The patient may complain of sensations of choking, giddiness, or faintness, and burst into a fit of crying or uncontrollable laughter. In other cases the patient falls to the ground, as a rule gently, or on to a chair or sofa, and at once passes into a state of convulsive movements. These, if they are witnessed by the physician, are as a rule easily discerned to be of a purposeful nature. There may be phases of tonic contraction in which body and limbs are held stiffly with hands clenched, but these never involve the respiratory muscles to such a degree as to produce cyanosis. The eyelids are generally closed, and resist attempts to open them; if they are opened, the eyeballs are rolled upwards under the upper lid. There are no true clonic twitchings. The patient may tear at her clothes, beat her head with her hands, or clutch at bystanders. She may gnash her teeth, and saliva may issue from the mouth, but the tongue is not bitten. There is no loss of sphincter control. Often the attack is punctuated by shrieks, groans or articulate cries. There is no true loss of consciousness. Though the patient does not reply to questions, she is influenced by what is said or done in her presence. The greater the alarm evinced by those around her, the more violent is the display of excitement. The attack may continue in this way for two or three hours. If the patient be ignored, the symptoms may at first increase in severity but if treated quietly, rapidly diminish. Neither during nor after the attack is there any disturbance of the reflexes. At its conclusion recovery is as a rule rapid and the patient is in a few moments again in possession of all her faculties. All knowledge of the episode may be denied. This is often a pretence, but may be due to a hysterical amnesia, in which case the patient becomes able as the result of treatment to recall the incidents of the attack.

**Disorders of the Special Senses.**—Hysterical blindness is by no means common; but many cases have been recorded arising out of the Great War. In most instances they occurred in the case of a man exposed to irritating gas and for the time being unable to see on account of acute conjunctivitis, photophobia and blepharospasm. Some of these patients remained blind for years before their symptoms were removed by psychotherapy. In such cases, of course,

there are no abnormalities to be found on opthalmoscopic examination. *Hysterical deafness* is not infrequent, and was another symptom commonly observed during the war in a man who had been temporarily deafened by the explosion of a shell. It is usually unilateral, but may affect both sides. Hysterical deafness may be diagnosed by the absence of any history or signs pointing to chronic inflammation of the middle ear, and by the performance of the tests for vestibular function. If under such circumstances the reactions to these tests are normal, the deafness is almost certainly of hysterical origin.

**The Cranial Nerves.**—Hysterical spasm of the orbicularis palpebrarum, or blepharospasm, is of quite common occurrence, and usually accompanies hysterical blindness. It commonly follows irritation of the conjunctivæ, as in chronic conjunctivitis, the usual cause in the war being irritant gas. The face and tongue also are sometimes affected by spasm. In this way a hysterical patient may simulate facial paralysis by contraction of one side of the face, the other side appearing weak by contrast while at rest.

*Hysterical mutism* is a not uncommon symptom, and has already been referred to. It may appear after a severe emotional shock, as, for instance, in the war cases, in which it may be imagined that the individual was for the moment struck *dumb with terror*. The patient is unable to make any sound whatever on attempting to speak, yet in some cases he may be able to cough quite audibly. In the condition of *hysterical aphonia* the patient is able to speak only in a whisper. This most commonly arises after an attack of catarrhal laryngitis, and may be regarded as a perpetuation of a symptom which was originally organic. On laryngoscopic examination the adductors of the vocal cords can be seen to be immobile. It is noteworthy that in a number of these cases the patient speaks in a high-pitched whisper without any hoarseness, which is in itself a point of diagnostic value. (*See* Paralysis of Laryngeal Muscles.)

**Sensory Symptoms.**—Complaints of pain or tenderness not infrequently occur as accompaniments of local symptoms of a hysterical nature. Thus there may be complaint of headache or backache and tenderness over the top of the head or the vertebral spines, or in association with hysterical contractures of the limbs there may be complaint of great pain on attempted movement. Hysterical anæsthesia has already been referred to, when it was stated that this symptom is usually produced as the result of neurological examination. It is often present also as an accompaniment of hysterical paralysis. It may usually be distinguished by the nature of its distribution, which does not follow any anatomical laws, but depends on the patient's own idea of anatomy. Thus, accompanying a paralysis of hand or foot, it is usually of a "glove" or "stocking" distribution.

**Motor Symptoms.**—These may be divided into paralyses and contractures and involuntary movements.

*Motor paralysis* is one of the commonest of all hysterical symptoms. It may take the form of a monoplegia, a hemiplegia, paraplegia of the legs, or paralysis of all four limbs. The paralysis is usually complete and may be flaccid, or accompanied by contracture of the muscles, the latter form being the commoner. As a rule, it is of sudden onset, commonly following physical or emotional trauma, or it may often appear in the form of a perpetuation of a weakness of organic origin, as, for instance, after a fracture for which the limb has been splinted for a considerable time. In both the flaccid form and that associated with contracture, the paralysis usually affects all groups of muscles equally without regard to cortical, segmental or peripheral nerve distribution. When the patient is requested to put the paralysed limb into action, in spite of much exhibition of effort, there is little or no movement. It is to be noted that, although there may be some atrophy of the muscles from disuse, this is never so marked as in the case of a lower motor neuron lesion, and that in the cases with contracture the rigidity does not conform either in type or

muscular distribution to the clinical picture of spasticity from a lesion of the upper motor neuron. Moreover, the anæsthesia which is frequently present follows no anatomical laws in its distribution, nor is there any abnormality of the tendon jerks. One phenomenon of positive value in the diagnosis of hysterical paralysis is the simultaneous or rapidly alternating contraction of the prime movers and their antagonists in attempting to perform any particular movement. Thus, if the patient with a hysterically paralysed arm be asked to perform the movement of flexion at the elbow, the observer by simultaneous palpation of both biceps and triceps can feel now one, now the other, contracting simultaneously, or in rapid succession, so that, in spite of much expenditure of muscular effort, either no movement at all or else a succession of jerks occurs. This phenomenon does not occur in any condition due to organic disease. In some cases a hysterical contracture may closely simulate an organic condition, but may usually be differentiated by careful neurological examination, including the investigation of the electrical reactions. A form of paralysis of quite frequent occurrence in hysteria has been described as astasia-abasia. In this condition the patient is able to perform all voluntary movements of the lower limbs with normal power and accuracy so long as she is lying in bed, but is unable to walk when set upon her legs. The involuntary movements of hysteria may assume the most diverse forms, of which various types of tremor may sometimes with difficulty be distinguished from those of organic disease. The signs, however, which are usually associated with the organic tremors of Graves' disease, disseminated sclerosis, paralysis agitans and other diseases are absent.

*The reflexes* in hysteria do not as a rule show any abnormalities, though it has been stated (Hurst) that in cases of hysterical anæsthesia of the abdomen the abdominal reflexes may be absent, to return with the disappearance of the anæsthesia.

*The functions of micturition and defæcation* are sometimes affected, the commonest symptoms being retention of urine on the one hand and dyschezia on the other. In such cases, great care must be taken to exclude the possibility of organic disease.

*Abnormalities of the posture and gait* in hysteria are as a rule easily distinguished from those which depend upon organic lesions. Disorders of gait frequently take the form of mannerisms, such as hopping or prancing, or even of walking sideways crab fashion. Among the postural abnormalities may be mentioned that of the bent back simulating that seen in some advanced cases of osteoarthritis of the spine.

*Trophic changes* do not occur as isolated symptoms in hysteria. Although blueness of the skin and brittleness of the nails may be seen in long-standing paralysis of hand or foot, these are but the concomitants of disuse. In some cases of contracture of prolonged duration, excoriation of the skin may occur in the neighbourhood of the joints.

*Hysterical dysphagia* is not uncommon, and may be associated with the sensation of *globus hystericus*. The muscles whose functions are involved in this disorder are those which take part in the first stages of the act of swallowing, and are under voluntary control.

So-called *hysterical vomiting* and *aerophagy* have been mentioned under the Tics (*see* p. 880), and are described on pp. 415 and 418.

*Hysterical polypnœa* is a condition in which there is extreme rapidity of respiration unassociated with any pulmonary disease. Although the rate may amount to sixty or seventy respirations a minute, the individual breaths are very shallow.

**Diagnosis.**—This depends, in the first place, upon the exclusion of signs of organic disease sufficient to account for the symptoms observed. There are, however, some symptoms which are in themselves of positive value in making the diagnosis, such as aphonia with isolated paralysis of the adductors of the vocal

cords, and the simultaneous movement of prime movers and antagonists in a paralysed limb or muscle.

The association of paralyses which could not be accounted for on the basis of a single lesion is also a suspicious circumstance, such, for instance, as an inability to open one eye together with an apparent paralysis of the same side of the face. The failure of a muscle to function in one movement, together with good power in the performance of another, may also be a valuable point in evidence. The circumstances under which the illness has arisen and the previous history of the patient, with special reference to her reaction to emotional difficulties, may also be useful; but the mere fact of the symptoms having immediately followed an emotional shock is not to be taken as evidence of their hysterical nature, since a cerebral hæmorrhage or an attack of genuine epilepsy may be precipitated by such conditions.

Of organic nervous diseases, that most likely to be confused with hysteria is disseminated sclerosis, but the mistake made is usually that of diagnosing the organic condition as functional.

In difficult cases, especially those in which a hysterical disability is superimposed upon an organic affection, it may be necessary to resort to psychotherapy as the final criterion. Symptoms which are removed by these methods must be of hysterical origin.

*Diagnosis of a Hysterical Fit.*—The features described above (p. 904) serve to distinguish this from an epileptic fit if the physician is able to witness an attack. Often, however, the diagnosis has to be made upon the history alone. Here points which are definitely in favour of epilepsy are the occurrence of attacks when the patient is alone, especially if he falls and hurts himself, true disturbance of consciousness, cyanosis, biting of the tongue, incontinence of urine, and a period following the attack in which the patient is dazed or behaves in an automatic manner. The epileptic often gives a groan at the commencement of the fit, but is thereafter silent; the hysteric frequently continues to cry out during the whole attack. The duration of the movements in an epileptic attack is brief as a rule not more than ten minutes; in the hysterical fit they are commonly of longer duration. The circumstances of onset have also to be considered. It must be borne in mind that an epileptic may also have hysterical fits, and that after an attack of minor epilepsy the patient may sometimes pass into a hysterical condition.

The **Prognosis** in hysteria is on the whole good as far as individual symptoms are concerned, although this depends to some extent upon early diagnosis and proper treatment. A person with hysterical paraplegia, if the condition be pronounced a grave one by the physician, may remain bedridden for years, yet even so she may recover spontaneously, the symptoms disappearing suddenly and being attributed in most cases to emotional shock. Of such a nature are the many "miraculous cures" effected by quacks. On the whole, it may be said that the longer a symptom has persisted the more difficult it is to remove; when the symptoms are of short duration they are easily caused to disappear. The underlying disorder of the mental processes, however, is by no means so easily remedied, and it is a notorious fact, therefore, that the hysterical patient is liable to relapses in which the original symptoms may be reproduced or new varieties may be displayed. In estimating the prognosis, due weight must be given to considerations of the patient's power of intellectual grasp and the environmental circumstances against which she has to contend.

**Treatment.**—Hysteria is a disorder of the mental processes, and treatment must therefore be directed towards the mind, the general title given to the methods employed being *psychotherapy*. The forms in which psychotherapy may be used in the treatment of hysteria are classified as *suggestion*, *persuasion* and *analysis*.

An outline will be given of these three methods, together with some indications

for the use of each, but it will first be of value briefly to restate the theory already enunciated of the causation of hysteria.

The condition, then, is one in which the patient is led by motives of which he is unconscious to accept the suggestion that he is suffering from bodily illness. The relative importance of these two causes, unconscious motive and suggestion, varies in different cases. When the unconscious motive is powerful, as in the case of a workman seeking compensation, the suggestion may be of trivial importance. On the other hand, a definite diagnosis of organic disease made by a medical man of standing may need no more support than a natural tendency to surrender to illness in order to produce a hysterical symptom.

Whatever the chain of mental processes which has led up to the hysterical condition, the penultimate link is the patient's conviction that his symptoms exist and are dependent upon physical causes over which he has no control. It is the aim of psychotherapy to remove this conviction, and this may be accomplished by any one of the methods already mentioned or by the use of all three in combination.

The object of all three methods is to replace the patient's conviction as to his symptoms by another and opposite belief.

The method of *suggestion* aims at achieving this end by making use of emotional or instinctive trends in the patient's mind, of which the most important are his implicit belief in the knowledge and power of the physician and his readiness to expect miraculous cures from treatment which is novel, spectacular, or mysterious. The success of this method clearly depends to a large extent upon the personality and reputation of the physician, as also upon the readiness of the patient to abandon the exercise of his critical faculties.

The technique of the method may be illustrated as follows: The physician is asked to see a woman with a paralysis of the arm. Having discovered that the paralysis is functional, he will remark to the patient that the condition is a nervous one, which is readily curable by means of a new form of electrical treatment; he will mention the fact calmly with the air of one accustomed to such cases and their cure, and may proceed to relate one or two instances which have occurred in his practice in which long-standing paralysis of this type has been completely removed by a single application of the new treatment. Having led the patient's friends and relations also to expect an immediate and certain cure, he will return later with a faradic battery and wire brush, and after further preparation of the patient's mind will apply the current to stimulate the muscle groups of the affected limb, at the same time insisting that she has recovered her powers of voluntary movement. Similar methods may be applied to the treatment of other hysterical symptoms. The actual instrument used is of no importance, provided that the method is new to the patient. If, for instance, faradism has previously been tried and has failed, some new means must be devised. The essential fact in successful treatment is the establishment of implicit confidence of patient in physician. It will be noticed that when this method is employed the conviction of cure by which the conviction of illness in the patient's mind is replaced rests not upon any reasoned chain of belief, but upon instinctive feeling.

Hypnosis may be regarded as a form of suggestion, which has been now largely abandoned in favour of more direct methods in the treatment of hysteria.

The object of *persuasion* is to induce the patient to replace the false conviction as to his illness by a reasoned belief. To take the instance of functional paralysis of the arm again, the physician will explain to the patient that there are no signs of disease of the muscles of the limb, or of the nerves controlling it; that a muscle which can be used for one movement must be equally available for another, and therefore that if she first relaxes the muscles of the affected limb, and then sets out with the single aim of performing a definite movement, she will be able to accomplish it. The advantage of this method over suggestion is that it leaves the patient with a conviction of cure which is more stable in that it depends upon a chain of reasoned arguments which she can reproduce for herself in time of need.

The disadvantages are that it makes a certain demand upon the intellectual, and especially the critical, faculties of the patient, and that it takes longer, and is perhaps for this reason less certain in its immediate effects.

In practice these two methods of suggestion and persuasion are usually combined, for it is clear that the success of the latter method must depend upon the implicit confidence of the patient in her physician as well as upon processes of cold reasoning.

The method of treatment by *analysis* has a much deeper purpose, namely, to discover the whole chain of mental processes leading up to the production of the hysterical symptoms, and to remove these by helping the patient to readjust his mental attitude in the light of the analysis. If the patient can be led to an insight into his own condition, and is able to realise for himself his state of mental dissociation, which is the nucleus of the hysterical disorder, he is not only enabled to discard his symptoms at will, but is in a much better position to guard himself against similar trouble in the future than if treatment has been directed to removal of the symptoms alone.

Such an analysis of the mental situation may be a comparatively simple matter, or it may call for much patience and skill on the part of the physician, and the use of certain technical methods (*see* p. 898). To take as a hypothetical instance again the case of a young woman with functional paralysis of the arm, the physician may discover that, having hitherto lived at home, she has lately for the first time in her life been away in domestic service, that she has found her new duties trying, has been feeling very homesick, and has not obtained the sympathy she desired either from her co-workers or her parents; further analysis of the patient's inner thoughts and feelings, as well as of her behaviour and utterances, leads to the conclusion that the unconscious motive for her illness has been the desire to escape from her unpleasant duties and to gain sympathy from those around her, the precipitating factor having been a trivial injury sustained at work. The patient is given this explanation of her malady and is encouraged to meet her emotional difficulties frankly, being told that when she does so her symptoms will disappear. It is clear that he who employs this method is at the same time availing himself of both suggestion and persuasion, which probably play a considerable part in the procedure, since appeal is made both to the patient's implicit belief in the physician and to her powers of reasoning about her own mental processes.

In the practical treatment of hysteria these three methods of suggestion, persuasion and analysis are generally used in combination, due consideration being given in every instance to the intellectual grasp of the individual, and to the time available for treatment, in deciding which shall be chiefly relied upon. There are some patients of low educational level for whom crude methods of suggestion alone are the best available. In all other cases it is advisable as a preliminary step to give the patient some kind of explanation of his illness in terms which will help him to understand the nature of the disorder and the rationale of the treatment to be adopted. After this it is well to begin with the removal or alleviation of the symptoms by means of suggestion and persuasion, which may be followed up by analysis in selected cases. Whenever possible the patient should be led to realise that his illness results from a disturbance of the mental processes processes which he can, if he will, examine and control. Since the perpetuation of hysterical symptoms frequently depends upon the patient's emotional reactions to factors in his environment, it is of value in many cases to have him removed to a nursing home or hospital before treatment is commenced, and to forbid him receiving letters or visitors until the cure is complete.

In conjunction with mental treatment carried out on these lines, the physical condition of the patient should be carefully considered, and every effort made to eliminate physical causes of bodily inefficiency, whether in the nature of septic foci or faulty habits of life.

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# DISEASES OF THE MUSCLES

## FIBROSITIS

(*Myalgia, Muscular Rheumatism*)

This name is given to a painful affection of the muscles or fasciæ which depends upon local changes of an inflammatory nature.

The pain is probably caused by swelling of the interstitial connective tissue in the muscles, their tendons and their fascial attachments, which results in compression of the nerve endings. The swelling is due to exudate either as the result of poisons (bacterial or chemical) deposited from the blood stream or of tearing of the tissues through strain or injury.

In one group of cases then the cause of fibrositis is to be found in some source of toxæmia, such as a septic focus or disorder of metabolism. In these cases exposure to cold appears to be an important factor in causing the local deposition. In the second group a history of muscular strain is to be obtained. No absolute distinction can be made between these two groups, since local injury may provide a nidus for the action of toxins.

**Symptoms.**—As a rule only one muscle or tendon is affected, and the pain at the beginning is localised to a single spot. It is experienced whenever the part involved is stretched or pulled upon either by contraction of the muscle directly affected or of its antagonists. Pressure at the site of the disease will also produce pain. If, on the other hand, the patient can be induced to relax his muscles completely, passive movements gently performed are painless, this point serving to distinguish the condition from arthritis of the neighbouring joints. The onset is usually acute, with sharp pain experienced on movement.

A common situation is in the lumbar muscles, when it is often called *lumbago*. Movements such as stooping become exceedingly painful, and walking is only accomplished with a stiff back. Often after an interval of three or four days the inflammatory changes spread downwards and involve the sheath of the sciatic nerve. If this happens, the sciatic neuritis overshadows and outlasts the original lumbago. The muscles of the shoulder girdle are often affected, especially the attachments to the humerus of deltoid and pectoralis major. In this case movements of abduction and adduction give rise to pain, and the arm is held to the side in walking, being sometimes supported at the elbow by the other hand to relieve tension in the affected muscles. Here again the inflammation may spread to the nerve sheaths of the brachial plexus, with resultant brachial neuritis.

The muscles of the neck are sometimes attacked, the condition being then commonly known as *stiff neck*. This is particularly apt to occur from exposure to cold, such as results from sitting in a draught.

More rarely one of the intercostal muscles is involved, so that breathing and coughing cause pain. The condition is then known as *pleurodynia*, and simulates pleurisy, but there is no rub, and constitutional disturbance is slight or absent.

Occasionally the muscles of the scalp are affected, when the acts of wrinkling the forehead and mastication are painful.

**Diagnosis.**—This depends upon the presence of definite muscular tenderness on deep palpation, and pain which is constantly brought on when the involved group of muscles is put upon the stretch. Here also, as in the case of neuritis, it

is most important to be on the look-out for signs of underlying disease of the nerve roots or the viscera, the pain from which is referred to a peripheral distribution, and may, especially in the case of visceral disease, give rise to muscular tenderness and rigidity. The lightning pains which occur in the early stages of tabes are very often erroneously put down to muscular rheumatism.

**Prognosis.**—The course of the illness, provided that it is properly treated and is uncomplicated by neuritis, is usually brief. When, however, proper rest cannot be obtained, the pain may persist for many weeks. Complete recovery is the rule, but recurrences are common, especially in the cases of traumatic origin.

**Treatment.**—In the earlier stages diaphoresis is of undoubted benefit, and may be prescribed in the form of a Turkish or vapour bath. Failing this, a hot bath, followed immediately by a dose of aspirin, before getting into bed between blankets with hot water bottles, will suffice, and often cuts short the attack. Once the affection has developed, rest is the essential part of treatment. If the arm be affected, it should be carried in a sling, otherwise rest on a couch is the quickest way to cure. Heat locally applied by means of the electric lamp and diathermy are undoubtedly useful. A further remedy for which good results are claimed is the injection into the muscle at the site of the pain of a solution of quinine and urea hydrochloride, such as is used in local anæsthesia.

If septic foci are considered to be the cause of the trouble, these should be eliminated. In other cases attention should be paid to the general *régime*. Over-indulgence in food and drink should be avoided. Muscular exercise should be regular but not excessive. Precautions should be taken against undue exposure to cold and wet. Of drugs a useful mixture is one containing 5 grains each of sod. sal., sod. bicarb., pot. iod., and pot. brom. In some cases, especially when fibrositis is associated with evidence of gout, atophan is useful.

## MYOSITIS

Inflammation of muscles is rare as a primary disease. General myositis, or *polymyositis*, occurs in various forms. In *dermatomyositis* the muscles of the extremities, and later those of the trunk, are swollen, œdematous, stiff, and painful on movement and pressure. The skin over the affected muscles is œdematous, and presents erythematous, erysipelatoid, or eczematous patches. There is moderate fever, and the spleen is enlarged. The cases last for a few months to two or three years, and are often fatal from implication of the muscles of deglutition and respiration, or recover with atrophy of the muscles. The muscles are found to be swollen, yellowish white in colour, soft and friable. The muscular fibres are swollen and granular, or hyaline and waxy, or contain vacuoles; and the connective tissue is infiltrated with leucocytes and œdema fluid. The cause is entirely unknown, but is probably microbic.

An allied affection is *hæmorrhagic polymyositis*, in which bleeding takes place into the inflamed muscles, petechiæ occur under the skin, and the patient has palpitation and tachycardia.

Myositis is sometimes associated with multiple neuritis (*neuromyositis*), and this may be seen in chronic alcoholism (*alcoholic myositis*). The muscles are painful and tender, and become hard and shrunken; while the skin over them may be œdematous. Under the microscope are found irregular areas of cirrhotic induration, and more acute cellular infiltration of the perimysium. A chronic myositis has also been described, affecting mostly the muscles of the head and neck, but also those of the gluteal, abdominal, lumbar, and deltoid regions. In early cases the muscles are found to be swollen, in older cases there are thickenings or indurated masses like cartilage. There may be tenderness, as well as sharp and cramp-like or aching pains. Their occurrence in the temporals, occipito-frontales, or upper parts of the sterno-mastoids or trapezius muscles is accompanied by paroxysms of severe headache, and the condition has been described as *indurative*

*headache*. Indeed, such localised indurations are said by some (C. Watson, Telling) to be of frequent occurrence, and to account for most of the pains called rheumatic as well as lumbago, stiff neck, and other forms of myalgia. They are best detected by oiling the skin, and carefully manipulating the relaxed muscles, when nodules firm in the centre, but unusually soft at the periphery and tender to pressure, will be recognised. This has been called *nodular fibro-myositis*.

Secondary myositis occurs in various infectious diseases (*infective myositis*), producing a diffuse swelling and infiltration of the muscle, or abscesses may form, as seen in pyæmia (*metastatic myositis*), septicæmia, glanders, typhoid fever, and malignant endocarditis. Trichiniasis, described below, is essentially a parasitic myositis.

*Tubercle* and *syphilis* also involve muscles. The former occurs as a metastatic deposit secondary to tubercle of the bones, glands, or viscera, or arises by direct invasion from adjacent parts. Syphilis produces a diffuse myositis in almost any stage of the disease, and in the tertiary stage the typical *gumma*. A local inflammation of the muscles sometimes occurs in the neighbourhood of the joints in rheumatic fever and gonorrhœal synovitis.

*Myositis ossificans* is a disease in which the muscles are converted into osseous tissue. It always begins in early childhood, invades first the muscles of the back, and runs a progressive course. Bony deposits arise also from injury, most commonly in the extensor cruris, and in the brachialis anticus (*traumatic myositis ossificans*), but it is doubtful whether this is truly an inflammation of muscle. A *myositis fibrosa* has also been observed.

**Treatment.**—The indications for treatment are the relief of pain, when present, by local applications, or in severe cases by morphia and other sedatives; the removal of the cause, as in alcoholic cases; and specific treatment, as in syphilis, when potassium iodide should be of use. Limited lesions in tuberculous, septic, and metastatic myositis may require incision and antiseptic methods. The indurative and nodular forms of myositis are benefited by firm massage of the nodules, and by such drugs internally as sodium salicylate, aspirin, and antipyrin.

## PARASITIC DISEASES OF MUSCLE

The muscles may be invaded by certain animal parasites. Of these the *Trichina* or *Trichinella spiralis* is the most serious, as it sets up a polymyositis, which is often fatal. *Echinococcus*, or *hydatid cysts*, and the *Cysticercus cellulosæ* are also found in muscle, but cause very little, and that only local, trouble.

### TRICHINIASIS

The disease, known also as *trichinosis*, is due to the nematode worm above named, which is found in enormous numbers in the voluntary muscles throughout the body. The disease is rare in England, but is not uncommon in Germany. The worms are parasitic in rats, and are in some way conveyed by them to pigs, being deposited by thousands in their muscles. Within forty-eight hours of the ingestion of such pork by human beings sexually mature trichinæ are found in the intestine. The females are more numerous and larger; they measure from $\frac{1}{12}$ to $\frac{1}{7}$ inch, while the males are from $\frac{1}{20}$ to $\frac{1}{14}$ inch, and differ from the females in presenting two small processes at the tail. Within seven days after ingestion embryos are formed within the eggs, and are discharged from the females already hatched. According to the view most generally accepted, the females bore their way into the villi and other parts of the mucous membrane, and the embryos are deposited in the lymph vessels or chyle vessels, by which they are carried into the blood and ultimately to the voluntary muscles. In the muscles they increase in size, and possibly move about in the course of the

muscular fibres. About the second week they reach the full size corresponding to this stage, namely, $\frac{1}{25}$ inch or a little less, and two or three weeks later they become coiled up, and, as a result of the irritation which they produce, are gradually surrounded with a capsule. This is oval, or rather fusiform with an oval bulging in the middle, and lies always parallel to the muscular fibres; it measures $\frac{1}{78}$ inch in length by $\frac{1}{300}$ inch in breadth. It is at first nucleated and transparent, but afterwards becomes calcified, especially at its ends. Calcification in the human subject probably does not take place under twelve months, and even then does not interfere with the life of the parasite within. Indeed, the parasite may remain in this condition for years, or it may perish, and be converted into a structureless mass. The muscles in which the trichinæ are deposited acquire a pale reddish-grey colour; the fibres lose their striation, and become brittle and homogeneous, with numerous minute fissures. With the exception of the heart, all the striated muscles of the body may be affected, but the capsules are most abundant in the diaphragm, the intercostal muscles, the biceps, and the muscles of the larynx and of the throat. As long as the worm remains alive in its capsule it has the power of developing into a sexually mature trichina on being taken into the stomach of a suitable host. Trichinæ are found not only in muscles, but in the connective tissue of other parts, especially the fat layers and coats of the intestine.

**Morbid Anatomy.**—The only characteristic change is the condition of the muscles. There are sometimes signs of hæmorrhagic catarrhal inflammation of the small intestine; the liver is often fatty; the spleen is not enlarged.

**Symptoms.**—These consist mainly of febrile reaction with local evidence of inflammation of the muscles. In some cases there are at first gastro-intestinal disturbances, such as epigastric pressure, nausea, vomiting, and diarrhœa, or perhaps constipation. But these are often slight, and the commencement is, like that of many febrile diseases, characterised by loss of appetite, sleeplessness, lassitude, and depression. Very soon the arms and legs become painful; the knees and elbows are either flexed or extended, but in each case any alteration of the position is extremely painful, and the patient avoids every movement. The muscles of the limbs are tender, and feel hard and swollen to the touch. The electric reactions of nerve and muscles are diminished; mastication becomes painful, and the jaws may be closed for weeks; the implication of the respiratory muscles causes shallow and interrupted breathing; and coughing, sneezing, and yawning are difficult or impossible. The inability to cough up the secretions aggravates the dyspnœa seriously. The movements of the eyeballs are painful, and the power of accommodation is said to be lost at the same time. Towards the end of the first week appears another symptom—namely, œdema. This occurs first in the eyelids; then the rest of the face and neck may be affected, and sometimes even the upper and lower extremities. Its causation is not clear. The fever is seldom very high or continuous; the temperature is generally below 102°, but may rise to 104°. The pulse is rapid; there may be profuse sweating, and a miliary eruption; and there are erythematous patches, wheals, or vesicles, as in dermatomyositis, or petechiæ and pustules. There is leucocytosis, mainly due to the very numerous eosinophils, reaching in some cases 80 per cent. of the leucocytes. The tongue is dry, red, and slightly furred; sometimes there are headache and stupor. Death may take place in the fourth or fifth week, or earlier, from exhaustion, pneumonia, or bronchitis; and if the patient recovers, convalescence is slow, and hindered by muscular pains, muscular atrophy, and persisting œdema.

**Diagnosis.**—There is a certain resemblance between trichiniasis and typhoid fever in the febrile reaction and diffused pains, but with the progress of the disease the differences become marked, especially the absence in trichiniasis of initial headache, rose spots, splenic enlargement, and the Widal reaction. Cases of trichiniasis occur in groups, since an affected animal is likely to be eaten by many

individuals or a family. A suspicion may be confirmed by an examination of the fæces for adult trichinæ, or of a portion of excised muscle for encapsuled forms.

**Treatment.** The trichinæ situate in the muscles are beyond our reach; we can only hope to destroy the parasites in the intestine. For this purpose castor oil or calomel in large doses may be given. Benzine, 1 or 2 drachms daily, in gelatine capsules; glycerine, a tablespoonful every hour or two; and picric acid, $\frac{1}{2}$ to 1 grain daily, have been recommended. The muscular pains may be treated with narcotics internally and chloroform or belladonna externally.

## NEW GROWTHS IN MUSCLE

The tumours found in muscle are *rhabdomyoma*, *fibroma*, *chondroma*, *osteoma*, *sarcoma*, *angeioma*, *lipoma*, *gumma* (*see* Myositis), and *carcinoma*. The last is frequently due to invasion from adjacent parts, as, *e.g.*, the pectoral muscles from carcinoma of the breast, the intercostal muscles from carcinoma of the lung, and the orbicularis oris from epithelioma of the lip.

## MUSCULAR ATROPHY

(*Amyotrophy*)

Atrophy of muscular tissue takes place under a variety of conditions, and has been divided into *simple atrophy* and *degenerative atrophy*. In the former the muscular fibrillæ diminish in size, while in the latter they diminish in number as well. The two conditions are not entirely distinct in their origin—that is, the same cause may in one case produce the first, and, operating for a longer time or more acutely, may bring about the second, severer form.

Simple atrophy is seen especially after acute or long illnesses, as a result of starvation, some kinds of intoxication, and locally from disuse, and from paralysis in cerebral lesions.

The different forms of arthritis, acute and chronic rheumatism, and gonococcal synovitis, are frequently accompanied by atrophy of the associated muscles; and this may be simple or degenerative. In the slightest degrees only one muscle is affected in the case of each joint; and these are the deltoid for the shoulder joint, the triceps for the elbow, the gluteus maximus for the hip, the extensor cruris for the knee, the pectoralis major for the sterno-clavicular joint, and the flexor brevis for the metacarpo-phalangeal joint of the thumb. In a higher degree of amyotrophy all the muscles connected with the joint are wasted; and in the most severe forms muscles remote from the joint may be involved, as, for instance, those of the whole arm and shoulder in arthritis of the wrist. The cause of arthritic amyotrophy is still obscure, but there are some grounds for thinking that lesions of the anterior grey cornua are determined by the arthritis, and that these cause the muscular wasting (Klippel and Weil). It would thus fall into the myelopathic group.

The degenerative variety is seen in the most pronounced form in the several lesions of the spinal cord and nerves which involve the lower neuron in one or other part, and hence bring about Wallerian degeneration in the periphery. These *myelopathic* and *neuropathic* forms have already been described (*see* Multiple Neuritis, Progressive Muscular Atrophy, Acute Poliomyelitis, etc.).

## PROGRESSIVE MUSCULAR DYSTROPHY

(*Myopathy*)

This is a condition in which there is progressive weakness and wasting of the skeletal muscles in the absence of any local or constitutional cause, and without clinical or pathological evidence of disease of the nervous system.

**Ætiology.**—The cause is unknown, but the disease probably depends upon some congenital abnormality of the muscular fibres. It commonly occurs in several members of the same family, and may be transmitted from one generation to another, usually through the females. Except in the case of the pseudo-hypertrophic form, which is commoner among males, it affects both sexes equally. The first symptoms are developed as a rule during childhood, and are always manifested before the completion of adolescence.

**Pathology.**—The changes are confined to the striated muscles, in which are found atrophy of some fibres, with vacuolation and fatty degeneration, hypertrophy of others and extensive fatty deposits in the interstitial tissue between the fibres, which also may show proliferative changes. The motor nerves and end plates are intact.

**Symptoms.** Several clinical varieties of the disease exist, and will presently be described. The features common to all types are the early age of onset and progressive weakness of certain muscle groups. With this may be associated atrophy or pseudo-hypertrophy, but the weakness precedes obvious changes of this kind, and the attention of the parents is usually attracted by clumsiness shown by the child in the performance of certain movements, such as walking or climbing upstairs, by a tendency to fall about, and by difficulty in regaining the erect posture after a fall. The child learns to compensate for the weakness by the acquisition of new methods of muscular co-ordination, and this may result in certain peculiarities of gait and posture, which are somewhat characteristic. The patient has a waddling gait, the feet are widely separated, and the body is thrown from side to side with each step; the gait is further modified by the tendency to walk on the toes, which results from the diseased calf muscles shortening and producing a modified talipes. In standing also, the legs are widely separated for the sake of equilibrium, and the back assumes the position of *lordosis*, or curvature with a deep lumbar concavity backwards. This is because the weakened glutei and extensors of the hip allow the pelvis to drop forwards, and the balance of the body is then only preserved by the shoulders being thrown back so that a line dropped from them falls even behind the sacrum. Another characteristic feature is the way in which the patient rises from the sitting position on the ground. He rolls over on his hands and knees and, if a chair or bed be near, pulls himself up by its means; but if alone, he lifts the knees from the ground so as to be on his hands and toes; then, swinging himself over towards one side, he places the opposite hand on its corresponding knee, and by its means straightens the leg. The same manœuvre is repeated with the other hand and knee, so that he now stands with legs wide apart and a hand on each knee. With a great effort, then, the back is gradually straightened as the hands are brought higher and higher up the thighs.

The weakness of the spinal muscles is also shown by the inability to pick up objects from the floor, and by the falling forward of the body if the patient is sitting and leans forward too much; further, there is great difficulty in ascending stairs, so that the help of the banisters is sought.

The electrical condition of the affected muscles is not at first much altered. When they have become very weak there is diminution to both faradic and galvanic electricity. There is no reaction of degeneration, and there is no fibrillary twitching, such as is seen in association with degeneration of the anterior horn cells. The tendon jerks are, after a time, diminished, and in advanced cases lost.

Beyond this the nervous system is normal; sensation is unaffected; the bladder is only involved occasionally towards the end; and the mental functions are mostly unimpaired, though, according to Ross, some degree of mental incapacity and even idiocy may co-exist.

After the first development of the symptoms the disease may remain stationary for two or three years, but the patient gradually gets weaker in the legs, the fibrosis leads to contracture in the muscles, the power of standing is lost, and the

patient is confined to bed. Finally, death from intercurrent illness, such as inflammation of the lungs or an acute infectious disease, takes place about puberty in the majority of cases; but in those that have developed slowly the fatal termination may be correspondingly delayed.

The following clinical types have been isolated. It must be remembered, however, that mixed or transitional varieties are also seen, and that there is a strong family resemblance between all forms.

*Pseudo-hypertrophic Type.*—This occurs chiefly in males; the symptoms begin in early childhood and are those associated with weakness of the muscles of the pelvic girdle, *i.e.* lordosis and waddling gait and difficulty in rising from the horizontal posture. The hypertrophy affects especially certain muscles, notably the calves, the deltoid, biceps brachii, infra-spinati, the erectores spini, the glutei and the vasti. The hypertrophied muscles as a rule feel hard and fibrous to the fingers, and, in spite of their great size, are very weak. The hypertrophy is quite commonly confined to a portion of the muscle in the early stages, with the consequent appearance of tumour-like masses in its substance. Fibrosis of the calf muscles frequently leads to contracture and talipes equinus at a relatively early stage (*see* Fig. 93), and the patient is then likely to become bedridden. In this type of the disease the facial muscles are not affected save for occasional pseudo-hypertrophy of the masseters.

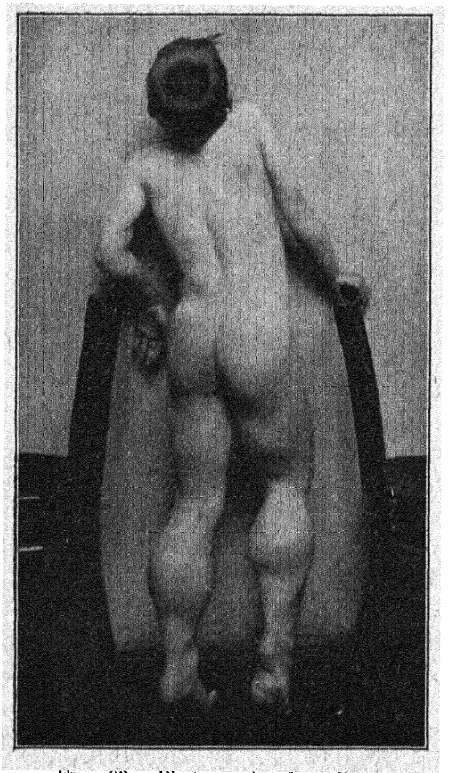

FIG. 93.—Photograph of a Boy suffering from the Pseudo-hypertrophic Type of Muscular Dystrophy.

*Scapulo-humeral Type.*—In this form, which affects both sexes equally, and begins rather later in childhood than that already described, the weakness and wasting commence in the muscles of the shoulder girdle, with consequent winging of the scapulæ and drooping of the shoulders. Owing to laxity of the muscles around the shoulder joint, the patient, if lifted by an arm placed under each axilla, is apt to slip through one's hands in a rather characteristic manner. The weakness and wasting subsequently affect the muscles of the pelvic girdle, giving rise to the anomalies of gait and posture already described. There is no hypertrophy in this type.

*Facio-scapulo-humeral Type* (*Landouzy-Dejerine*).—This closely resembles the preceding type except for the affection of the facial muscles, which is a constant and, as a rule, an early feature. The muscles chiefly affected are the orbiculares palpebrarum and oris. When the patient is instructed to let his eyes close there remains a gap between the lids, which gives him a curious, almost cadaveric expression. The weakness of the lips gives rise to difficulty in performing such movements as sucking, pouting or whistling, whilst the features are characteristically lacking in emotional expression.

The progress of the disease in this form is often very slow, and the weakness

may be for some time limited to the facial musculature. The shoulder girdle is involved sooner or later, the pelvic girdle only in the last stages.

*Distal Type.* This somewhat resembles the peroneal form of muscular atrophy (*see* p. 798) in that the wasting begins in the small muscles of the feet and hands, and affects progressively the muscular groups nearer the trunk. Clinically it may be distinguished from the peroneal form of atrophy by the involvement of the facial musculature, together with the absence of fibrillary twitchings in the affected muscles, and pathologically by the complete absence of the changes in the spinal cord which are characteristic of the other.

**Diagnosis.**—The familial and hereditary incidence, early age of onset, and slowly progressive course render the diagnosis a comparatively simple matter. The absence of fibrillary tremors is a valuable point in distinguishing the myopathies from the atrophy due to anterior horn cell degeneration. The sudden onset and stationary nature of the paralysis in anterior poliomyelitis serve to differentiate this illness.

**Prognosis.**—In those cases which begin early and advance rapidly the prognosis is proportionately more serious. In those in which the facial muscles are first affected the progress of the disease is slow. In the pseudo-hypertrophic form the patient is likely to become bedridden at an earlier stage from the development of contractures. Yet there is a certain number of patients in whom the progress is so slow that they are able to live independent lives, marry and support their children. Recovery never occurs.

**Treatment** can at best retard the progress of the disease a few months or years. Drugs probably have no influence, and electricity little, if any. Carefully planned gymnastic exercises have done good, and rubbing, massage, and passive movements may be of some use. When the gastrocnemii are so shortened as to prevent the patient standing, the tendons should be divided.

## THOMSEN'S DISEASE

(*Congenital Myotonia*)

This disease consists of a peculiar rigidity of the muscles, which comes on whenever they are called into contraction by voluntary impulses after a period of rest. Thus, if the patient wishes to walk and tries to rise from his seat, his muscles become rigid, and he is unable to move; the rigidity lasts a few seconds, and then relaxes so that he can at length get up. His first few steps are attended with the same difficulty, but soon the contractions become more natural, and shortly the trouble ceases altogether, so that he walks with complete freedom and ease. If, however, he should stop for a minute, the muscles become rigid on his beginning to walk again. Quick movements are thus impossible immediately after rest, and sometimes accidents occur, as, for instance, when the patient is descending from a train, and having placed one foot on the ground, he is unable to bring the other out quickly after it, and falls in consequence. Involvement of the muscles of the arms may give rise to a considerable degree of disability in manual workers. After grasping an object firmly, especially if this action is prolonged, the patient is unable to relax his grip sometimes for several minutes. The tendency to rigidity seems to be increased by cold and emotional stress. On inspection there is as a rule no abnormality of the muscles affected, nor is their power diminished. Their excitability to direct mechanical stimulation, however, is altered. When the belly of the muscle is struck with a percussion hammer the resultant contraction, instead of disappearing at once, persists for several seconds. This so-called " myotonic reaction " cannot always be obtained in all the muscles involved, and the ease with which it can be elicited varies from time to time. The closure contraction to strong galvanic currents is much prolonged beyond the time of application, and with continued application of the current a series

of contractions has been seen to pass in a wave-like manner from the kathode to the anode. The reactions to faradism are often normal, but may be exaggerated in force and duration. The disease often lasts a very long time, but it may subside.

**Ætiology.**—It happens often early in life, may affect more than one member of a family, and is commonly regarded as being congenital.

**Pathology.**—In a case reported by Dejerine and Sottas, the patient died of nephritis. The muscles were large, protruding, tending to be more globular in shape, of a duller red colour, and less elastic than normally. The number and size of the muscle nuclei were found to be increased; the fibres varied much in size, some being very large indeed, as if swollen. Sometimes the sarcous elements were separated, and the sheath was filled with granules; or the muscle substance was degenerated and vacuolated. The connective tissue was not hypertrophied or fatty, as in pseudo-hypertrophic paralysis. These results agree closely with what had been seen in portions of muscle excised during life. The muscle nerves, nerve trunks, spinal cord, and bulb were healthy

**Treatment.**—Nothing has been found of use. Thomsen, who suffered from it himself, advised a life of continued activity.

## MYOTONIA ATROPHICA

In this disorder stiffness of the muscles similar to that of Thomsen's disease is associated with muscular atrophy. The stiffness, or myotonia, is especially noticed in the inability to relax the flexor muscles after grasping strongly with the hand. The muscles affected by atrophy are the orbicularis oris and orbicularis palpebrarum (so that there is some resemblance to the Landouzy-Dejerine type of muscular dystrophy), the masseters, temporals, sterno-mastoids (severely), the muscles of the forearm, the vasti femoris, and the anterior tibial and peroneal muscles.

The tendon jerks are frequently absent, although the muscular atrophy is insufficient to account for this symptom.

Outside the nervous system there are other symptoms of a degenerative nature, the most striking being premature baldness, general wasting and loss of weight, and in males atrophy of the testicles with impaired sexual desire. The disease generally begins in middle life and is slowly progressive. It occurs frequently in several members of the same family, as a rule affecting only one generation. It is often associated with cataract, and with a history of cataract in the forebears. Pathologically no constant changes have been found in the nervous system. The muscles show degenerative changes.

## AMYOTONIA CONGENITA

(*Myatonia Congenita*)

In this disease the muscles are extremely flaccid and wanting in tone, and the child lies about in any position and cannot sit up. The condition is noticed at or shortly after birth. Later the child is unable to walk; all its actions are feeble, and passive movements at the joints are unusually free. The response of the muscles to faradic excitation is diminished, and the deep reflexes are absent. In many cases there is slow and progressive amelioration, but the majority die of intercurrent affections during childhood. The disease is differentiated from the myopathies by the absence of marked muscular wasting, the tendency to improvement, and absence of familial distribution. The pathology is obscure, but there is some evidence in favour of a congenital arrest of development of the lower motor neurones. Massage, faradism and exercises assist improvement.

## FAMILY PERIODIC PARALYSIS

In this curious complaint, the patient suffers from attacks of gradually developing paralysis of the muscles of the trunk and limbs, which lasts on each occasion several hours and then completely passes off.

**Ætiology.**- This disease has also been observed in several members of a family, and is transmitted to the descendants of the sufferers. It affects the two sexes equally, and the first attack has generally occurred between the ages of six and twenty-four.

**Symptoms.**—The paralysis often begins at night. The muscles of the legs, then of the arms, and lastly those of the trunk and neck, gradually lose power, so that in the course of four or five hours the patient is quite unable to move his limbs. The weakness affects first the proximal parts of the limbs, and the distal parts later. The intercostal muscles are weakened, so that the breathing is shallow and feeble; and probably also the diaphragm is involved. The muscles supplied by cranial nerves are generally spared, and the sphincters remain unaffected. The heart may become dilated and the pulse rapid and irregular. The reflexes are gradually lost; and the reactions to both faradic and galvanic currents, which get less and less as the weakness increases, are entirely lost with complete paralysis. Sensation and the mental state are unimpaired. After a few hours improvement begins; the muscles regain power in the reverse order of their loss of it, and after another six to twelve hours power is completely restored as well as the reflexes and electrical reactions.

The attacks at first occur at intervals of months, but they become more frequent until they may occur weekly or oftener; as middle age is reached they again become less frequent.

The **Pathology** is at present obscure. The probability seems to be that some toxin is operating upon the muscular fibres; and some interesting observations on the relations of the urine and of creatinin excretion to the attacks have been made.

**Treatment.**—Diuresis should be encouraged by salines and mineral waters in order to get rid of toxins.

## MYASTHENIA GRAVIS

This is another disease profoundly affecting the action of the muscles; and in the absence of any constant change in the nerve centres or nerves, while lesions are frequently found in the muscles, it must be regarded for the present as a primary disorder of those structures, probably toxic in origin.

**Ætiology.**- It is slightly more frequent in women than men (112 to 108, Starr) and begins most often between the ages of twenty and thirty, but no age is exempt.

**Morbid Anatomy.**—Several cases have now been examined *post mortem*. In more than a quarter the thymus has been persistent or enlarged, or the subject of lymphosarcoma; but in others it has been absent in accordance with normal conditions. In nearly all cases there are found collections of lymphocytes (lymphorrhages) in the muscles, and in some organs such as the thymus, liver, pancreas, kidney and adrenals; the blood and lymph glands are healthy.

**Symptoms.**- The characteristic feature of the disease is weakness of the voluntary muscles, which are very rapidly exhausted by exertion, but recover their power after rest. In severe cases the weakness persists, and death often results either suddenly or with dyspnœa from respiratory paralysis. The muscles most frequently and generally first involved are those of the eyes, head, and neck, so that the patient has ptosis, diplopia, immobility of the face, difficulty in mastication or in swallowing, defective articulation, nasal quality of the voice with weakness increasing up to aphonia, and inability to support the head upright.

But nearly all the muscles in the body may be affected, and in 12 per cent. of the cases it has commenced in the muscles of the limbs; the patient may be unable to sit up, can only walk a few yards without stopping, or his respiration is impeded, and he has dangerous attacks of dyspnœa. In the limbs the proximal muscles are attacked more often than the distal; women find their arms tire when doing their hair. The condition is very variable in its intensity, and is aggravated by emotion, by cold, and by the menstrual function in women. The knee jerk is generally active, but may be exhausted by repeated stimulation, sensory symptoms are seldom present, and the sphincters are not affected.

It has recently been shown that the sugar tolerance is low in cases of this disease (**5**). The affected muscles mostly, but not in every case, react in a special manner to electrical currents –the *myasthenic reaction*. If the faradic current is applied to the muscles, they contract normally, but if it is continued, they soon become exhausted and fail to contract any further. If then the electrodes are removed, the muscle recovers, and then contracts well to the current, again becoming soon exhausted. Contraction to the galvanic current is persistent, and is scarcely at all affected by the length of application.

Temporary improvement may take place, and long remissions have been recorded with relative freedom from the symptoms, but a fatal result generally ensues from respiratory failure or from choking. Forty-five per cent. of the cases collected by Starr died within six months, but many have lived for ten years or more.

**Diagnosis.**—It is likely to be mistaken for hysteria, diphtherial paralysis, and bulbar paralysis. From the latter it may be distinguished by the absence of atrophy in the muscles, by the myasthenic reaction and the varying degrees of weakness.

**Treatment.**—Little can be done for it; the patient should be kept warm and at rest, and should be carefully fed and dieted.

Strychnine appears to improve power and prolong life in some cases (**6**). Large doses must be given. Hypodermic injections should commence at gr. $\frac{1}{32}$ of strych. sulph., and may be increased to gr. $\frac{1}{4}$ twice daily. If this has a good effect, it may be continued indefinitely, or equivalent amounts may be given by the mouth. Many other remedies have been tried, including various forms of organic extracts, but without obvious benefit.

Respiratory embarrassment should be a signal for absolute rest. A temporary fatigue paralysis of the respiratory muscles may sometimes be overcome by artificial respiration.

## SOME OTHER DISORDERS OF MUSCLE

In addition to the spasm and paralysis, which are the result of central and peripheral nervous disorders, and which have been described previously (*see* p. 763), some disorders may be mentioned of which it is perhaps difficult to say whether they are due primarily to failure of nerve or of muscle, or whether they are due sometimes to one and sometimes to the other.

**Muscular Exhaustion or Fatigue.**—The gradually lessening capacity of a muscle to contract as the result of excessive use is generally regarded as being due to the saturation of the muscle with the chemical products of its work, such as sarcolactic acid, extractives, and possibly a special toxin, though it has been also stated that fatigue in the ordinary way is much more largely due to the effect of the poisons upon the nervous centres than to their influence upon the muscles themselves. The actual consumption of the muscular fibre itself in the process cannot be entirely disregarded.

The loss of muscular power after exhausting illnesses and febrile attacks and in advancing years is probably referable to the nervous system as much as to the muscular apparatus.

The conditions found *post mortem* in myasthenia gravis suggest that in this disease the muscles are subject to some toxic influence.

**Fibrillary Contractions** (*Fibrillary Tremors*).—These have been mentioned among the symptoms of nervous diseases (*see* p. 731). But they occur in conditions of practically normal health. The most common form is a twitching of the orbicularis palpebrarum muscle, causing a slight flickering movement of the eyelid, which is felt by the patient, though it may escape the notice of a bystander. It is sufficiently common to have received the name "live blood" from the public. The same fibrillary contractions occur sometimes in other muscles, such as the deltoid or the biceps. They may be troublesome for an hour or two, or for a few days, and then pass away entirely. Their occurrence is not readily explained, whether they happen in apparent health or in connection with muscular atrophy.

In the latter they are common in the atrophies due to degeneration of anterior horn cells, such as progressive muscular atrophy, and may occur in disease of the peripheral nerves. Of course they cannot take place when the wasting is complete, but only while the degeneration is progressing. If they are dependent directly upon the changes in the anterior cornua of the cord, it must be supposed that the degenerating cell body sends erratic stimuli to the separate muscular fibrils. Such stimuli are attributed to hyperexcitability, though it is not clear why a degenerating cell should be over-excitable. When occurring in the healthy person, the explanation is even less satisfactory, the little trouble passes away entirely, and nothing remains to show a permanent change in nerve or muscle. There appears to be nothing to show whether it is caused by a temporary or functional change in the nerve-cell or by a primary functional disorder of the muscular fibril.

**Fidgets.**—This appears to be a small matter, but it is of interest in connection with this subject. The condition of physical restlessness, to which the name is popularly given, is often brought on by excessive exertion and consists of a sense of discomfort in the limbs, mostly the lower extremities, which is followed by a contraction of a whole muscle, sudden and sometimes briefly clonic, by which a temporary relief of the discomfort is obtained. The contraction is involuntary and very difficult or impossible to control, and it has the effect of moving the limb to a small extent. The sensation appears to be in the muscle; at any rate, it is deep-seated, and it is not a pain, only the sense of a desire to move. If the contraction is reflex, as it probably is, it may still be that the sensory fibres in the muscle are poisoned by the chemical products of muscular action.

When the "fidgets" occur in these circumstances—but they are not limited to them—they can be generally efficiently treated by massage of the lower extremities, by which it may be supposed the removal of the poison products will be facilitated. A change of position, as, for instance, from recumbent to sitting, will sometimes suffice.

**Cramp.**—This is apparently an involuntary, slow, forcible, prolonged and painful contraction of a muscle, not preceded by any sensation whatever, and only slowly giving way to complete relaxation. It may be repeated at once, or at an interval of some minutes; it may occur only rarely and at long intervals.

This is also a frequent result of over-exertion, and occurs often in bed after a long day's walk, or other continuous effort. Though often spontaneous, it may be induced by a voluntary effort in the muscle affected.

Its exact causation is as difficult to state as is that of the previously described muscular disorders. In the fatigue cramps the influence of muscle poisons is naturally suggested; and this is supported by the well-known occurrence of cramps in certain diseases, such as cholera, choleraic diarrhœa from food-poisoning, and some other exhausting diseases. Poisons are certainly concerned in the cramps of tetanus, of strychnia, of tetany, and of alcoholic or other forms of toxic neuritis. In the first two of these the muscles apparently receive their

stimuli from the poisoned spinal cord; in tetany the existence of Chvostek's sign shows that irritation of the peripheral nerves will produce the spasms (*see* p. 586); but in multiple neuritis it is perhaps not so obvious that the muscles are not set in action by the poison circulating in their own substances as by the change in the nerve trunks. The same may be said for gout and Bright's disease, in which cramps are not infrequent. The spasms of hysteria and the so-called professional cramps (*see* p. 833) are, on the other hand, more clearly central in origin.

The treatment of cramps is not very satisfactory. Where a toxic action can be recognised, the removal or neutralisation of the toxin is obviously the object to be aimed at. When after fatigue or otherwise a single muscle is the subject of cramp, the antagonistic muscle should be at once put into action, so as to stretch the affected muscle, when the spasms will generally give way promptly. For frequently repeated cramps massage may be usefully employed.

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# DISEASES OF THE SKIN (DERMATOSES)

It is not possible in a general text-book of medicine to present a complete review of the diseases of the skin, and in the following section much of importance has been omitted. In the present edition, however, a new classification has been adopted, based largely on that devised by Darier in his incomparable *Précis de Dermatologie*, and it is hoped that this will provide the student with a clearer conception of the ætiology and morphology of cutaneous disorders. Dermatology has unfortunately in the past been regarded as a subject apart, and has been both neglected by the general physician, and misunderstood by professed dermatologists. In reality it is clinically one of the most important of the special branches of medicine, since the skin is the mirror in which the state of the general health can be read, and a knowledge of the ætiology of its commoner disorders provides the key to the proper understanding of the pathology of disease. Of recent years dermatology has become more and more a part of general medicine, and it is as essential nowadays for the physician to acquaint himself with modern dermatological teaching as it is for the dermatologist to keep abreast with the advances made in medical pathology.

The skin is liable to the same pathological conditions as other organs and structures in the body, and the classification of its diseases is based upon these.

Thus there are changes in vascularity, inflammations, new growths, structural changes due to the presence of micro-organisms, such as those of tubercle or leprosy, lesions resulting from the circulation of disease-toxins, such as those of scarlatina, syphilis, small-pox, and other exanthemata, the invasion of animal and vegetable parasites other than bacteria, hypertrophy and degeneration of the separate structures of the skin, and changes of pigment.

We have to consider also the disorders of certain organs contained in the skin, namely, the hair, the sebaceous glands, and the sweat-glands or coil-glands.

Many disorders of the skin have been already described, as, for instance, the eruptions characteristic of the exanthems, the cutaneous lesions of syphilis, lepra, glanders, actinomycosis, and elephantiasis, and the forms of hæmorrhage known as purpura.

The discrimination of the various diseases is based upon a careful observation of the local changes, or lesions, which take place in the skin, combined with a consideration of their distribution, duration, and associated and antecedent circumstances.

The large majority of the complaints called skin-diseases are inflammatory in origin. They are due to mechanical or chemical irritants, to toxins of different kinds circulating in the blood, to drugs taken internally, and to local infection by micro-organisms. Altered conditions of the special structures of the skin, such as the hair, hair-follicles, and sebaceous and sweat glands, cause inflammation of the adjacent skin; and the animal parasites, which infest man, also bring about secondary inflammatory lesions, which are distinctive of their presence.

Such lesions show the greatest possible variety in their distribution, their duration and the extent to which the different layers of the skin are involved,

whether chiefly the epidermis, the cutis, the subcutaneous tissue or the sebaceous glands and the sweat-glands.

A number of these diseases long ago received specific names derived from their appearance, but not really connoting the fact of inflammation. Indeed, this was perhaps hardly recognised, from the fact that of the four criteria of inflammation taught by the older physicians, *rubor*, *calor*, *tumor*, *dolor*, only the first was prominently present. The cause of some of these diseases is definitely a toxin circulating in the blood, others are clearly due to micro-organisms acting locally, and in others again neither of the conditions can be certainly recognised.

## PRIMARY LESIONS

**Hyperæmia.**—This consists of redness, of varying extent, due to the blood vessels being distended with blood. It may be—(1) an *active hyperæmia* from vasomotor action; (2) an *early inflammatory hyperæmia*, with slight swelling, tenderness, and some indications of pyrexia; or (3) a *venous hyperæmia*, or passive congestion, with a more blue or livid colour than in the other forms. In all cases the redness disappears on pressure, to return when the pressure is removed—quickly in the first two cases, slowly in the last.

**Hæmorrhages.**—Cutaneous hæmorrhages form larger or smaller spots of bright red, dark red, or purple colour: they do not disappear on pressure. As a rule, the blood is gradually absorbed, and the colour fades into brown or brownish-yellow, or becomes successively brown, green, and yellow, in the larger subcutaneous hæmorrhages. A yellowish-brown stain may be left for a long time. The smaller spots are called *petechiæ*, the larger *ecchymoses*: if they form streaks they are called *vibices*. In *capillary ecchymosis* a very fine mottling is produced, which looks like a hyperæmia, until it is found to persist under pressure. Exceptionally the skin involved in hæmorrhage sloughs and leaves an ulcer.

**Papules** or **Pimples.**—Small red or pink elevations of the skin, solid, or at least not visibly containing fluid. They arise mostly in the cutis, but may be imitated by accumulations of epidermic scales. They may be pointed (acuminate), obtuse, or flat (plane).

**Vesicles** or **Vesiculæ.**—Small blisters, from 1 to 5 mm. in diameter, due to the accumulation of clear fluid under the upper layer of the epidermis. They are frequently inflammatory, seated upon an inflamed base, and containing a yellow albuminous serum.

**Blebs** or **Bullæ.**—Large vesicles, from 5 mm. to an inch or more in diameter. The fluid contents are clear, or slightly turbid, or bloodstained. They are often situate on an inflamed base: they heal by discharge of the contents, and the drying and shedding of the epidermic scale.

**Pustules** or **Pustulæ.**—Vesicles or bullæ containing pus.

**Scabs** or **Crusts.**—Irregular flat masses of dried serum, pus, or blood, or a mixture of these materials, forming upon and adherent to the raw surface which has secreted them, and frequently the result of a vesicle, pustule, or bulla.

**Wheals** or **Pomphi.**—A circumscribed œdema of the corium, producing a pale pink or white elevation of the skin.

**Scales** or **Squamæ.**—Collections of epidermic cells in the form of flakes. Sometimes, as in seborrhœa, there is a large admixture of the fatty matter of sebum. Scales vary from the small branny particles of measles (furfuraceous) to the large exfoliations seen in exfoliative dermatitis and in some cases of scarlatina, or the thick adherent masses of psoriasis.

**Scratch Marks.**—Linear lesions of the skin, from a third to two inches in length, produced by the nails, and bearing small crusts of blood. Ultimately, if deep enough, they become linear or fusiform cicatrices. Their direction generally bears a definite relation to the position of one or other hand, and parts of the body which the hands cannot reach are exempt from them.

**Excoriation.**—A patch of skin deprived of the upper layer of the epidermis, and exposing the stratum mucosum. It is of a vivid red colour, and tender to the touch; and it secretes a small amount of serum, which may dry into a crust.

**Chaps, Rhagades,** or **Rimæ.**—Cracks or fissures through the epidermis, reaching the stratum mucosum or corium beneath, very sore and apt to bleed.

**Sore** or **Ulcer.**—A loss of substance involving the whole epidermis and part or the whole of the corium. The base is covered with granulations, and secretes pus. It heals by scarring.

**Scar** or **Cicatrix.**—The new growth of connective tissue, which results from the healing of sores, involving the papillary layer and deeper corium. When recent, they are pink or bluish in colour. Finally they become dead white, and contract in size.

**Nodules.**—Solid elevations larger than papules. They have been called tubercles—a name which is now best limited to the specific lesion which is the first change of phthisis.

**Macules.**—Small areas of discoloration of the skin, which are unaccompanied by alteration in its surface or consistence. They may arise from a preceding hyperæmia, and then disappear shortly. They are generally more permanent if arising independently.

## GROUP I

# DISEASES CAUSED BY EXTERNAL INFECTION OF THE SKIN AND ITS APPENDAGES

In the great majority of diseases of the skin, in which the pathological changes are due to the active growth in it of micro-organisms, infection takes place from without; it must be remembered, however, that even the healthiest and cleanest skin is never sterile, but harbours on its surface, and particularly at the mouths of the pilo-sebaceous follicles, at the muco-cutaneous junctions, and in its natural folds, a large number and variety of organisms. Staphylococci, particularly a variety of *staphylococcus albus* (sometimes called the *S. cutis communis*), may be looked on as normal habitants of the skin, and two other organisms are so frequently present as to be, perhaps, justifiably considered as such, namely, the microbacillus of acne and the so-called "bottle-bacillus," which is probably a species of yeast. It cannot, however, be too strongly emphasised that the healthy skin must not be regarded as a hot-bed of microbial growth; on the contrary it is remarkably amicrobial. Microscopical sections through absolutely normal skin show that organisms occur *as isolated units* on *the surface* of the horny layer, never penetrating even its superficial layers; they are also seen lying at the mouths of the pilo-sebaceous follicles, but never in the sweat-ducts. As Sabouraud has emphasised, a distinction must be drawn between the mere presence of micro-organisms on the surface, and their occurrence *in a state of active growth;* except in certain moist folds, such as between the toes and in the axillæ, where organisms are normally in a state of vigorous multiplication (without, however, in health producing any effect beyond decomposing the secretions), there is no active microbial growth on healthy skin, whereas in the most minute pustule or other infective lesion organisms are present in enormous numbers. Moreover, in seborrhœa and comedo the sebaceous follicles contain myriads of acne bacilli, and in the scales of pityriasis simplex the "bottle-bacillus" can be seen in profusion.

The horny layer forms a natural protection against microbial invasion of the skin. When this is damaged, however slightly, an opportunity for such invasion occurs, and any micro-organisms present, either normally, *e.g.*, staphylococci, or accidentally, *e.g.*, streptococci, are liable to take on active growth and produce pathological effects. The pilo-sebaceous orifices may be looked on as weak points in the horny barrier, and the staphylococci that lurk there are likely to set up a folliculitis when the skin is damaged by an irritant.

It will thus be understood that diseases of the skin, caused by external infection, may arise from (1) active growth and invasion of organisms, already present in an inactive state, owing to damage to the skin or some other factor which lowers its resistance, *e.g.*, many forms of staphylococcal, and, to a less extent, streptococcal infections; (2) direct or indirect contagion with virulent organisms from an infected person or animal, *e.g.*, impetigo contagiosa, ringworm, syphilis, warts and lupus verrucosus; (3) invasion of the skin from infected mucous membranes, *e.g.*, impetigo from rhinitis or otorrhœa, sycosis of the upper lip from rhinitis or from spread, *vâ* the nasal duct, of a staphylococcal blepharitis. It is, moreover, obvious that once a focus of virulent bacterial growth has been established, the risk of invasion of other parts of the skin is greatly increased.

Rarely the skin is infected with living organisms *vâ* the blood stream, *e.g.*, in pyæmia, in certain cases of lupus vulgaris, in the secondary stage of syphilis, and possibly in some cases of furunculosis.

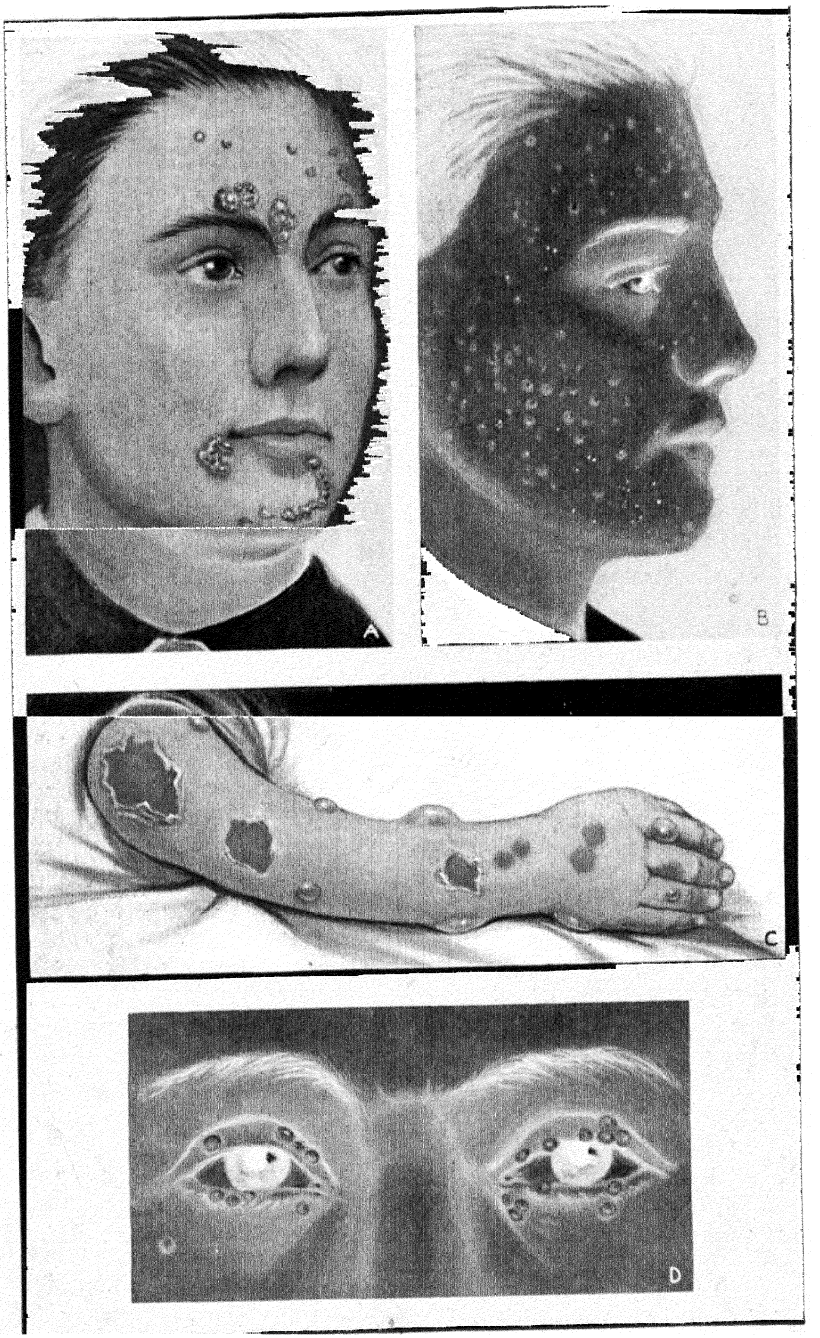
By far the commonest organisms to infect the skin are the pyogenic cocci, the acne bacillus, and the "bottle-bacillus," or spore of Malassez, and the conditions caused by these organisms will be dealt with first. With regard to the pyogenic cocci certain differences between staphylococcal and streptococcal infections may be tabulated as follows:

| *Staphylococci.* | *Streptococci* |
|---|---|
| (1) Some strains, at any rate, may be considered as natural habitants of the skin. | (1) Are more likely to invade the skin either from some previous host, or from mucous membranes. |
| (2) Tend to involve the pilosebaceous follicles, so that most staphylococcal eruptions are primarily follicular. | (2) Have no predilection for the follicles, but tend to involve the natural folds of the skin and to form fissures. |
| (3) Do not, as a rule, cause lymphangitis or adenitis. | (3) Are prone to invade the lymphatics and cause adenitis. |
| (4) Are powerfully chemiotactic for polymorphonuclear leucocytes, thus producing "laudable pus." | (4) Are less pyogenic, and tend to provoke an exudation of serum containing only a few cells. |
| (5) Staphylococcal lesions tend to remain *pure*, *i.e.*, do not become secondarily infected with other organisms. | (5) Streptococcal lesions always become secondarily infected with staphylococci, which may then form primary follicular lesions - *e.g.*, a staphylococcal sycosis may follow a streptococcal impetigo. |

## STREPTOCOCCAL INFECTIONS

Impetigo is the term given to a superficial infection of the skin with pyogenic organisms, and is characterised by the formation of pustules or bullæ, appearing rapidly on healthy skin, the contents of which dry up to form crusts under which healing takes place. Instead of developing on healthy skin, infection with the same micro-organisms may complicate some pre-existing pathological lesions, particularly eczema, resulting in pus formation and subsequent crusting. Under these circumstances the lesions are said to have become secondarily *impetiginised*.

Of primary impetigo there are two distinct varieties: (1) *Impetigo contagiosa*




A. IMPETIGO CONTAGIOSA.—Note the early vesicular lesions, the honey-coloured crusts, the surrounding inflammatory halo, the streptococcal fissures at the angles of the mouth, with the V-shaped patch spreading out from the right one (*la perlèche*).

B. ACNE VULGARIS.—Note the comedones and the follicular papulo-pustules.

C. PEMPHIGUS NEONATORUM. Note the large bullæ, contrasting with the smaller vesicles of ordinary impetigo contagiosa, the raw surfaces exposed by rupture of the bullæ, and the shrivelled remains of the horny layer at the periphery of the denuded areas.

D. MOLLUSCUM CONTAGIOSUM.—Note the pearly tumours with their central depressions, through which the cheesy contents may be extruded.

[*To face p.* 927.

(Tilbury Fox), due to infection with the streptococcus pyogenes longus; (2) *impetigo of Bockhart*, due to staphylococcal infection.

### IMPETIGO CONTAGIOSA (*See Plate XLV., A*)

In this variety infection of the skin with the streptococcus takes place just beneath the horny layer. The primary lesion is a minute red spot, which very rapidly becomes a superficial vesicle or bulla, owing to the exudation of fluid, and the vesicles or bullæ may be considered as the characteristic lesions of the disease. Where the horny layer is thin, as on the face, they are flaccid, but in other parts, particularly on the palms and soles, they may be tense. In the early stage the contained fluid is clear serum, and from it the streptococcus may often be grown in pure culture, but secondary infection with staphylococci soon takes place, and the fluid then becomes cloudy from the presence of leucocytes; in this stage the vesicles are surrounded by a red halo of hyperæmia. The fluid may escape owing to rupture of the roof of the vesicle, or may dry up spontaneously; in any case the dried serum forms characteristic crusts, which are yellow, or amber coloured, or sometimes brown from admixture with blood or dirt. After a few days the crusts fall, leaving a pink stain to mark the site of the original lesion. Should a crust be removed before healing has taken place beneath it, a raw, oozing, and sometimes bleeding surface is exposed, which soon becomes encrusted again. Healing takes place by complete regrowth of normal epithelium and no scar is left. The subjective symptoms are usually slight, but there may be some itching or burning, and in children fresh lesions are produced by scratching. Various types of impetigo contagiosa have been described.

In *impetigo circinata* there is comparatively little exudation, and the lesions tend to spread peripherally while healing in the centre, thus forming circinate patches with crusted margins; the condition is often mistaken for circinate ringworm. By confluence of the patches extensive gyrate figures may result (*I. gyrata*).

*Impetigo bullosa*. In this variety the lesions form large bullæ, the roofs of which are flaccid, except where the horny layer is thick, as on the palms and soles. It is rarely seen in this country, except in young children. The so-called pemphigus neonatorum (*see* Plate XLV., C) and dermatitis exfoliativa neonatorum (Ritter's disease), in which the horny layer may be stripped up and shed over large areas, are now recognised as being severe forms of bullous impetigo. A considerable percentage of cases prove fatal. In the tropics the bullous variety may occur in adults (*Pemphigus contagiosus tropicus* of Sir P. Manson), particularly in hot damp weather; it usually affects chiefly the flexures such as the axillæ and groins.

**Ætiology.** The cause of impetigo contagiosa is a streptococcus pyogenes longus. The chief predisposing factors are trauma of all kinds, whereby the horny layer of the skin is damaged, and exposure to infection with virulent streptococci. As its name implies, it is contagious, and is apt to be communicated from one member of a family to another by indirect and direct contact, and also occurs in epidemic form in schools and other communities. The so-called *scrum-pox* is impetigo contagiosa spread by contact on the football field. Apart from contagion, it may arise spontaneously, and is apt to complicate itching conditions, such as scabies, and particularly pediculosis capitis. Impetigo of the scalp is most frequently due to the latter cause. It may begin around the nostrils, or on the upper lip, by infection of the skin with a streptococcus derived from an acute rhinitis or purulent nasal discharge, and around the ear from the discharge of a chronic otitis media. It frequently arises from fissures at the angle of the mouth (*la perlèche*). This is common in children suffering from adenoids, who sleep with open mouths and dribble saliva, which is often infected with virulent streptococci, so that the skin at the labial commissures is macerated.

In pemphigus neonatorum inoculation of the skin with a streptococcus may occur apparently during the transit of the child through the maternal passages, but more commonly it may be traced to active streptococcal lesions on the skin of the mother, nurse, or medical attendant.

**Morbid Anatomy.**—Microscopical section through a vesicle shows that this is situated at the level of the stratum granulosum, so that the roof is formed by the stratum corneum and the base by the upper part of the prickle-cell layer. The fluid consists of serum containing leucocytes; chains of streptococci may be seen along the floor of the vesicle, and groups of staphylococci near the roof and sides. The epidermis beneath is œdematous, and leucocytes may be seen in the intra-epithelial lymph channels. The capillaries in the papillæ are dilated and there is slight inflammatory infiltration.

**Prognosis.**—If suitably treated the disease is usually cured in from one to three weeks. In debilitated and neglected children the inflammation may spread more deeply, destroying the epidermis and producing ecthymatous ulceration. Bullous impetigo of the new-born is fatal in a considerable percentage of cases, probably because little or no immunity against virulent infective organisms exists in early life.

**Treatment.**—Crusts should be first removed with soap and water, with a 2 per cent. solution of lysol, or in extensive cases by applying a boric-starch poultice, or fomentations of a 1 in 4,000 solution of mercuric chloride. A weak mercurial ointment or paste should then be applied twice daily, *e.g.* ℞ Hydrarg. ammoniati gr. x, ung. paraffini ad ℥i, or ℞ Hydrarg. oxidi flavi gr. x, Past. zinci comp. B.P.C., ad ℥i. Persistent fissures at the angles of the mouth, nose, or ears should be painted with a 2 per cent. solution of silver nitrate in spirit. ætheris nitrosi. In cases of impetigo of the beard region in men the shaving brush should be kept constantly in a 1 per cent. solution of lysol.

The French *eau d'Alibour*, which consists of zinc sulphate 7 parts, copper sulphate 2 parts, safran 0·40 parts, and boiled water, saturated with camphor and filtered, 300 parts, is a valuable alternative application in impetigo. It should be diluted with from three to six parts of hot water, and swabbed on the infected areas several times daily with cotton wool, after which a mercurial ointment or paste may be supplied.

Pemphigus neonatorum should be treated with a daily boric bath, sponging with diluted *eau d'Alibour*, copious powdering of the healthy parts of the skin with boro-talc powder, and the application to the affected regions of a boric or very weak mercurial ointment. The use of the latter over large areas is inadvisable owing to the risk of mercurial intoxication by absorption from the denuded skin. In severe cases the injection of antistreptococcal serum is justifiable. The greatest care must be taken to keep the infant warm.

### ECTHYMA

This condition may be regarded as a deep form of streptococcal impetigo, in which destruction of the epidermis occurs, and an ulcer results. The lesions begin as pustules or bullæ containing serum, which, as in ordinary impetigo, is either cloudy or, more rarely, frankly purulent according to the degree of secondary staphylococcal infection; the serum dries to form a thick, adherent crust of a dirty yellow colour, or reddish-brown from admixture with blood, and sometimes almost rupial in character. Around the crust may be seen in comparatively early lesions the shrivelled roof of the original bulla, and beyond this a red area of inflammation, with sometimes considerable œdema. On removal of the crust the underlying ulcer is exposed. It is not, as a rule, very deep, its borders are sharply defined, and its base is red; from it exudes a purulent or sero-sanguinous fluid, containing tissue-*débris*, elastic fibres, altered red blood corpuscles, leucocytes, and cocci. Under suitable conditions and treatment the lesions dry up,

the crusts are shed, and the ulcers heal, leaving scars, usually surrounded by a pigmented border. Ecthyma is most frequently seen on the legs, the buttocks, particularly in infants, and the back; circulatory stasis is an important contributory factor, so that it is apt to occur on varicose legs, and may be the starting point of a varicose ulcer. General debility, acroasphyxia, alcoholism, and diabetes are predisposing causes, and the condition may complicate a pruritus, scabies, and pediculosis. During the war it was a cause of much disability among our louse-ridden soldiers, and the streptococcus fæcalis appeared often to be the infecting organism (MacCormac).

**Anatomy.**—The ulcerated lesion shows necrosis, leucocytic infiltration and intense congestion of the dermis, with dilatation of the vessels, which are surrounded by sheaths of cells.

**Treatment.**—The local treatment should be carried out along the lines already suggested for impetigo. In obstinate cases exposure of the lesions to sunlight or the ultra-violet rays will usually hasten healing. In debilitated persons prolonged rest out of doors, and the administration of tonics, particularly the Syr. ferri iodidi B.P. and the Syr. calcii lactophosphatis B.P. are indicated.

### DERMATITIS GANGRENOSA INFANTUM

This very rare condition has already been referred to under the name of *varicella gangrenosa* (see p. 45), but, although it is usually a sequel of chicken-pox, it may follow other eruptions, such as vaccinia and the infective dermatitis known as "napkin-rash." It occurs usually in marasmic infants, enfeebled by previous acute disease, improper feeding and neglect, rickets or tuberculosis, but it has been seen in well-cared-for children. The lesions consist of sharply punched-out ulcers often covered by a black slough; they may heal, but in severe cases extend peripherally, producing vast areas of gangrenous ulceration. The children as a rule are acutely ill, with high fever; death results from pyæmia and septic bronchopneumonia. In cases that recover permanent wide-spread scarring is, of course, present throughout life.

**Ætiology.**—Probably the original infecting organism is, as in ordinary ecthyma, a streptococcus pyogenes longus, but secondary invaders, such as staphylococci, doubtless play an important and sometimes predominant part. There is considerable evidence that infection with the *Bacillus pyocyaneus* may in some cases, at any rate, be responsible for the development of the condition.

**Treatment.**—The same principles should be followed as indicated under pemphigus neonatorum.

### INTERTRIGO

The eruption known as intertrigo (*erythema intertrigo, eczema intertrigo*) is the inflammatory redness which occurs in the natural folds of the skin, for example in the groins, between the buttocks, in the axillæ, under the mammæ, behind the ears, and in fat people in the hypogastric fold and around the neck. The eruption corresponds closely to the parts of the skin that are in contact; the surface is raw, denuded of the upper layers of the epidermis, and it secretes a whitish, turbid fluid, different from the yellow serum or sero-pus of eczema, and not drying into crusts, unless mixed with the medicinal substances applied to it. At the bottom of the fold is often a fissure, which is easily seen when the opposing surfaces are separated. Intertrigo may give rise to no subjective symptoms, but sometimes itching is complained of, and secondary eczematisation, with lichenification, may result from constant scratching.

**Ætiology.**—The mechanical friction of two opposed surfaces of skin, the accumulation and decomposition of secretions, and the moisture of the parts are predisposing factors; it is obvious, therefore, that lack of personal cleanliness and obesity favour the development of intertrigo. Sabouraud considers that the

inflammation is due to streptococcal infection, and this is probably true, at any rate of many cases.

**Diagnosis.**—Intertrigo must be distinguished from *erythrasma*, the colour of which is brownish or yellowish-pink and which has a sharply defined border, but is not necessarily limited to opposing surfaces, from *epidermophyton inguinale*, which has a characteristic raised border and spreads beyond the actual folds, and from *psoriasis*, *eczema*, and *seborrhœic dermatitis* involving the flexures.

### INFANTILE ERYTHEMA OF JACQUET

(*Napkin Rash*)

Under this title is known a form of dermatitis, which is common in the napkin area of infants, and which may be considered as a variety of intertrigo. The eruption occurs on the buttocks, lower abdomen, genitals, and inner sides of the thighs, *i.e.*, on the areas in which there is friction from the napkin. It may also be seen on the calves and on the parts of the thighs opposed to them when the child lies with its legs drawn up, and also on the heels. In the early or erythematous stage the appearance is that of ordinary intertrigo, but the colour may be of a curious reddish-coppery hue; vesicles may then appear, leading to the formation of erosions, which, in turn, may be transformed into flat red papules, resembling somewhat those of congenital syphilis; lastly, the erosions may terminate in punched-out ulcers, which often coalesce.

**Ætiology.**—Irritation from napkins kept too long soiled with urine and fæces is an important predisposing cause, but the eruption is particularly common in marasmic infants with diarrhœa, and in those suffering from carbohydrate dyspepsia in whom excessive intestinal fermentation occurs, leading to the production of fatty acids, the resulting acid stools being extremely irritating to the skin. The secondary lesions are doubtless due to infection with streptococci, which abound in the intestines in cases of carbohydrate dyspepsia.

**Diagnosis.**—The importance of the eruption lies in the fact that in its severer forms it may easily be confused with a congenital syphilitic eruption (see p. 121), and Parrot, who first described it, wrongly attributed it to syphilis. The main points in the differential diagnosis are that a napkin rash occurs on parts exposed to pressure and friction, and not on the flexures, which are protected from contact with the napkin; there are no lesions on the palms and soles, the colour of the eruption is less coppery, and mucous patches on the arms and at the angle of the mouth are absent. Nevertheless, doubtful cases are met with, and careful investigation for syphilis should then be made both in the parents and child.

**Treatment.**—In ordinary intertrigo the opposing surfaces of the skin should be separated by pieces of linen smeared with Lassar's paste; or a talc dusting powder containing salicylic acid 2 per cent., boric acid 10 per cent., may be employed. Scrupulous cleanliness is essential, and, after washing the skin, it may be sponged with a 25 per cent. spirit lotion. The method recommended by Sabouraud, of painting the affected surface with R. Tinct. iodi mitis 1·0 Spirit. vini rect. ad 10·0, and then applying a zinc cream or dusting powder, is often very efficacious. In Jacquet's erythema it is usually necessary to correct the diet with a view to diminishing fermentation by limiting the intake of carbohydrate; a dose of castor oil, or small doses of calomel, and an alkaline mixture should also be prescribed. The local treatment consists in frequent changes of the napkin, the plentiful use of a dusting powder, such as that already mentioned, and the application of a weak mercurial ointment or paste should secondary infection have occurred.

### STREPTOCOCCAL FISSURE

As has already been stated, streptococci are prone to infect the folds of the skin, and to produce fissures therein and at the mucocutaneous junctions; in the

latter situations the mucous membrane is often the primary source of infection. Thus fissures in the naso-labial folds are usually secondary to an acute or chronic rhinitis, and those at the labial commissures—which form the starting point of *la perlèche*—may result either from a streptococcal stomatitis, gingivitis, or pyorrhœa, or from maceration with infected saliva in children who dribble. Fissures are also common in the retroauricular region (retroauricular intertrigo) and beneath the lobes of the ears.

*Retroauricular intertrigo* is often wrongly diagnosed as eczema, or seborrhœic dermatitis; it provides a very good example of chronic streptococcal infection of a natural fold. Behind one or both ears is seen a moist, crusted area, and beneath the crusts the surface is bluish-red; at the angle of the fold is the fissure, which divides the infected area into two halves, and is exposed on drawing the ear forward. Sometimes at the margin may be seen remains of the original streptococcal vesicle in the form of a shrivelled fragment of the raised horny layer.

*La perlèche*, or intertrigo of the labial commissures, may be confused with the mucous patches of syphilis and with labial herpes. It consists of a fissure at one or both angles of the mouth with a V-shaped inflammatory area spreading outwards, and covered with macerated greyish-white epidermis. It often co-exists with impetigo contagiosa, and may, like other streptococcal fissures, be a source of reinfection. It is contagious, being spread by kissing or by the exchange of pens and pencils, which have been held in the mouth of an infected person. It is often associated with dyspepsia of the fermentative type, due to an excessive carbohydrate dietary.

**Treatment.**—A streptococcal fissure should be painted with a 2 per cent. solution of silver nitrate in spirit, ætheris nitrosi, and then smeared with a mercurial ointment.

### RELAPSING STREPTOCOCCAL LYMPHANGITIS

This condition occurs most commonly on the face or around the ears. The malar regions and upper lip are common sites. Clinically it resembles ordinary erysipelas, the affected area being swollen, reddened, and somewhat shiny. There is usually little or no constitutional disturbance, owing to the slight virulence of the infecting streptococcus. Exposure to cold wind may provoke an attack. From repeated attacks permanent blocking of the lymphatics may result, with the production of one form of elephantiasis nostras. Persistent swelling of the upper lip and malar regions is not uncommon from this cause.

**Ætiology.**—The condition is due to streptococcal infection of the lymphatics of the part. In most cases there is present a streptococcal fissure from which reinfection periodically takes place. In some cases there is no external fissure, but, as a rule, some chronic focus of streptococcal infection in the mucous membranes of the mouth or nasopharynx is present.

**Treatment.**—If a streptococcal fissure is responsible, its cure will lead to a cessation of the attacks. In other cases any source of streptococcal infection in the mouth, nose, or nasopharynx should be sought for and treated. When persistent elephantiasis has supervened, removal of the original source of infection may be followed by a gradual disappearance of the swelling, but not always. Autogenous streptococcal vaccine does not appear to be of value.

## STAPHYLOCOCCAL AND OTHER INFECTIONS ASSOCIATED WITH THE SEBORRHŒIC STATE

It is obvious to any one with a sufficiently large experience of skin diseases that certain of them tend to occur in persons conforming to definite clinical types. Thus erythema induratum is seen almost exclusively in young girls, with fat,

œdematous-looking legs, in which there is marked circulatory stasis, particularly in the postero-external surface of the lowest third, where the lesions appear; the stagnation of the blood-flow in this situation, of course, favours the embolism of circulating tubercle bacilli to which the lesions are due. For the same reason the papulo-necrotic tuberculides are usually seen in patients with marked acro-asphyxia, and this is also true of lupus erythematosus. Persons with a hereditary tendency to the various toxic idiopathies (p. 224), *i.e.*, those with the hay-fever-asthma-eczema-urticaria-prurigo complex, are in a large proportion of cases ichthyotics. The commonest and most important predisposing condition, however, as regards affections of the skin, is what may be termed the *seborrhœic state*. Darier in 1906 described this under the name "la kérose"; he considers it a chronic morbid state of the skin characterised by: (1) a dirty yellow or greyish discoloration; (2) an accentuation of the pilo-sebaceous orifices; (3) some thickening of the epidermis. Anatomically there is slight diffuse hypertrophy of the horny layer, with a tendency to fine desquamation, a modification of unknown nature in its fat content, and a hyperkeratosis round the mouths of the pilosebaceous follicles. His view is that seborrhœa is one of the *complications* of his "kérose," and must not be considered as synonymous with it, and that the latter also provides the necessary or usual substratum for the development of pityriasis simplex, certain forms of alopecia and hypertrichosis, oily hyperidrosis, acne, rosacea, and seborrhœic dermatitis. As regards the ætiology of the condition he considers that heredity, or the influence of ill-health and faulty hygiene in the parents, prepare the soil, and that (1) a more or less disordered sexual evolution and (2) gastrointestinal fermentation and constipation due to improper diet, an excess of nitrogenous food, and eating too quickly with insufficient mastication, play the determining *rôle*.

There is no doubt that this conception of Darier is on the whole true; thus a tendency to the development of seborrhœic manifestations is certainly hereditary, and the influence of digestive disorders is generally admitted. But seborrhœa, by which term is meant an excessive secretion and an alteration in the composition of the sebum, together with the hyperidrosis that accompanies it, should be regarded not as *complications* of some underlying morbid state of the skin, but as *manifestations* of what has been called "*the seborrhœic state*" (**1**). Admittedly heredity may be a predisposing factor of importance, but given the necessary abnormal conditions of life, the seborrhœic state may supervene even in the healthiest person. This often occurs, for example, in the case of trained athletes when they abandon their games and adopt a sedentary life, without at the same time restricting their indulgence in the pleasures of the table. It would seem that the state depends on the excessive formation of acid bye-products which are relatively insoluble and with difficulty excreted by the urine, unless a sufficiency of alkaline bases is available; and it is their excretion *viâ* the skin that is doubtless responsible for the hyperactivity of the sebaceous glands (seborrhœa) and sweat glands (hyperidrosis oleosa), which characterises seborrhœic persons, and for the modification of the fat-content of the horny layer referred to by Darier. Presumably, too, the presence of these acids in the skin favours the growth of the micro-organisms on which the complications of the seborrhœic state depend, namely, the "bottle-bacillus," the acne bacillus, and various strains of staphylococci. The excessive excretion of acid by the skin is proved by the fact that the sweat of seborrhœic persons may be strongly acid, even in situations, such as the palms of the hands, where there are no sebaceous glands. *The mucous membrane of the mouth*, when tested with litmus paper, is also found to be acid, sometimes intensely so, but, if free salivation be provoked and the saliva collected, the latter is found to be of the normal alkaline reaction. This would suggest that the acid reaction of the mouth is due to the production of acids by bacterial fermentation. Moreover examination of the stools of seborrhœic persons has shown excess of streptococci and other acid-producing

organisms. It is probable, therefore, that one, and perhaps the chief source of the acids produced in the seborrhœic state is excessive intestinal fermentation. The flatulence, which is so common a symptom in patients with seborrhœic manifestations, is doubtless due to the gas formed by this fermentation, since it is rapidly controlled by the restriction of soft carbohydrate foodstuffs in their dietary. *The urine* in the seborrhœic state has been studied by several observers. The alkaline tolerance is, as a rule, markedly raised, so that very large doses of alkali may be required to render the urine alkaline (**1**). In some cases, usually of long standing, the ammonia-ratio and acid-ratio are high (**2**). Observations on the hydrogen-ion concentration of the urine in the seborrhœic state have shown that the pH value in normal controls (from which seborrhœic persons were carefully excluded) varied from 6·4 to 6·8, whereas in patients with seborrhœic manifestations it varied from 4·8 to 5·8 (**3**). Striking benefit accrues in these cases from the administration of alkali in sufficient quantity to restore the pH value to normal. A point of great interest is that the urine of a patient, even with severe seborrhœic manifestations, may be found to be slightly acid or even neutral, but that, when alkali is administered, its acidity increases progressively up to a certain point before becoming alkaline.

The diseases of the skin to which the seborrhœic state predisposes are as follows :—

| | |
|---|---|
| Pityriasis simplex | Infection with the "bottle bacillus." |
| Comedo | Infection with the *Bacillus acnes*. |
| Acne vulgaris | Infection with the acne bacillus and the *Staphylococcus albus*. |
| Impetigo of Bockhart | Infection with staphylococci. |
| Seborrhœic dermatitis | Infection with the "bottle bacillus" and staphylococci. |
| Sycosis coccogenica | Infection with staphylococci. |
| Sycosis nuchæ | Infection with *Staphylococcus aureus*. |
| Boils | Infection with staphylococci. |
| Carbuncle | Infection with staphylococci. |
| Dermatitis infectiosa eczematoides | Infection with *Staphylococcus aureus* and epidermal sensitisation to staphylococci. |
| Acne necrotica | Infection with the *Staphylococcus aureus*. |
| Multiple abscesses of infants | Infection with staphylococci. |
| Miliaria rubra | Infection with staphylococci. |
| Hidradenitis | Infection with staphylococci. |
| Granuloma pyogenicum | Infection with *Staphylococcus aureus*. |

To this list may be added the so-called *seborrhœic alopecia*, and a form of eczema, which is not apparently caused by infection with or sensitisation to a micro-organism, and which may be termed *seborrhœic eczema* in contradistinction to seborrhœic dermatitis and infectious eczematoid dermatitis. It should also be noted that certain forms of *streptococcal* infection, *e.g.* impetigo contagiosa, intertrigo, and *perlèche*, are commoner and more persistent among seborrhœic persons. *Rosacea*, too, may be in part regarded as a seborrhœic manifestation, since the pityriasis, the comedones, papules and pustules that complicate the primary erythema are due to infection with the bottle bacillus, acne bacillus, and staphylococci.

In the chronic seborrhœic state a number of the above conditions may, and commonly do co-exist. In the typical adult seborrhœic, *on the scalp* the hair is scanty—in men on the vertex and over the temples, in women over the whole scalp, but particularly on the vertex—there is pityriasis capitis, often complicated by seborrhœic dermatitis, which tends to spread behind the ears, in the folds of the pinna, and down the external auditory meatus. The skin of *the face* is greasy particularly in the centre of the forehead, at the sides of the nose, and

adjacent parts of the cheeks, and on the chin, in which situations the sebaceous glands are largest; actual droplets of sebum and sometimes of sweat—even in cold weather—may be seen at the orifices of the sebaceous follicles and sweat-ducts, and here and there careful observation will show superficial scaly patches of seborrhœic dermatitis and small inflammatory follicular papules; acne comedones and pustules, rosacea, chronic blepharitis with hordeolæ, and, in men, sycosis of the beard region, eyebrows, and temples may be present. On *the trunk* seborrhœic dermatitis of the presternal and mid-vertebral regions ("flannel rash") is common; on the back the pilosebaceous orifices are prominent and plugged with inspissated sebum or actual comedones, and every degree of acne, from a few follicular papules and pustules to deep purulent nodules or abscesses with deep scarring (acne indurata), may be seen. Boils and even carbuncles are also common. On *the limbs* the joint flexures are apt to be involved with diffuse seborrhœic dermatitis, which may also involve the penis and scrotum and the internatal cleft. Recurrent attacks of vesicular eczema (one form of "dysidrosis" or "cheiropompholyx") of the palms and hands may also occur. The *mucous membranes* show the same lowered resistance to infection as the skin, so that in the seborrhœic person chronic nasopharyngeal catarrh is the rule and gingivitis with pyorrhœa is common. Frequently an exacerbation or an acute outbreak of seborrhœic dermatitis coincides with an attack of acute coryza. The conjunctivæ are also often affected, particularly in rosacea, a staphylococcus being the usual infecting organism. In men chronic balanitis, with periodical acute attacks, occur.

**General Treatment of the Seborrhœic State.**—The diet should be regulated according to the climatic conditions and the amount of physical exercise taken. The great majority of people among white races not only eat far more than they need, but they live very largely on the entirely unnatural foodstuffs that civilisation has gradually evolved. Particularly harmful are ordinary white bread, soft farinaceous foods, and concentrated sweetstuffs, although they may be permitted in moderation provided abundant exercise is taken regularly. A normal diet should consist chiefly of a little fresh meat and fish, eggs, uncooked vegetables in the form of salads, cooked green vegetables, fresh and stewed fruit, wholemeal bread and butter, but it must be remembered that persons whose digestion has already been enfeebled by an improper diet are often unable at first to tolerate uncooked vegetables and fruit. Starch should always be taken in a form that requires thorough mastication; concentrated sweet food should be forbidden entirely, and an adequate supply of mineral salts and vitamins must be ensured. In persons already seborrhœic, the administration of alkaline salts, particularly sodium bicarbonate, over a long period is advisable. A point of the greatest importance is that several tumblers of a good natural or mineral water should be drunk daily between meals.

It will be convenient to consider here *pityriasis simplex* and comedo.

## PITYRIASIS SIMPLEX

The term *pityriasis* has, unfortunately, been applied to several distinct affections of the skin. Derived from πίτυρον, bran, it merely signifies scaliness. *Pityriasis versicolor* is due to infection of the horny layer with a fungus, the *microsporon furfur*. *P. rosea* is a specific disease due to infection with an organism hitherto undiscovered; *P. rubra pilaris* is also a specific entity of unknown causation, and *P. rubra* is an erythroderma.

*Pityriasis simplex* is a non-inflammatory, desquamative affection of the horny layer, commonly known as *dandruff* or *scurf*. In its simple form it affects chiefly the hairy regions, particularly the scalp (*P. capitis*), but, as will be seen later, the causal organism is concerned with staphylococci in the production of the inflammatory condition known as seborrhœic dermatitis (*P. circinata*). As

Whitfield has pointed out, the earliest stages of the affection are seen in young children, on whose scalps it begins, usually on the vertex, as little patches of dry scurf around the hair follicles. It gradually spreads as circular scaly areas, not unlike ringworm, until it becomes diffuse. From about the age of puberty onwards, when there is likely to be concomitant seborrhœa, the scales become greasy.

**Ætiology.** Although by some dermatologists it is considered as unproven, there can be no doubt that simple pityriasis is due to infection of the horny layer with the organism known as the "bottle-bacillus" or spore of Malassez, which is constantly present in the scales in a state of active multiplication. As Sabouraud has emphasised, there is a very striking similarity in the microscopical appearances of vertical sections through the scales of pityriasis simplex and of pityriasis versicolor, the microsporon furfur in the one case and the "bottle-bacillus" in the other lying between the layers of horny cells in an identical state of active growth. Botanically, the "bottle-bacillus" is probably a yeast. It is remarkably pleomorphic, and appears as large yeast-like cells, which show budding, or smaller bodies, which may be arranged in short chains. Most observers state that they have failed to cultivate it artificially in pure culture. There is no doubt that infection with this organism is spread by direct and indirect contact. When it occurs in babies, in whom pityriasis simplex of the scalp, owing to secondary staphylococcal infection, may be the starting point of one form of "infantile eczema," examination of the scalp of the mother or nurse will usually reveal a heavy infection, and the indiscriminate interchange of hair brushes is doubtless a potent cause of indirect spread. The seborrhœic state is the most important predisposing factor, and the seborrhœic skin obviously offers a suitable soil for the growth of the "bottle-bacillus."

**Treatment.**—The general treatment of the seborrhœic state (*q.v.*) is indicated, as it is extremely difficult to eradicate the infection by local measures alone. The scalp should be washed frequently, once a week in women, twice or oftener in men. At the beginning of treatment, soap spirit (spiritus saponatus, B.P.C.), or ether soap may be employed, but they should not be persisted with. Certain proprietary shampoos, containing tar, are the best and pleasantest for regular use. In mild cases the thorough daily application of a hair-lotion is sufficient, *e.g.* R. Resorcin, Acidi salicylici āā gr. x; Hydrarg perchloridi gr. 1/6; Ol. lavand. m. ii (Ol. Amygd dulc. q.s.); Acetoni dm. i; Spirit myrciæ (indust) a doz. i, the resorcin being omitted in persons with grey or fair hair. The lotion may be shaken on to the scalp through a sprinkler-cork and then well brushed in, or applied with a spray. In severe cases, particularly if there is much seborrhœa, the additional use of a pomade is essential, *e.g.* R. Liq. picis carbonis dm. i; Sulphur præcip, Acidi salicylici āā gr. xx; Vaselin alb. dm. ii; Ol. cocois nuciferæ ad oz i; Perfume q.s.; this should be thoroughly rubbed into the scalp on the nights previous to shampooing, and the above lotion (without the perchloride of mercury) used daily. It cannot be too strongly emphasised that the popular idea that the scalp should be washed only occasionally is erroneous, and is responsible very largely for the prevalence of dandruff. Provided that strongly alkaline and spirituous shampoos (which render the hair brittle, and in women, combined with the deleterious action of dyes and waving, produce the condition known as trichorrexis nodosa) are not employed, the head may be washed as often as desired.

## COMEDO

(*Blackhead*)

The *comedo* is one of the lesions of acne vulgaris (*q.v.*). It is a small, horny, cocoon-like mass, embedded in the mouth of a pilo-sebaceous follicle, and consists of concentric sheaths of horny cells, surrounding a partitioned cavity, in which lie masses of the acne-bacillus, inspissated sebum, and the debris of a dead

lanugo hair. The summit of the comedo is coloured dark-brown or black, not (as was formerly thought, and, by an oversight, stated in the previous edition of this work) owing to adherent dirt, but to an oxidation product of keratin (ultramarine); the lower part of the comedo is of a dirty yellow colour. Sometimes "double" or "multiple comedones" are seen, *i.e.*, two or more closely approximated comedones communicating at the base. The comedo is a retention cyst, and is due to hyperkeratosis at the mouth of a follicle, provoked doubtless by the irritation, mechanical or toxic, of the myriads of acne bacilli growing in the pilo-sebaceous follicle. *The acne bacillus may, therefore, be considered as the cause of the comedo,* and the essential predisposing factor to the formation of true comedones is seborrhœa, which favours the active growth of this bacillus.

Apart, however, from the comedones of ordinary acne, various external irritants are capable of inducing comedo-formation, which may be followed by the development of pustules. The commonest of these is camphorated oil, which gives rise to the so-called "grouped comedo" of infants and children, whose chests have been assiduously rubbed for bronchitis. The suppuration in camphorated oil acne may be severe and lead to extensive scarring. Turpentine liniment sometimes produces the same condition, and in adults various tars, particularly oil of cade, and chlorine vapours may respectively be responsible ("tar-acne," "chlor-acne").

The **Treatment** of comedo is dealt with under Acne vulgaris.

## ACNE VULGARIS (*See Plate XLV., B*)

The term *acne* (according to Littré, altered by a copyist from ἀκμή, a point) has been applied, since Willan and Bateman first introduced it, to a number of distinct eruptions in which the pilo-sebaceous follicles are affected. It is now less widely used, being as a rule reserved, apart from *acne necrotica seu varioliformis,* for the common disease *acne vulgaris*—polymorphic or juvenile acne.

The epithet "polymorphic" indicates that in acne there is not a single characteristic lesion, but rather a number of different forms of eruptive elements, some of which predominate in one case, others in another. The composite nature of the eruption depends partly on the fact that two distinct micro-organisms are concerned in its production, and partly on the relative activity of these two organisms, which varies in different patients. The essential predisposing factor in the development of acne is seborrhœa, and the earliest lesion is usually the *comedo* (*q.v.*), a retention cyst, the formation of which is provoked by the active growth of the *acne bacillus* in the pilo-sebaceous follicle. The other organism concerned is a *staphylococcus albus,* which is probably alone responsible for the inflammatory lesions met with in the disease, namely, the papules, papulo-pustules, superficial or deep follicular pustules, and deep indurated abscesses, although some observers have thought that the *acne bacillus* itself may be pyogenic. In some cases comedones in large numbers are present alone—*acne punctata;* in others reddish papules, usually formed around comedones and due to staphylococcal perifolliculitis, occur intermingled with simple comedones—*acne papulosa;* in others, again, the staphylococcal infection is more acute, the papules are bright red and acuminate, suppuration takes place at their summits, and the yellow pus produced either exudes or dries up to form crusts, under which the papule involutes, leaving a minute scar—*acne pustulosa;* in the more severe cases large, indurated, deep-seated nodules are seen, some of which undergo a slow transformation into indolent pustules, from which a quantity of pus may be evacuated, and which on healing leave scars of considerable size and depth—*acne indurata.* Sometimes bluish-red, fluctuating abscesses are formed, which may involve the hypoderm. The so-called "butyric cysts" result from degeneration of large comedones, and contain a semi-fluid, cheesy substance, having a

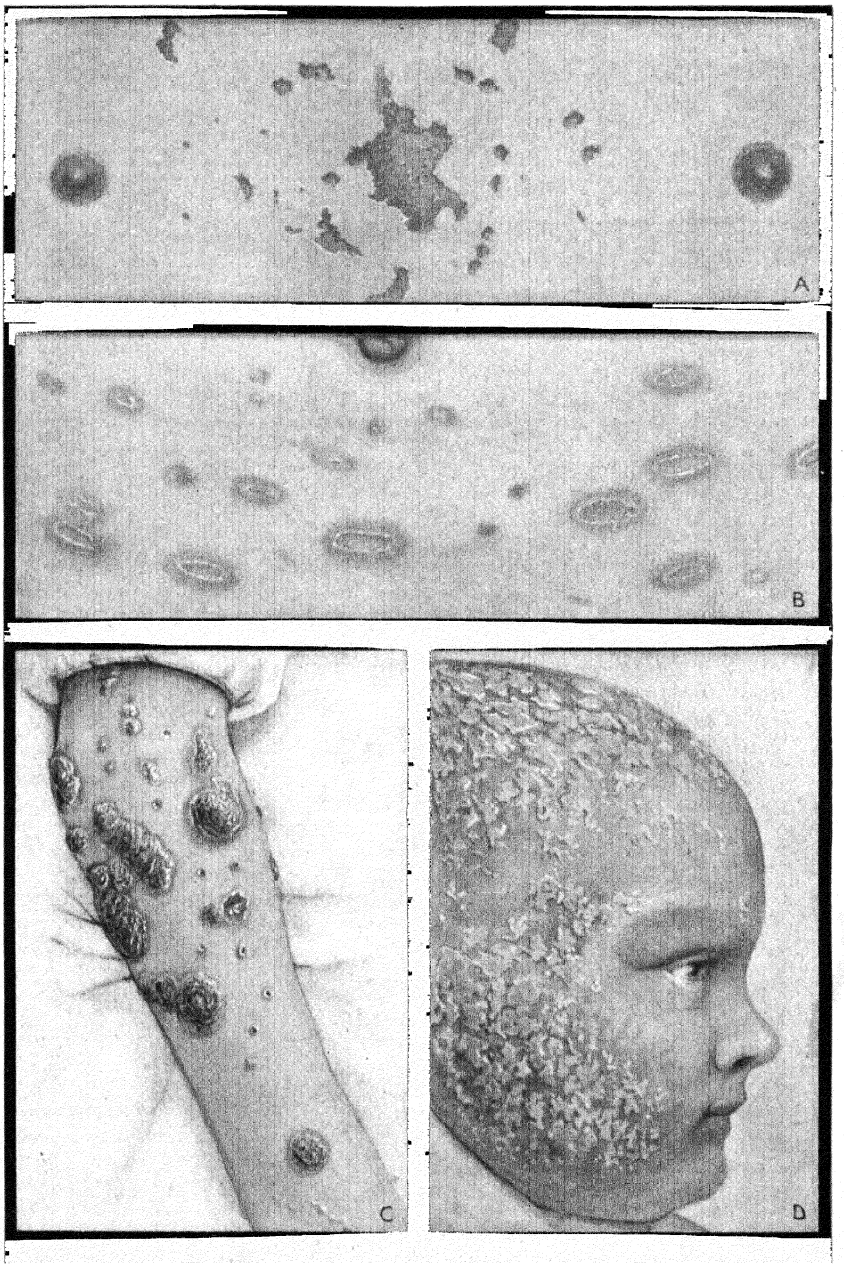
peculiarly nauseous smell. Mucoid cysts, due to cystic degeneration of long obstructed sebaceous glands, are more rarely seen.

As has been indicated, these different types of lesions coexist in the same patient, and all degrees of severity are met with, from a few comedones and occasional papulo-pustules to the worst form of acne indurata with large, painful nodules and abscesses, and extensive deep scarring. The distribution of acne is more or less constant, the eruption occurring almost exclusively on the face and back, less commonly on the front of the chest and over the deltoids; on the back, it involves chiefly the scapular regions and only rarely descends below the waist line.

**Ætiology.**—The various lesions which together constitute acne vulgaris occur exclusively in seborrhœic subjects, in whose sebaceous follicles the acne bacillus grows in profusion. This bacillus is the cause of the comedo, the other lesions being due to infection with a staphylococcus. A person with acne is presumably sensitised to these two organisms, until, as age advances, immunity is developed. In this connection, it has been shown that in a large majority of cases positive complement-fixation tests are obtainable to the acne bacillus, staphylococcus, and to strains of *B. coli* obtained from the intestines of acne patients (**4**). The disease usually first makes its appearance about the age of puberty, when there is a great increase in the development and activity of the sebaceous glands. It may, however, occur in mild form several years before puberty. It reaches its acme of severity, as a rule, between the ages of sixteen and twenty-five, and then tends to decline, being succeeded by other manifestations of the seborrhœic state, such as cavities, furunculosis, sycosis, and seborrhœic dermatitis. In some cases, however, it may persist till late in middle life, and it is by no means uncommon to find comedones and acne papulo-pustules even in the aged. Apart from the influence of sexual maturity, the predisposing causes of acne are those of the seborrhœic state, and of particular importance are an *unsuitable dietary*, above all an excess of sweet and starchy foods and certain forms of fat (as Kenneth Wills has emphasised, pig-fat is especially injurious), *lack of fresh air, sunshine and exercise*, and *chronic constipation* with intestinal toxæmia. The worst examples of the disease are usually seen in thick-skinned persons of the scrofulous type, with the symptoms and signs of intestinal autointoxication. Many of such patients have suffered from tuberculosis of glands, peritoneum, bones or joints, and it is in these that deep scarring is most likely to occur. The undoubted influence of constipation is of interest in connection with the above-mentioned positive complement-fixation tests to the *B. coli*.

**Treatment.**—*Internal.*—The internal treatment of acne vulgaris must depend on the symptoms presented by each individual patient. Thus in some cases there is anæmia, hypochlorhydria, chronic constipation, and an excess of indican in the urine; for these, morning saline aperients, pills containing iron and aloes, and dilute hydrochloric acid in doses of 20 to 40 minims after meals are indicated. In full-blooded patients, on the other hand, an alkaline mixture containing sodium bicarbonate, potassium citrate, and magnesia taken before meals is often beneficial. As regards the diet, it is usually important to restrict the amount of carbohydrate, and sweets, cakes, pastries, puddings, jam and marmalade should be forbidden altogether; plenty of fresh fruit and green vegetables should be taken, and it is very important to insist that several tumblers of water, either hot or cold, be drunk during the day, preferably about half an hour before meals. Avoidance of excess of tea and coffee is also essential, and whereas beer or stout in moderation are probably beneficial, wines and spirits certainly do harm. Vaccine therapy in acne is not so uniformly successful as in furunculosis, but is sometimes very helpful, particularly in cases with much staphylococcal infection; an autogenous preparation made from the acne bacillus and the *Staphylococcus albus* and *aureus* is usually preferable to a stock vaccine. In the pustular form of the disease injections of collosol manganese and the oral

# PLATE XLVI.




A SEBORRHŒIC DERMATITIS (*Pityriasis circinata Seborrhœa corporis*, "*Flannel-rash*"). Note the characteristic position of the eruption in the presternal region, the early follicular inflammatory lesions, the formation of the patches by involvement of adjacent follicles and infection of the interfollicular areas, their yellowish-red colour, and slight scaliness.

B. PITYRIASIS ROSEA.—Note the small pink papular elements, and the characteristic oval patches, lying with their long axes parallel to the line of the ribs. These patches have a pink outer border and a fawn-coloured central area, between which is a fine collarette of white scales with their free edges lying inward.

C PSORIASIS.—Note the distribution on the extensor surface of the limb, the heaped-up silvery scales, the dry appearance of the eruption, and the sharp line of demarcation between the patches and the surrounding normal skin.

D INFANTILE ECZEMA.—Note the distribution of the eruption on the scalp and cheeks, the central part of the face being practically free, its moist appearance and ill-defined margins, and the yellowish crusts of dried serous exudation.

that has damaged the horny layer. Thus it may follow inunction with ung. hydrargyri for syphilis or pediculosis pubis, the application of liniments, plasters, etc., and is frequently seen around a boil that has been poulticed or fomented, the warmth and the maceration of the skin favouring infection with the staphylococci contained in the pus from the boil. It may be produced artificially by rubbing a culture of staphylococci into the skin. When it occurs on the beard region in men it may, from extension of the infection, lead to the development of deep staphylococcal folliculitis or sycosis coccogenica.

**Treatment.**—The pustules should be opened and the pus evacuated as far as possible. The affected area should then be bathed with boric lotion, dried, and dusted freely with the pulv. acidi salicylici comp. B.P.C., and then covered with lint. On the scalp, washing with a weak lysol solution, followed by the application of a 2 per cent. ointment of hydrarg oxidi flavi, is usually successful, or the lesions may be painted with a weak solution of iodine in spirit.

### SEBORRHŒIC DERMATITIS (*See Plate XLVI., A*)

The term "seborrhœic dermatitis" includes a group of eruptions which have received a variety of names from different observers; for example, *lichen circumscriptus* (Willan and Bateman), *lichen annulatus serpiginosus* (Erasmus Wilson), *pityriasis circinata* (Bazin), *seborrhœa corporis* (Duhring), *eczema seborrhœicum* (Unna), seborrhœides (Audry, Brocq), "flannel-rash," etc. Darier has proposed for them the term "*eczématides*," thus indicating their close clinical and histological relationship to eczema. The confusion that still reigns concerning them is unnecessary, for they are due to the active growth in the skin of staphylococci and the "bottle bacillus," and the differences that are observed in their clinical appearances are doubtless dependent on the particular strain or degree of virulence of the infecting staphylococcus, the resistance of the patient, and the part of the skin affected.

Following Darier, one may recognise three main types of seborrhœic dermatitis (1) The figurate type (*pityriasis circinata*, figurate medio-thoracic dermatosis of Brocq, *seborrhœa corporis* of Duhring, *pityriasis steatoides* of Sabouraud, "flannel-rash"), (2) the pityriasiform type; (3) the psoriasiform type. But it must be admitted that these distinctions are artificial, since they depend partly on the particular site involved, and they may all co-exist in the same patient.

1. *The Figurate Type.*—This is seen most typically in the presternal and interscapular regions, whence it may spread laterally, and on the scalp, from which it may extend to the forehead (*corona seborrhœica*), the temples, and behind the ears. Its evolution can best be studied on the trunk. It will be observed that the inflammatory process begins at the mouths of the pilo-sebaceous follicles, so that the eruption is primarily follicular; the interfollicular areas are then involved, with the formation of petaloid patches, which by coalescence become figurate. These patches are of a pinkish or bright red colour at the spreading margins, which are slightly raised, yellowish or fawn-coloured in the centre. They are covered with greasy scales. The eruption may cause considerable itching, or may pass unnoticed and be present for years. It is very common in seborrhœic persons, particularly in those who wear flannel next the skin or excessive underclothing, and often co-exists with other seborrhœic eruptions, such as acne, rosacea, sycosis, furunculosis, etc.

2. *The Pityriasiform Type.*—This type represents a more diffuse form of seborrhœic inflammation of the skin. It occurs as oval, round, or irregular patches, slightly scaly, dry or greasy, according to their situation, and pink or pinkish-yellow in colour. They occur on and around the scalp, the neck, the upper portion of the trunk, and in the joint flexures, such as the axillæ, antecubital fossæ, popliteal spaces, and groins, and in the inter-natal cleft. They may also be present on the extensor surfaces of the limbs in persons with dry skins, the

vulnerability of the horny layer doubtless favouring staphylococcal infection. Sometimes acute outbreaks of this form of seborrhœic dermatitis occur on the trunk, and the eruption may closely resemble pityriasis rosea, but the presence of the "bottle bacillus" in a state of active growth in the scales of the former suffices to distinguish between them. Secondary eczematisation may take place, particularly on the scalp, ears, neck, and joint-flexures, so that the eruption becomes red, vesicular and moist from serous exudation.

3. *The Psoriasiform Type.*—In this form the inflammation is deeper and more intense than in the preceding varieties. The patches are more infiltrated, of a darker red, and covered with thicker more or less adherent scales. They may resemble psoriasis very closely; in fact, in some cases it may be impossible to make a differential diagnosis without a careful microscopical examination. The eruption may occur as a single chronic patch, or may extend widely and form confluent areas of considerable size. Itching is, as a rule, a prominent symptom, and may be intense; from constant scratching and rubbing lichenification may result.

**Ætiology.**—Seborrhœic dermatitis may be regarded as a diffuse catarrh of the skin due to microbial infection—in other words an infective epidermodermatitis. It is characterised by the presence in the lesions of staphylococci and of the "bottle bacillus" in a state of active growth. It thus differs essentially from psoriasis, the lesions of which are remarkably amicrobial and the scales of which are free from "bottle bacilli," and from pityriasis rosea, in which this organism is also absent. The differences that are seen in the various forms of seborrhœic dermatitis depend chiefly on the type and virulence of the infecting staphylococcus. Thus in the figurate and the milder forms of the pityriasiform types, a *Staphylococcus albus* is usually found to be the causal organism, whereas in severer cases of the latter, and in the psoriasiform variety one finds a *Staphylococcus aureus* or *citreus*. The seborrhœic state is the principal predisposing factor to the active growth of these organisms, and, as has been suggested, it is probably the excretion of certain fatty acids *viâ* the sebaceous glands that favours their growth; for this reason the distribution of seborrhœic dermatitis usually corresponds to the areas in which the sebaceous glands are largest and most active. Needless to say, lack of personal cleanliness and the wearing of excessive clothing are additional predisposing causes.

**Treatment.**—The internal treatment is that of the seborrhœic state, and the local measures to be adopted will depend on the type and severity of the inflammatory process. The figurate variety, that is seen so frequently on the back and chest, is rapidly cured by applications of sulphur, *e.g.*, R Sulphur præcip. gr. x to xx, Resorcin gr. x, Past. Zinci Comp. B.P.C. ad oz. i. For the diffuse pityriasiform patches, which occur chiefly in the joint-flexures, on the ears, and neck, sulphur is apt to be too irritating, and ichthyol and tar in an ointment or paste are more suitable, *e.g.* R Ichthyol gr. v, Liq. Picis Carbonis gr. xxx to dm. i, Past. Zinci Comp. B.P.C. ad oz. i. In the psoriasiform type more active preparations are necessary, such as oil of cade, the crude coal-tar paste recommended for chronic patches of eczema, and even chrysarobin. The X-rays are of great value, but the dosage must be carefully controlled.

In all cases of seborrhœic dermatitis it is essential to treat the scalp, which is so often the original source of infection. In men it should be shampooed with a tar soap twice or thrice a week, and on the nights before shampooing the following pomade should be well rubbed in: R Sulphur præcip. gr. xv to xxx, Acidi Salicylici gr. x to xx, Vaselin alb. dm. ii, Ol. cocois nuciferæ ad oz. i, Perfume q.s. Every morning the following lotion may be used: R Resorcin gr. x (to be omitted in persons with fair or grey hair), Acidi Salicilyci gr. x, Hydrarg. perchloridi gr. ½, Ol. Lavandulæ m. ii, Ol. amygd. dulc. m. x, Spt. myrciæ. (indust.) ad oz. i. In women the hair should be shampooed once or twice a week, and the pomade rubbed in the previous evening; the above lotion should be used daily, the almond oil

being omitted from the prescription ; but care should be taken to avoid irritation of the face and neck by the sulphur in the pomade, and a bathing-cap should be worn on the night it is applied.

In cases of very acute seborrhœic dermatitis, or when eczematisation has occurred, soothing applications should be used, such as calamine liniment or an ichthyol cream, *e.g.*, R Ichthyol gr. v, Ol. Olivæ, Zinci Oxidi āā oz. ss. Adip. lanæ anhyd. dm. i, Aq. Calcis dm. iii, and olive oil should be employed to clean the affected parts morning and evening.

In chronic or recurrent cases an autogenous staphylococcal vaccine is of great value, and should be combined with careful dieting, an out-of-door life, and the long-continued administration of alkalies. Intravenous or intramuscular injections of peptone, and intramuscular injections of a non-specific protein, such as "Aolan," also often have a remarkable effect in these persistent cases, particularly if there is a tendency to eczematisation. Lastly, when an out-of-door life is impracticable, great benefit accrues from artificial light baths with a carbon-arc or mercury-vapour lamp.

## SYCOSIS COCCOGENICA

By the term *sycosis* is meant a pustular folliculitis of the hairy parts ; two forms are recognised, one, *simple sycosis* or *sycosis coccogenica*, due to infection of the follicles with a staphylococcus, the other, *tinea sycosis*, due primarily to infection with a ringworm fungus, but later complicated by invasion with pyogenic organisms. The commonest sites of infection in coccogenic sycosis are the beard and moustache regions in men, but a similar folliculitis occurs on other hairy parts, such as the eyebrows, axillæ, pubis, and scalp. The lesions are deep follicular pustules and papules, which are pierced by the hairs. They may be discrete, but more commonly are confluent, occurring in irregular patches, or in severe cases involving the whole of the beard and moustache regions. The skin between the follicles is usually affected with a diffuse staphylococcal dermatitis (so-called "seborrhœic sycosis"). The degree of inflammation varies ; in some cases it is severe, so that the affected parts are red and swollen and covered with yellowish crusts, formed by the dried sero-purulent exudate ; in others there is no swelling, but either discrete pustules and papules, arranged singly or in confluent patches, or a diffuse scaly dermatitis dotted with yellow follicular pustules. On extracting the hairs from infected follicles, their roots are seen to be surrounded by a gelatinous sheath, which is the epithelial sheath infiltrated with pus ; this sign is evidence of involvement of the depth of the follicle, and is not met with in the superficial folliculitis that occurs in the impetigo of Bockhart. Sycosis coccogenica is essentially a chronic affection, and often most resistant to treatment owing to the deep involvement of the follicles and the consequent impossibility of penetration of local antiseptic applications.

**Ætiology.**—The infecting organism is usually a *Staphylococcus aureus*, although the white and yellow varieties are sometimes responsible. The condition is never seen except in seborrhœic persons, and usually other seborrhœic manifestations, such as pityriasis, seborrhœic dermatitis, acne and boils are or have been at times present. The most severe and resistant cases occur in those whose resistance to infection has been low from childhood, the subjects of chronic infective eczematous dermatitis of the scalp, ears and flexures, chronic blepharitis, rhinitis, and naso-pharyngeal catarrh. The condition may arise on the beard region after an attack of seborrhœic dermatitis or furunculosis, from direct infection in the barber's shop, and from the extension of the superficial staphylococcal folliculitis so common in seborrhœic persons with a stiff beard. It may also follow an attack of impetigo contagiosa, the secondarily infecting staphylococci invading the follicles. The possibility of this complication should always be borne in mind in cases of impetigo of the beard region in men. Sycosis of the

moustache is nearly always secondary to an acute or chronic nasal catarrh ; it may be unilateral at its commencement. As Sabouraud has pointed out, it not infrequently occurs in those who have suffered from childhood from a chronic staphylococcal blepharitis, virulent staphylococci passing down the nasal duct and invading the follicles of the upper lip when the moustache begins to grow.

Apart from these various sources of infection and the underlying seborrhœic state, there is a specific lack of resistance to staphylococci in patients with sycosis, which would seem to be both general and local. A differential leucocyte count may show a low proportion of polymorphonuclear white corpuscles, and chronic sepsis in the mouth or elsewhere may be an important factor in this lowered resistance.

**Treatment.**—The main principles of treatment of the underlying seborrhœic state, already detailed, should be followed, and everything possible should be done to increase the patient's resistance to infection. If practicable, he should live an out-of-door life. Heliotherapy or artificial light baths are, perhaps, the most potent means we possess of raising the bactericidal power of the blood against staphylococci, and it is probable that in the future they will provide the method of choice in the treatment of sycosis. Vaccine therapy is valuable, but is seldom successful alone ; an autogenous preparation is preferable to a stock one. It should be persisted with in suitable dosage even after apparent cure. Injections of nuclein, of colloidal manganese, of non-specific protein, such as milk, and the administration of stannoxyl by the mouth have been tried with only moderate success. Fresh brewer's yeast sometimes seems to have a definite controlling effect.

In sycosis of the moustache, it is essential to treat the intra-nasal infection responsible, and oral and nasopharyngeal sepsis must in all cases be dealt with if present.

By far the most efficacious method of local treatment is radiotherapy. A single epilation dose may be given, or fractional doses at suitable intervals. After the hair has fallen the case often appears cured, but relapse will occur with the re-growth of hair unless an antiseptic ointment containing sulphur or mercury be well rubbed in ; even when this is done, recurrence is common. The patient should always be warned that he must remain clean shaven for at least a year after apparent cure.

### SYCOSIS NUCHÆ

(*Acne Cheloid ; Dermatitis Papillaris Capillitii*)

This condition is analagous to ordinary sycosis, and like it occurs exclusively in males who are seborrhœic. It is a perifollicular inflammation of the nucha, and is characterised by the formation of dense cheloid-like sclerosis of the connective tissue. On the affected area comedones, deep follicular pustules, and abscesses are seen. Sometimes bluish-red papillomatous vegetations arise, which bleed easily and discharge an offensive semipurulent secretion. These vegetations later become transformed into cheloid bands. Many of the hair-follicles are destroyed by the chronic inflammation, but some are spared, so that characteristic little tufts of hair are left projecting from the cheloid mass.

**Ætiology.**—The condition, as has been said, is confined to males, many of whom suffer or have suffered from ordinary acne or sycosis. It is without doubt due to a chronic infection of the nuchal skin with a *Staphylococcus aureus*, and probably the dense nature of the connective tissue in this situation is responsible for the peculiarly indolent nature of the inflammatory process and for the resulting cheloid-like formation.

**Treatment.**—Apart from the general measures advised in sycosis the only treatment that is really satisfactory is radiotherapy. Full pastille doses should be given at intervals of a month until a cure is obtained.

## BOILS

(*Furuncles, Furunculosis*)

These are local infections by a *Staphylococcus pyogenes*, starting in the neighbourhood of the hair follicles. They are common on the back of the neck, the trunk, and the buttocks; and they may be caused by the pressure, friction, and irritation of clothes. There is often an antecedent state of ill-health, such as that present in convalescence from acute illnesses, and in diabetes, when the resistance to organisms is less than normal.

**Symptoms.**—The trouble begins with a painful red and slightly raised spot or pimple, which gradually enlarges, and becomes hard, infiltrating the surrounding skin, and extending deeply at the same time. The colour becomes dusky red, and ultimately the apex softens, a little pus being extruded, and later a whitish-yellow slough of necrosed tissue. When this has been discharged, the inflammation subsides, and the sore gradually heals by granulation, leaving a small scar.

In some persons, boils are constantly forming in all parts of the body, and this may go on for months or years (furunculosis).

**Treatment.**—*Local.*—In the early stage a boil may sometimes be aborted by the application of Unna's mercury and carbolic plaster. For the relief of pain hot fomentations with a 1 in 4,000 solution of mercuric chloride may be used, but poultices and boric fomentations should be avoided, as they macerate the skin and favour the production of fresh pustules in the vicinity of the original boil. When softening has occurred, the centre of the boil may be pierced with a sharpened match-stick dipped in pure carbolic acid, and a dry sterile pad then applied. The surrounding skin should be dusted over with an antiseptic powder.

*Internal.*—Boils are common in diabetes and in hyperglycæmia; at the same time it must be remembered that in the presence of boils and carbuncles, as well as in other infections, the blood-sugar may be raised, only to fall to normal when the infection is overcome. It is important in cases of chronic furunculosis to search for some focus of infection, such as oral sepsis, which may be a predisposing cause. In plethoric persons with a highly acid urine the administration of large doses of alkalies, *e.g.* Sod bicarb. ʒj, Potass. citrat. gr. xxx, thrice daily before meals and saline aperients is indicated; on the other hand, for debilitated, anæmic patients, in whom there is often hypochlorhydria and intestinal toxæmia with marked indicanuria, full doses of hydrochloric acid combined with arsenic, strychnine and iron are indicated. Recently a method of treatment by intramuscular injections of collosol manganese has been introduced, and is worth a trial in obstinate cases. Three or four injections in doses of from 0·5 c.c. to 1·5 c.c. should be given at intervals of three days. Though strikingly successful sometimes, this method often fails completely; still more uncertain is that introduced by Frouin and Grégoire of giving pure metallic tin mixed with its oxide ("stannoxyl") by mouth. Brewers' yeast in doses of 1 to 2 ounces per diem is sometimes of great value in the treatment both of furunculosis and acne. Probably the most reliable method of all, however, in furunculosis is by means of a vaccine of the *Staphylococcus aureus*. Either an autogenous or a polyvalent stock preparation may be used, and the injections should be given at weekly intervals at first, in doses of 100 to 2,000 million, when the larger doses are reached, the intervals should be longer.

## CARBUNCLE

This is a similar infection by staphylococci, leading to extensive inflammation, induration, and necrosis of the skin and subcutaneous tissue. It differs from a boil in its extent, and in the fact that there are numerous foci of suppuration, leading to discharge through several openings instead of one. The patients are generally of middle age or older, and diabetes mellitus is a very common

antecedent. The common seat of the lesion is the back of the neck, but it also occurs often on the back. It begins as a flat, indurated area of red or purplish-red colour; this extends and becomes more elevated above the level of the skin, spots of softening occur, and discharge of pus takes place. The hard mass forms a slough, which comes away, leaving a deep ulcer; and this ultimately heals by granulation, leaving a scar. A carbuncle may be from 1 to 3 inches in diameter, and occasionally very much more. There is much constitutional disturbance, with pyrexia; septic infection is very likely to occur, or death may take place from exhaustion.

**Treatment.**—This is largely surgical. If recognised early, carbuncle may be injected with carbolic acid; at a later stage it has been customary to relieve tension and facilitate separation by making a crucial incision by the knife, or scraping out and applying pure carbolic acid may be practised.

### DERMATITIS INFECTIOSA ECZEMATOIDES

Under this title Engman, and later Fordyce and Sutton, have described a form of eczematoid dermatitis which usually follows some localised infective lesion of the skin, such as a chronic patch of varicose dermatitis, occupational eczema, furunculosis or carbuncle, wounds, chronic otorrhœa, discharging sinuses, etc. It is not uncommonly seen after an attack of scabies with secondary infection, in chronic osteomyelitis with infection of the overlying skin, and after compound fractures. The eruption, as a rule, begins as eczematous patches around the original infective lesion, and then there appear *at a distance from this* more or less sharply circumscribed circular or oval patches, most commonly situated on the arms, thighs and legs. These patches are highly characteristic, and may be seen to arise from a single vesicle or vesico-pustule on an erythematous base; the inflammation spreads peripherally until a circinate patch is formed; the vesicles are large, and dry up to form discrete yellowish crusts. The so-called *nummular eczema* is usually this form of eczematoid dermatitis.

**Ætiology.**—The type of inflammation of the skin which we call *eczema* is an indication of epidermal sensitisation, and in dermatitis infectiosa eczematoides the sensitising agent is a staphylococcus, usually the *S. aureus*. Sensitisation takes place owing to the existence of some focus of staphylococcal infection in the skin. Positive cutaneous reactions to the staphylococcus are often obtained, and Sutton has pointed out that in about 25 per cent. of cases urticarial lesions may accompany the eruption, thus indicating that systemic, as well as epidermal, sensitisation has occurred. Eyre has shown that specific agglutinins to the infecting staphylococcus are often present in the blood.

**Treatment.**—It is essential first to treat the original focus of infection, to which the sensitisation of the skin is due. When this is cured the further outbreak of eczematous patches sometimes, but not always, ceases. Desensitisation should, in persistent cases, be attempted by increasing doses of an autogenous staphylococcal vaccine. In some cases intravenous or intramuscular injections of peptone are successful. Locally the eczematous patches should be covered with a paste containing tar and ammoniated mercury, *e.g.*, ℞ Liq. picis carbonis m. xxx to dm. i, Hydrarg. ammoniat. gr. v, Past. Zinci Comp. B.P.C. ad oz. i; they may be painted, previous to the application of the paste, with a 2 per cent. solution of silver nitrate in Spt. ætheris nitrosi, and in some cases a crude coal tar paste is the most effective application. Most patients who develop the condition are seborrhœic, and the administration of alkalis, as Sutton has noted, is often valuable.

### ACNE NECROTICA

(*Acne varioliformis, Acne frontalis*)

This is a comparatively rare affection, characterised by the formation of indolent, inflammatory, follicular papules at the apices of which develop vesiculo-

pustules. These dry up to form crusts, and the lesions slowly involute, leaving characteristic pitted scars. The sites of predilection are the forehead and temples, the scalp, and the midline of the back and chest. Like sycosis, it is essentially chronic, and very liable to recurrence.

**Ætiology.** The infecting organism would appear to be the *Staphylococcus aureus*, but there is considerable evidence to show that an underlying chronic tubercular infection is responsible for the peculiar characters of the disease. Some go so far as to classify it definitely among the tuberculides. It is certainly true that staphylococcal lesions in tubercular persons are more liable to leave scars than in the non-tuberculous. The patients are always seborrhœic.

**Treatment.** Temporary disappearance of the lesions can be brought about by the local application of ointments containing sulphur, salicylic acid, resorcin and oil of cade. Mercurials are less efficacious. An autogenous staphylococcal vaccine given over a long period is helpful. Probably heliotherapy will prove to be the most successful method of treatment, as in other staphylococcal infections.

## MULTIPLE ABSCESSES OF INFANTS

This condition, which is not a common one, is seen as a rule in marasmic children suffering from improper feeding, enteritis, bronchopneumonia, etc. It is apt to be associated with the infantile erythema of Jacquet. The abscesses begin as hard, deep nodules in the skin, particularly in situations liable to soiling, such as the buttocks and thighs, and to friction or pressure. Early lesions can be felt before they are visible, and the colour of the nodules remains unchanged until the abscess approaches the surface, when the overlying skin becomes red or bluish, and, unless previously incised, ruptures with the discharge of liquid pus. In neglected infants ulcers or fistulæ may supervene, and secondary adenitis, with subsequent softening, may be a complication.

The nodules vary from a pea to a hen's egg in size, and the quantity of pus they contain is often astonishing. The diagnosis is easy, although the nodules have been mistaken for gummata, which are very rare in infants, and for cold tuberculous abscesses.

**Ætiology.** The abscesses were formally thought to be pyæmic in nature, but it has been shown that they are due to infection with a staphylococcus (*S. aureus* or *albus*), *via* the sweat-ducts (**5**). The depth of the lesions is thus explained. With them may coexist small superficial intra-epidermic pustules which are formed at the orifices of the sweat-ducts.

**Treatment.** The most important part of the treatment is to attempt to raise the infant's resistance by attending to the underlying debilitated condition. Locally scrupulous cleanliness, the incision with a small knife of the abscesses, boric baths, and the copious use of a boro-talc powder, are the only measures necessary. The infants should, if possible, be kept in bed out of doors.

## MILIARIA RUBRA

(*Prickly heat: Lichen tropicus*)

This eruption is most commonly seen in Europeans who inhabit tropical countries, but negroes are never affected. It occurs in temperate climates in those who perspire freely and who are seborrhœic. It consists of innumerable minute red spots with tiny central vesicles containing a turbid fluid. These spots are hardly elevated, and they are not follicular. The trunk and the proximal segments of the limbs are the sites of predilection. Histologically there is seen to be congestion, with the presence in the rete of minute vesicles, formed either, as in impetigo, by cleavage or, as in eczema, by spongiosis; these vesicles contain numerous polymorphonuclear leucocytes, and arise frequently, though

not invariably, around a sweat-duct; they are not, as Pollitzer stated, due to dilatation of the ducts.

**Ætiology.**—The eruption is commoner in males than females, and appears suddenly after profuse sweating, provoked by a rise of temperature, the taking of hot alcoholic drinks, a hot bath, or violent exercise. The vesicles contain staphylococci in a state of active growth, and Darier's view that the eruption is due to an auto-inoculation of the skin with these organisms, caused by maceration of the epidermis with sweat, is probably correct. As he points out, there is frequently present some active infective focus, such as a boil, impetigo, seborrhœic dermatitis, acne pustules, etc. As predisposing factors may be mentioned the seborrhœic state, the wearing of excessive clothing (and particularly of flannel next the skin), over-eating and dyspepsia, and the abuse of alcoholic drinks. The fat, plethoric, cocktail-drinking European in the tropics is the commonest victim.

**Treatment.**—A brisk purge, twenty-four hours' starvation, copious drinks of barley water and fresh lemon juice, and the administration of alkalies in large doses will rapidly cut short an attack. Locally, tepid baths containing zinc sulphate dm. ii to iii, followed by the application of a boro-talc powder, will suffice. Recurrence can be prevented by instituting a rational dietary, the avoidance of alcohol, the wearing of light, porous clothing, the taking of alkaline drinks, regular bathing, and the habitual use of a boric dusting powder.

## HIDRADENITIS

Frequently mistaken for furunculosis, this affection occurs almost exclusively in the axillæ and, more rarely, in the perinæum. The lesions consist of oval, inflammatory nodules, which are often extremely painful; they may involute without breaking down, but more commonly soften, burst and discharge a creamy pus, but, unlike a boil, no core. The inflammation is essentially indolent, and each nodule may last from ten days to a fortnight. Recurrence is very common, and in severe cases both axillæ in their entirety may be involved with multiple lesions.

**Ætiology.**—The nodules, like those of the condition known as "multiple abscesses of infants," are due to staphylococcal infection of the sweat-glands, and not of the pilo-sebaceous follicles. The affection is commoner in women than in men, and occurs in seborrhœic persons who perspire freely. It is a not uncommon complication of generalised seborrhœic dermatitis, particularly when the axillæ are involved, or of infectious eczematoid dermatitis.

**Treatment.**—The measures already indicated for the treatment of the seborrhœic state and of staphylococcal infections should be instituted. Locally, the axillæ should be painted daily with R. Tinct. Iodi. mitis 1·0, Spirit methyl. ad 10·0, and a boro-salicylic dusting powder applied freely.

## GRANULOMA PYOGENICUM

(*Botryomycosis hominis*)

This term has been applied to small, pedunculated tumours of the skin similar to those which sometimes develop in horses in the wound left by castration. They were thought to be due to infection with a special fungus, the Botryomyces, but the mulberry-like masses which were mistaken for parasitic elements are really degenerated cells. The tumour varies in size from a pea to a nut. It is red in colour, round or slightly flattened vertically, and its surface is smooth or mamillated. It is not, as a rule, covered by epidermis but rests on the dermis, from which it is separated by a groove, at the bottom of which, on drawing the tumour to one side, the pedicle can be seen. It is firm and elastic to the touch, and bleeds easily; sometimes it is covered with a yellowish crust. The lesions

are seen most commonly on the hands and fingers, but may also occur on the face, feet, legs, lips, scalp and even the tongue. Unless removed they tend to persist indefinitely. Histologically they consist of embryonic connective tissue with large numbers of newly-formed, friable capillary blood vessels, such as are seen in an angeioma.

**Ætiology.**—The tumours occur on the site of an abrasion or wound, which has suppurated some weeks or months previously. Irritation by caustics would often seem to be a factor predisposing to this development. The *Staphylococcus aureus* appears to be the infecting organism responsible for their production.

**Treatment.**—The lesion should be removed, and the base cauterised to prevent recurrence. The local application of the ultra-violet rays has been found to cause their disappearance.

The diseases of the skin due to infection with bacilli are as follows :—

| | |
|---|---|
| Comedo . . . | *B. acnes* (p. 935). |
| Tuberculosis . . | *B. tuberculosis.* |
| Leprosy . . . | *B. leprœ* (p. 133). |
| Glanders . . . | *B. mallei* (p. 136). |
| Anthrax . . . | *B. anthracis* (p. 137). |
| Diphtheria. . . | *B. diphtheriæ* (p. 66). |
| Soft sore . . . | Strepto-bacillus of Ducrey-Unna (p. 954). |
| Rhinoscleroma . . | Bacillus of Frisch (p. 955). |

## TUBERCULOSIS

Tuberculosis involves the skin in several ways, and the various forms of cutaneous tuberculosis may be classified as follows :—

(1) *Miliary tubercles* in which the skin, like other organs, is affected by a general miliary tuberculosis.
(2) *Lupus vulgaris.*
(3) *Lupus verrucosus seu verruca necrogenica.*
(4) *Scrofuloderma.*
(5) *Tuberculous ulcer* } usually treated in books on surgery.
(6) *Tuberculous gumma* }

These last five forms are due to the actual growth of the tubercle bacillus in the skin or subcutaneous tissue.

(7) *The Tuberculides,* of which there are several varieties, and which may be regarded as cuti-reactions in a person already sensitised to tubercle, provoked by endogenous inoculation of the dermis or hypodermis by tubercle bacilli, carried in the blood stream from some distant tuberculous lesion.

(8) *Lupus pernio and the Sarcoids,* which together represent the cutaneous manifestations of the *Benign Lymphogranuloma of Schaumann,* the tuberculous origin of which is not yet absolutely proven.

### LUPUS VULGARIS (*See Plate XLVII., B*)

This, the most familiar form of tuberculosis of the skin, is happily becoming much less common, and it is comparatively rare now to see the terrible mutilations that the disease is capable of producing. The primary lesion of lupus is a characteristic miliary nodule, embedded in the skin and representing histologically

a minute infective granuloma ; it may conveniently be called a *lupoma*. In size it ranges from that of a pin's head to that of a large pea ; in colour it varies from yellowish- to brownish-red, with sometimes a bluish tinge ; it may form a raised papule or nodule on the surface of the skin, or may not project at all ; it has a translucent look and is soft in consistency, being easily pierced by a sharpened match. A patch of lupus may begin by the appearance of a single nodule of this kind, which gradually enlarges, while near it others are formed, or by two, three or more arising simultaneously. The patch spreads by the enlargement of individual nodules and the formation of new ones, and there is a tendency for these isolated lesions gradually to coalesce. Under vitropression lupus nodules present a very characteristic appearance. They look, as Hutchinson aptly said, like specks of apple-jelly embedded in the skin, and their translucency is due to the local disappearance of the elastic and connective-tissue network.

If left untreated a patch of lupus tends to undergo one of two changes. In some cases spontaneous cicatrisation takes place in the centre, while the patch tends slowly to extend at the borders by the formation of fresh nodules. But in the central, soft, white scar a few nodules persist or reappear, and this characteristic feature is of importance in the differential diagnosis between lupus and the serpiginous syphilides. In other cases the overlying epidermis is destroyed, so that there is ulceration with secondary pyogenic infection and the formation of impetiginous crusts.

There is very considerable variety in the clinical appearances of the disease according to the predominance of one or other of the above processes. Thus in some cases the nodules are small and superficial, and the patch is red, scaly, and projects hardly or not at all above the level of the surrounding skin. This type (*L. erythematoides*) is often mistaken for lupus erythematosus ; on vitropression the characteristic apple-jelly nodules may be seen, but sometimes are only recognisable on microscopical examination. In other cases the nodules are large, confluent and raised to form a tumour (*L. tumidus* or *hypertrophicus*). Sometimes, probably as the result of secondary pyogenic infection, an ulcerating patch develops papillomatous vegetations, soft at first and later becoming warty, thus resembling lupus verrucosus (*q.v.*) (*L. papillomatosus*). Patches which ulcerate at the margin, while undergoing spontaneous cicatrisation in the centre, imitate ulcerating syphilides (*L. serpiginosus*). Formerly the non-ulcerating forms of lupus were termed *L. non-exedens*, the ulcerating *L. exedens*, but this distinction is somewhat artificial. The most malignant type of ulcerating lupus—*L. vorax seu phagedaenicus* is fortunately rare ; it may rapidly produce the most frightful destruction and deformity.

The course of lupus is essentially chronic and its spread insidious. Although spontaneous involution with scarring commonly occurs, the disease extends peripherally, fresh nodules arise in the scarred area, and, except in *lupus disseminatus*, it is exceedingly rare for a natural cure to result.

*Distribution.* Lupus affects both the skin and mucous membranes. It may spread from the skin to the mucous membranes, but, as Audry insists, it is probable that the reverse is true far more commonly than was originally supposed. On the skin, the face is the commonest site (78 per cent. according to Sequeira). Emlyn Jones found that the disease started about the nose in 28 per cent., and in other parts of the face, including the auricles, in 48·4 per cent. In 1·8 per cent. the lips, in over 13 per cent. the neck, and in 8·5 per cent. the trunk or extremities were the primary sites. It is rarely seen on the scalp, forehead, upper eyelids, genitals, axillæ, or on the palms and soles.

The *mucous membranes* are affected in a large percentage of cases (43·25 per cent. among Sequeira's cases, 80 per cent. in Christiansen's series at the Finsen Institute). The nose, mouth, palate, pharynx, larynx, tongue, nasal duct, lachrymal sac, conjunctiva, and middle ear may be involved. The nose is by far the commonest site, and is frequently the primary source of involvement of

the skin. Sometimes the infection spreads up the nasal duct to the lower eyelid and conjunctiva, or from a primary tuberculous lesion of the conjunctiva it may spread downwards and invade the nasal mucous membrane and thence the skin. On the palate, pharynx, and in the mouth lupus appears as a pinkish-red, mamillated patch, with here and there small ulcers, which may extend until the whole area is ulcerated and crusted. On the gums it produces swollen, red, fleshy granulations, and the teeth often become loose. On the tongue, a rare site, a papillomatous patch is the rule, but ulceration may occur.

**Ætiology.**—The disease is due to the actual growth in the skin or mucous membrane of the tubercle bacillus, which may be either of a human or bovine strain, the proportion varying in different countries. From the researches of Griffith and others it is evident that in most cases the strain, whether human or bovine, is of low virulence. Although lupus may begin at any age, in more than half the cases it appears before the tenth, and in over 80 per cent. before the twentieth year. It is more than twice as common in females than males, and occurs far more in Northern Europe than in the South, partly no doubt because of the lack of sunlight, and is comparatively rare in the Colonies, the tropics, and in America. It affects, as a rule, scrofulous persons, who have been infected with tubercle in early life, and a strong family history of tuberculosis is often obtainable. It is almost invariably associated with tubercular adenitis, and sometimes with tuberculosis of bones, joints, or the peritoneum. As Besnier pointed out, phthisical persons do not develop lupus, whereas patients with lupus frequently become phthisical.

As regards the actual mode of infection of the skin, this may occur (1) directly from without; (2) by extension from deeper tubercular foci or from mucous membranes; (3) through the blood-stream. The first method is probably common, and doubtless accounts for the frequency of the disease in or around the nose, the organism being carried by dust or by the fingers; cases in which it has started in vaccination or tattoo marks, or after piercing the ears for ear-rings, have been recorded.

The second method is instanced by the occurrence of lupus in the skin over tuberculous glands, which have broken down or been excised, and by the spread of infection from the nasal mucous membrane, or *viâ* the nasal duct from the conjunctiva. The third method accounts for the multiple disseminated patches of lupus that appear most commonly after measles or whooping-cough, and it is probable that infection of the skin through the blood-stream is commoner than is usually believed.

**Morbid Anatomy.**—The epidermis is usually atrophied, and beneath lies a mass of tubercular tissue, consisting of lymphocytes, epitheloid and plasma-cells, and here and there typical giant cell systems. There is destruction of the elastic and connective tissue, and of the blood vessels, and the newly-formed tissue undergoes degeneration without actual caseation. The histological picture, of course, varies according to the type of the disease and the presence or absence of ulceration and secondary infection. Tubercle bacilli may be demonstrated, but, as a rule, only after prolonged search through serial sections.

**Diagnosis.**—The conditions for which lupus is likley to be mistaken are tertiary syphilis, rodent ulcer, nodular leprosy, lupoid sycosis, certain forms of sarcoid, and lupus erythematosus; it is not uncommonly from carelessness confused with impetigo, impetiginised eczema and psoriasis.

From a tertiary syphilide it may be distinguished by the age of onset, by the length of history (since, with the exception of lupus vorax, lupus takes many years to produce lesions of the same extent as a syphilide of a few weeks or months standing), by the soft consistency of its nodules and their apple-jelly appearance on vitropression, and by the presence of nodules in the central scar. Necrosis of bone is never caused by lupus. The Wassermann reaction should always be done, even if the diagnosis of lupus is certain, since there appear to be mixed cases—

usually lupus in a patient with congenital syphilis—in which antisyphilitic treatment leads to striking improvement.

From lupus erythematosus the diagnosis may be difficult in certain cases. The chief points of distinction are the symmetrical distribution of lupus erythematosus (although by gradual spread lupus vulgaris may produce a bilaterally symmetrical patch over the nose and cheeks); the involvement of the scalp, edges of the ears, and fingers; the absence of ulceration and non-involvement of cartilage; and the different appearances on vitropression.

The small nodular sarcoid may imitate lupus very closely, but the nodules are harder, the histology is characteristic, and guinea-pig inoculations are always negative.

The age of onset, the hard, pearly, nodular edge, and the clean-cut ulceration distinguish rodent ulcer from lupus.

**Complications.**—Apart from the disfigurement and mutilation that the disease may cause, the chief complications that occur are erysipelas and squamous-celled carcinoma. An attack of erysipelas may have a beneficial and even curative effect, as in the cases described by Hallopean and Sequeira. Although carcinoma is a rare complication of lupus, particularly in women, its incidence has greatly increased since the introduction of the X-rays, the long-continued application of which is entirely unjustifiable in this disease.

**Treatment.**—It must be remembered that the vast majority of patients with lupus have other tuberculous foci apart from the skin, and that a considerable proportion die from visceral tuberculosis. For this reason general treatment directed towards raising the patient's resistance in every possible way should be instituted. Heliotherapy, as carried out by Rollier at Leysin, is the method of choice, and is undoubtedly superior to any other known therapeutic measure. In England and countries which obtain relatively little sunshine excellent results are achieved with the carbon-arc and mercury-vapour lamps. At Alton true heliotherapy is combined with the artificial light baths. The introduction of this treatment has revolutionised the therapy of lupus and other forms of chronic tuberculosis, and has rendered unnecessary many of the local measures that were formerly employed. In addition, the internal administration of iodine and cod-liver oil is of great value. The former should be given in the form of a 10 per cent. solution in rectified spirit, of which 5 drops in milk may be taken at first twice daily, the dose being gradually increased up to as much as 60 drops *per diem* if well tolerated. Injections of tuberculin, if given prudently over a long period, are undoubtedly beneficial. Remarkable improvement is also sometimes obtained with intramuscular injections of calomel, but the results are inconstant.

**Local Treatment.**—Before employing any local measures for the destruction of the lupus tissue, it is advisable, in view of the remarkable results now being obtained by heliotherapy or the artificial light baths, to observe to what extent spontaneous healing will take place under such treatment combined with the internal administration of iodine and cod-liver oil. From the cosmetic point of view local phototherapy with the Finsen arc lamp undoubtedly gives the best results, the resulting scars being smooth and supple. The method is, however, very tedious and expensive, and cannot be employed in the mucous membranes. The use of the X-rays should, as is now generally admitted, be reserved for the ulcerating, vegetating or papillomatous types of the disease. It may be combined with the application of the galvano-cautery or with periodical scarification. Large doses of X-rays or their continued use over long periods is entirely unjustifiable owing to the risk of a subsequent radiodermatitis with ultimate epitheliomatous degeneration of the scar. The thermo-cautery and galvano-cautery are useful adjuncts to treatment, and are of particular value in lupus of the mucous membranes. Recently, diathermy has been employed with excellent effect. Scarification, if correctly performed, gives scars hardly less satisfactory than those obtained with the Finsen lamp, but it is a tedious and painful method; it

is, however, the method of choice for rapidly extending, vegetating lupus (*L. vorax*), and for the treatment of nodules embedded in scars. In France the filiforme douche is being used with good results for the destruction of lupus tissue. Various caustics, such as arsenious acid, pyrogallic acid, trichloracetic acid, the liquid acid nitrate of mercury, and a combination of salicylic acid and creosote in plaster form have a selective action on lupus tissue, but cause considerable pain, particularly arsenious acid. Adamson's method of employing the liquid acid nitrate of mercury is simple and gives excellent results. It is carried out as follows :—The lupus nodules are gently rubbed for from one to two minutes with a piece of cotton wool twisted round the closed ends of a finely pointed pair of forceps, and moistened with the liquid. No subsequent dressing is necessary, although it is perhaps advisable to keep the treated surface covered with a piece of clean lint or gauze. A good deal of inflammatory reaction ensues, and a scab forms, which becomes detached in course of time, leaving a smooth pink surface. The subsequent scar compares favourably with that obtained by any other method, except, perhaps, the Finsen light. The method is rather painful, but an anæsthetic is not usually required. A considerable area can be treated at one sitting, and the applications can be repeated until all the active lupus tissue has been destroyed. In cases in which the nodules are large and deep, they may first be scraped under anæsthesia, and then treated with the acid nitrate of mercury or zinc chloride.

### LUPUS VERRUCOSUS

(*Verruca necrogenica*)

This variety of tuberculosis of the skin is caused by direct inoculation of tubercle bacilli. It may arise in phthisical or other patients suffering from "open" tuberculosis as a result of auto-inoculation. More commonly it is met with in medical men, nurses, or students in contact with tuberculous patients, or in pathologists, *post-mortem* attendants, veterinary surgeons, butchers, and others who handle human or animal cadavers infected with tubercle. The lesions contain considerable numbers of tubercle bacilli, far more than those of lupus vulgaris, but less than tuberculous ulcers. They are met with usually on the hands, particularly on the thumb, but they also occur on the elbows, knees, feet, buttocks, peri-anal region, neck, and even on the face. The earliest lesion is a small erythematous nodule from which, later, a bead of pus can be expressed ; it gradually enlarges to form a warty plaque surrounded by a bluish halo of erythema. In a fully-developed patch three zones can be recognised : at the periphery an erythematous area, within this a number of papillomatous nodules of a bluish-violet colour covered with adherent crusts, and in the centre either a depressed scar or brownish warty projections separated by fissures. A small quantity of pus, in which tubercle bacilli may be found, exudes from the surface, and the base of the plaque is indurated. The lymphatic glands are infected early, and the viscera may become involved.

**Treatment.**—The method of choice is excision of the affected area of skin as soon as the diagnosis is established, or total destruction of the lesion with the actual cautery. Sequeira recommends removal of the warty surface with pyrogallic or salicylic acid, followed by the application of X-rays.

## THE TUBERCULIDES

In 1896, Darier introduced the term "tuberculides" to designate a group of eruptions which occur in persons having a manifest tuberculous infection, and which, although differing from each other in their clinical appearances, are frequently co-existent in the same patient, thus indicating their relationship and

common origin. The following classification of these eruptions has been suggested by Darier :

A. Cutaneous Tuberculides, in which the lesions are dermo-epidermic.
   1. *Lichenoid Tuberculides.*
      Lichen scrofulosorum.
      Lichen nitidus.
   2. *Papulo-necrotic Tuberculides.*
      Folliclis.
      Acnitis.
   3. *Acneiform Tuberculides.*
      Acne cachecticorum, a variety of the preceding.
   4. *Lupoid Tuberculides.*
      Benign Sarcoid of Boeck.
   5. *Erythemato-atrophic Tuberculides.*
      Lupus erythematosus.
      Certain atrophodermias.
   6. *Erythematous Tuberculides.*
      Lupus erythematosus disseminatus.

B. Subcutaneous Tuberculides.
   Erythema induratum scrofulosorum.
   Subcutaneous sarcoid of Darier-Roussy.
   Disseminated nodular sarcoid.

In the case of some of these dermatoses, however, actual proof of their tuberculous nature is lacking. The undoubted tuberculides are : *Lichen scrofulosorum*, *Papulo-necrotic tuberculide*, *Erythema induratum scrofulosorum*.* *Lupus erythematosus* is certainly not always a tuberculide, although it may be in some cases.

**Ætiology of the Tuberculides.** The tuberculides result from the embolism in a blood vessel either of the skin or subcutaneous tissue of tubercle bacilli carried in the blood-stream from some local tuberculous lesion. They occur in persons who have been usually, if not always, sensitised to tubercle by an infection in early life, and whose resistance to the infection is, as a rule, good. The actual lesions represent inflammatory reactions produced by the interaction of the ant bodies present in the sensitised tissues and the bacilli, this reaction bringing about destruction of the latter, so that the lesions heal with or without scar formation. It is obvious that for their occurrence the presence of antibodies is essential, and this probably explains why disseminated patches of true lupus may follow an attack of measles in a tuberculous person, since measles causes a temporary disappearance of tuberculous antibodies, so that the bacilli survive and multiply. An important factor in the genesis of tuberculides is circulatory stasis ; the papulo-necrotic variety is seen in scrofulous persons with a chilblain-circulation, the sites of election being those in which the blood flow is most stagnant, and erythema induratum occurs almost exclusively in young females with cyanotic, œdematous-looking legs. As might be expected, owing to the rapid destruction of the bacilli, it is only rarely that they are demonstrable in tuberculide lesions. They have, however, been found in lichen scrofulosorum and erythema induratum. Inoculation of guinea-pigs with tissue from the lesions has more often given positive results. Injections of tuberculin not only give rise to focal cutaneous reactions, but may provoke an outbreak in a tuberculous person of tuberculides not previously present. Moreover, minute doses of

* Schaumann considers that *the sarcoid of Boeck and Lupus pernio* which, as he has shown, are merely the cutaneous manifestations of his "benign lymphogranuloma," are caused by infection with a bovine strain of the tubercle bacillus, but this view is not universally accepted. Definite evidence is wanting as to the cause of *lichen nitidus*, though the histological appearances of the lesions are in favour of its tuberculous origin.

tuberculin, given at suitable intervals, have a striking curative effect in erythema induratum and the papulo-necrotic tuberculides.

Apart from the types of tuberculide already referred to, mention must be made of the eczema scrofulosorum of Boeck, and of the probable tuberculous origin of erythema nodosum and even of erythema multiforme in rare cases.

*Eczema scrofulosorum* is seen in young children with glandular and other forms of tuberculosis. It occurs as characteristic eczematous plaques, usually of a pinkish-brown colour, dry and scaly or oozing and crusted, situated most commonly on the face, trunk, and limbs. Definite evidence of active tuberculosis was found in over 80 per cent. of eighty cases (**6**). The eruption is not infrequently accompanied or followed by lichen scrofulosorum.

*Erythema nodosum*, as has been indicated, is usually a manifestation of sensitisation to a streptococcus, but it is also one form of trichophytide, and strong evidence has been adduced by Landouzy, Gougerot and others that it may in certain cases be a tuberculide.

### LICHEN SCROFULOSORUM

An eruption consisting of small papules, at first red or pink, later fawn-coloured or almost yellow, arranged in roundish groups, or circles, or segments of circles. On the older papules a minute scab is formed, and after a time the papules subside and leave only a yellowish pigmentation. They occur on the trunk, especially at the sides, and rarely on the limbs; the occurrence on the limbs is more frequent in children than adults. Itching is absent or very slight. The disease progresses by the appearance of fresh crops of papules from time to time, so that it may last for months or years.

Many patients have enlarged lymphatic glands, caries, or other bone lesions, or ulceration of the skin; but phthisis is not common. It occurs in both sexes, but has been seen more often in males. It is most common in children, and rare after early adult age.

The papules have a characteristic tuberculous structure with epithelioid cells and giant cells, but the tubercle bacillus cannot always be demonstrated.

### ERYTHEMA INDURATUM

(*Bazin's Disease*)

This form of cutaneous tubercle occurs most often in adult females in feeble health, and affects especially the lower and outer back part of the legs. Deep-seated indurations, the size of peas or nuts, form under the skin; at first they can only be felt, but afterwards encroach on the skin, and cause dusky red or purplish projections. They may disappear, and reappear from time to time, or they break down into ulcers with surrounding œdema. They may be mistaken for gummas, and exceptionally they may be independent of tubercle, as shown by the failure of the tuberculin test and the absence of bacilli.

### FOLLICLIS

(*Papulo-necrotic Tuberculide*)

This name is given to an eruption of scattered round papules, situate in the deeper part of the skin, and feeling like shot. They occur in any part of the body, but especially on the hands and feet. They vary in size from that of a pin's head to that of a lentil, and are dark red or purple in colour. They may disappear spontaneously, or they may form pustules, and small ulcers covered by crusts, which on healing leave pitted scars.

**Treatment.** Since the presence of the undoubted tuberculide eruptions is certain evidence of active tuberculosis in some part of the body, usually in

lymphatic glands, bones, or peritoneum, their general treatment is that of the underlying infection, as in lupus vulgaris (*q.v.*). Darier recommends the long-continued administration of tuberculin, combined with small doses of arseno-benzol. In erythema induratum rest in bed until the lesions have disappeared is advisable, particularly when ulceration has occurred.

## SOFT SORE

(*Soft Chancre, Chancroid*)

A soft sore or chancre is a round or oval ulcer, which develops very rapidly after a short incubation period, and is auto-inoculable. At the site of invasion by the specific infecting organism (Bacillus of Ducrey-Unna) a small inflammatory papule appears within twelve to twenty-four hours, which soon becomes converted into a pustule on a bright red base. On rupture of the pustule, the characteristic ulcer is formed—*i.e.*, within four or five days from the date of inoculation. *The base* of this ulcer consists of greyish granulations, covered with a yellow, creamy, purulent film; *the edges* are sharply cut or undermined, and careful examination will show slight crenation—a valuable diagnostic sign; *the base* is œdematous, but not indurated like that of a chancre; *the periphery* of the lesion is bright red. The ulcer exudes a purulent secretion, from which inoculation of adjacent areas of skin takes place. It is, therefore, rare to see a single lesion, multiple ulcers, arising either from simultaneous inoculation at more than one point, or from auto-inoculation from an original single sore, being the rule. By the confluence of several lesions, the size of which varies from a pea to a finger-nail, a considerable area of ulceration may result.

The neighbouring *lymphatic glands*, on one, or more rarely, both sides, are, as a rule, enlarged and painful within a few days from the appearance of the sore or sores, and there is a tendency for one gland in particular to be involved, which is very apt to soften and break through the skin, producing the soft or chancroidal bubo.

*The situation* of soft sores varies, but the vast majority are seen on the genitals and adjacent parts. In men the prepuce, the corona and cervix, and the frenum, which may be perforated, are most commonly affected; in women, the vestibule, the fourchette, the labia minora, the glans clitoridis, and the anus. By auto-inoculation, neighbouring regions, such as the pubis, internatal cleft, and inner surfaces of the thighs, may be involved. Extra-genital soft sores are excessively rare, and usually, though not invariably, arise by auto-inoculation from genital lesions.

**Complications.**—Reference has already been made to suppurating bubo; in addition lymphangitis, abscess of the dorsal lymphatics of the penis (bubonulus), phimosis, paraphimosis, phagedena, and gangrene may result. Phagedena arises both from the actual sores, and also from a suppurating bubo.

**Ætiology.**—The cause of soft sore is a strepto-bacillus described by Ducrey and Unna. It is a short gram-negative bacillus, easily stained by methylene-blue. It is with difficulty grown artificially, but has been cultured on peptonised human skin and certain other special media. The organism may be successfully inoculated on monkeys. Infection usually takes place directly by sexual intercourse, but may rarely be indirect.

**Diagnosis.**—The chief characteristics of soft sore are the short incubation period, the tendency to rapid ulceration, the auto-inoculability and the resulting multiplicity of the lesions, the early involvement of the lymphatic glands and their liability to suppuration, the lack of induration of the base, the crenated edge of the ulcer, and the painful course of the affection. It must be distinguished from a syphilitic chancre, herpes progenitalis, the lesions of scabies, mucous patches, tertiary gummatous ulceration, and epithelioma.

The following differences between hard and soft chancres should be noted (Pusey) :—

| *Hard Chancres* | *Soft Chancres* |
|---|---|
| Incubation period two to five weeks. | Incubation period two or three days ; becomes an ulcer in four or five days. |
| An erosion or superficial ulcer, with sloping edges and scant secretion. | An angry-looking ulcer with a bright-red halo, undermined edges, crenated border, and profuse purulent secretion. |
| Indurated cartilaginous base. | Slight inflammatory induration. |
| Is almost painless. | Often very painful. |
| Is not auto-inoculable. | Is auto-inoculable, and therefore multiple lesions of different ages and sizes are common. |
| If multiple, due to multiple primary inoculations, the lesions being of the same age. | |
| Adenitis is bilateral. | Adenitis usually unilateral. |
| Adenitis indolent and painless ; the enlarged glands are multiple and discrete. | Adenitis acute, painful and inflammatory, and likely to result in suppurating bubo. |

It must be remembered, however, that a simultaneous inoculation with Ducrey's bacillus and the *Spironema pallidum* may occur, and for this reason every soft sore should be considered as potentially a hard chancre. Frequent and persistent examinations for the spironema of serum taken from *the edges* of the ulcer, after a preliminary cleansing of the surface, should be made.

**Treatment.**—The patient should, if possible, be kept at rest, since exercise favours suppurative adenitis. The ulcer should be cleaned several times daily with hot solutions of perchloride of mercury (1 in 2,000), potassium permanganate (1 in 1,000), or carbolic acid (1 or 2 per cent.), and iodoform or aristol powder then applied freely. Another method is to destroy the ulcer by swabbing it thoroughly with pure carbolic acid, applied with a swab of cotton-wool ; an acute inflammatory reaction occurs, and a slough is formed and shed, leaving a healthy wound.

## RHINOSCLEROMA

This is a dense infiltration of the septum and alæ of the nose, rendering it as hard as ivory, thick, and rigid. The surface is smooth or irregular, the colour normal or brownish red ; the mucous membrane is affected as well, and the orifices may be blocked by its swelling. Not infrequently the adjacent upper lip, and sometimes the cheeks, are involved. The changes are a dense infiltration of the corium and papillæ with plasma cells, the presence of large translucent degenerated cells (Mikulicz cells), and bacilli, the bacilli of Frisch, which have a close resemblance to the pneumonia bacilli of Friedländer, and are found chiefly in the Mikulicz cells, but also in the plasma cells and tissue. Removal by the knife or destruction by caustics may be required, but X-rays should be tried first.

## DISEASES OF THE SKIN DUE TO INFECTION WITH THE HIGHER FUNGI

The diseases of the skin caused by infection with the higher fungi may be classified as follows :

(1) *Epidermomycoses.*

| | |
|---|---|
| Favus . . . | *Achorion Schönleinii.* |
| Microsporosis . . | Microsporon Audouini. |
| | ,, lanosum vel caninum. |

| | |
|---|---|
| Microsporosis . . | Microsporon felinum. |
| | ,, equinum. |
| Trichophytosis . . | Trichophyton, of which there are about thirty species. |
| Epidermophytosis . | Epidermophyton inguinale. |
| Epidermycosis of the tropics. | |
| Pityriasis or Tinea versicolor. | Microsporon furfur. |
| Erythrasma . . | Microsporon minutissimum. |
| Lepothrix. | |

(2) *Dermatomycoses.*

| | |
|---|---|
| Actinomycosis . . | Actinomyces bovis (p. 140). |
| Mycetoma pedis (Madura foot) | Streptothrix maduræ (p. 143). |
| Sporotrichosis . . | Sporotrichum Beurmanni (p. 143). |
| Blastomycosis . . | Blastomyces. |

### FAVUS

In this disease, rare in England, the fungus attacks the epidermis and the hair follicles; it may at first form patches like those of ordinary ringworm, but soon there appears a small, bright yellow, circular disc, with a depressed centre and a gradually thinning margin. This, the "favus cup," is caused by the fungus elements separating the layers of the epidermis and lifting them up, except at the central point where the hair follicle joins the skin. This characteristic lesion may occur on the scalp, or on any other part of the body, the forearm for instance, determined by contagion; and it is conveyed not only from man to man, but to man from domestic animals—rabbits, dogs, cats, and others. When numerous cups have formed they become aggregated together, and form a thick continuous yellow crust, with an irregular honeycombed surface, giving off an offensive odour resembling that of mice. The hair sacs are destroyed, the hairs fall out, and baldness results; moreover, the favus masses often become a nidus for pediculi, and eczema and impetigo complicate the original lesion. If the masses are examined under the microscope after soaking in liquor potassæ, the mycelium and spores (conidia) of the *Achorion Schönleinii* are seen. The conidia are larger and more varied than those of the ringworm fungi, and the mycelium is shorter and more jointed.

The *nails* may be also invaded by the fungus of favus. They present an appearance similar to that in ringworm (*see* p. 958), and sometimes a distinct cup forms under the nail. The nail may be examined in the same way as in ringworm.

Two other species of achorion have been seen in man in very rare cases: *Achorion Quinckeanum*, the fungus of mouse favus, and *Achorion gypseum*.

**Treatment.**—The crusts must be softened by oil or poultices and removed; parasiticides and epilation are needed here, and for most cases the Röntgen rays should be used as they are for ringworm. The nails, if affected, may be treated in the same way as when they are diseased by the microsporon or trichophyton. The disease is very obstinate, and after apparent cure often breaks out again. It is well to care for the general health by good food and tonic medicines

### RINGWORM (*See Plate XLVIII., D*)

The diseases which are commonly included under this term are ringworm of the head (*Tinea tonsurans*), ringworm of the body (*T. circinata*), ringworm of the beard (*T. sycosis*), and Burmese ringworm (*T. marginata*). They are dependent upon the growth of three or four closely allied fungi. It will be better

to describe the clinical features of the various forms first, and state what is known of the organisms afterwards.

*Tinea tonsurans* (*Ringworm of the head*). This disease is the great scourge of schools and allied institutions among the poorer classes. It is frequent in children, rare in infancy, and not easily caught by adults. It spreads by contact, and by the use of hats, caps, brushes, combs, and towels used in common.

It generally first appears as a round patch, on which the growth of hair is much thinner than elsewhere. On close examination the skin is seen to be pink, perhaps a little swollen, and covered with minute branny scales. Besides the thinly scattered long and healthy hairs are seen a number of broken stumps of hair, opaque, black or dark brown in colour, twisted and bent. If an attempt be made to extract one of these broken hairs with a pair of forceps, it will almost certainly break off short; if then moistened with a drop of liquor potassæ and placed under the microscope, the condition is explained. The substance is quite opaque, the natural structure of the hair is unrecognisable, and the hair seems to be converted into a mass of fungus spores (conidia). This is, however, only a sheath of spores surrounding the hair, which is itself occupied chiefly by mycelium tubes running in a longitudinal direction. These may be more readily observed in hairs which are less completely diseased.

The patch spreads by the implication of hairs at its circumference, and fresh patches form in other parts of the scalp. As these enlarge they become more completely denuded of long hair, though they nearly always present a considerable quantity of the short stumps which have been described; and these may be surrounded and mixed with scabs, crusts, or sebaceous matter, or with a fine whitish powder, of which probably the fungus elements form a part. The patches spread slowly; some may heal in the centre as they extend at the edges; or the patches may coalesce, and nearly the whole scalp may be affected. Sometimes, on the other hand, one or two patches persist, without improvement, but without spreading. The disease may last for years, but eventually dies out, and the hair is perfectly restored. There is rarely much inflammation; but occasionally the hair follicles inflame, coalesce, and form a red or pink swelling which is soft and boggy to the touch, and discharges pus from a few points; the hairs are loosened and fall out, and the patch may remain bald when the other parts of the scalp have recovered. The condition is called *kerion*.

Probably the fungus invades the hair close to the scalp, and pushes down towards the bulb. The hair in the follicle is thus weakened or destroyed, and as it is forced outwards by the newly formed epithelial plates, it breaks off. The newly formed epitheluim is, in its turn, invaded as soon as it gets into the horny condition. It was shown by Sir Frederick Taylor and Thin that the fungus only invades the hair itself, and cannot be found in the cells of the root sheath or in the structures of the hair follicle.

*Tinea circinata* (*Ringworm of the body*).—This occasionally co-exists with tinea tonsurans, but often occurs alone. The fungus invades the epithelial scales and downy hairs of the skin and produces a circular patch, from ½ to 1 inch in diameter, slightly raised above the surface, sharply defined, pink in colour, often papular, and covered with fine scales. If the surface be scraped with a scalpel, and the scrapings be placed, with a little liq. potassæ, on a glass slide, the mycelium and spores of the fungus will be seen. The patches increase by extension at the circumference, and may as they spread heal in the centre. Occasionally a few vesicles form on the surface from the irritation of the parasitic growth, thus in part justifying the former name, herpes circinatus. They occur on the face, neck, and arms most frequently; they are, as a rule, few in number, and may be solitary.

*Tinea marginata* (*Tinea cruris*, *Eczema marginatum*, or *Burmese Ringworm*).—This form of ringworm, due to the *Epidermophyton inguinale*, of which at least three varieties exist, is very common in tropical countries, where it is known as dhobie (washerman's) itch, owing to the fact that it often results from infec-

tion of the underclothing during washing. It occurs chiefly in the groins, but may also involve the axillæ, and may spread from the groins backwards over the perineum and buttocks and upwards over the lower part of the abdomen. It may begin like an acute intertrigo, but more commonly the primary lesions consist of a few circinate patches, which coalesce to form red symmetrical, semicircular areas, with sharply defined and slightly raised edges, on the inner surfaces of the thighs and the contiguous parts of the scrotum. As the disease spreads at the margins, recovery tends to occur in the centre. The lesions are much more extensive, more inflamed, and more obstinate than those of tinea circinata. The disease has become much commoner in England of late since the War, and it often occurs in epidemics in schools, colleges, and other institutions. The usual source of infection is doubtless the water-closet seat.

The epidermophyton fungus is also responsible for a very important condition known as *eczematoid ringworm of the extremities*, in which the infection involves the toes, the soles, the fingers, the palms, and sometimes the nails. On the feet it usually begins as indolent, scaly patches between the toes, spreading outwards on the lower and sometimes the upper surfaces towards the heads of the metatarsals; the edges consist of the raised horny layer, while the skin within them appears reddened and glazed or actually raw. Frequently eczematous vesicles are formed, so that the condition resembles pompholyx, and sometimes recurrent acute attacks of eczema occur, usually in hot weather, with much inflammation and the formation of numerous vesicles and bullæ. Secondary streptococcal infection may result and lead to lymphangitis spreading up the leg. Most cases of so-called intertrigo of the toes and many cases of eczema of the feet are really examples of eczematoid ringworm, and in a doubtful case the under-surfaces of scales from the margins of the patches and the roofs of vesicles should be examined under the microscope for the fungus. On the hands the disease may resemble an erythemato-squamous eczema of the palms, or it may occur at the roots and sides of the fingers, as on the toes, and may spread upwards to their dorsal surfaces. Acute attacks resembling pompholyx are not infrequent, especially in hot weather. It is to the researches of Whitfield and Sabouraud that we owe the recognition of the parasitic origin of this form of eczema of the hands and feet, and the discovery is of great importance, since many unrecognised cases have been, and still are, treated for years as gouty eczema, or intertrigo, or pompholyx.

*Tinea sycosis* (*Ringworm of the beard, Hyphogenic sycosis*).—The hair follicles of the chin and cheeks are here inflamed by the presence of the fungus; they suppurate, and the hairs become loosened. Induration and swelling of the intermediate skin also occur. It differs from sycosis, already described (*see* p. 941) in that it first attacks the hairs, and loosens them early, so that their extraction is painless; it spreads more rapidly and produces deeper infiltration. Microscopic examination shows the fungus, in which the mycelium is more abundant than the spores.

*Tinea of the nails.*—The invasion of the nails by a fungus is called *onychomycosis*, and this may be a ringworm or favus, either of which is probably conveyed by scratching other affected parts. The nail becomes elongated and curved over the end of the finger, with a thick edge, rough, uneven surface, and dirty yellow colour; it is also brittle and readily splits. If fragments or scrapings are soaked with liq. potassæ, and examined under the microscope, chains of spores of the fungus are seen.

**The Fungi of Ringworm.**—The character by which these fungi are differentiated is the size of the spores. *Tinea tonsurans* is in England chiefly due to a variety in which the spores are small, measuring from 2$\mu$ to 4$\mu$. It is called *Trichophyton microsporon*, or *Microsporon Audouini*. As usually seen, the shaft of the hair is penetrated by the mycelial threads, and the spores form a dense sheath, five or six spores deep, around the hair; and this sheath is con-

tinued beyond the follicle on to the stump or fragment of the hair which still remains. The common ringworm, caused by the *microsporon*, is almost confined to children, is very contagious and very obstinate, so that months may elapse before it is cured; but it ultimately gets quite well.

The other ringworm fungus is named *Trichophyton megalosporon*. The spores measure from 3$\mu$ to 6$\mu$, commonly 5$\mu$, and are thus larger than those of microsporon; they attack the root of the hair first and grow upwards. Two varieties have been described: in one, *Trichophyton endothrix*, the spores and mycelium are contained entirely within the shaft of the hair; in the other, *T. endo-ectothrix*, the spores form a sheath outside the hair (as they do in microsporon), and only the mycelium tubes lie within it. But these distinctions are not universally accepted. Cases due to megalosporon are much more amenable to treatment, and those due to the endo-ectothrix variety more than those due to endothrix. As to the further relations between the fungi and the clinical forms of disease very different statements are made, which are in part explained by the different prevalence of the fungi in countries remote from one another. In England the microsporon causes the large majority of cases of ringworm of the head; *Trichophyton megalosporon endothrix* is the chief cause of tinea circinata, and of tinea of the nails (onychomycosis); and *T. megalosporon endo-ectothrix* occurs in tinea sycosis and in a few cases of tinea tonsurans, and is the chief cause of kerion. Though contagion is, especially in ringworm of the scalp, mostly from child to child, the microsporon and megalosporon endo-ectothrix are sometimes caught from the horse, dog, cat, cattle, or birds.

*Tinea marginata* is due to another parasite, *Epidermophyton inguinale* (Sabouraud).

Several other forms of trichophyton occur in tropical countries. One, *T. rubrum*, causes an eruption all over the body; others, *T. violaceum* and *T. acuminatum*, a chronic dry ringworm of the palms and soles; and others cause suppurating ringworms of the backs of the hands.

**Treatment.**—The principles of treatment in tinea tonsurans are (1) destruction of the spores of the fungus by means of parasiticides locally applied; (2) the complete removal of the hair from the affected area (*epilation*), so that no further material remains as a soil for the fungus.

The difficulty of the first method lies in the fact that the spores multiply within the hair follicle, and are, therefore, to a large extent, protected from the parasiticides employed. It may succeed in slight cases, but in the severer cases it generally needs the assistance of the second procedure; and indeed the stronger parasiticide applications themselves loosen the hairs by inflaming the follicles and the surrounding skin.

The Röntgen rays have proved to be the most efficient means for loosening the hairs, and a cure can be effected by their means in one-tenth of the time that is necessary in the older methods.

In either case the first thing is to cut the hair quite close, and remove all crusts and scabs by soaking with oil and subsequent removal and washing. The extent to which hair is removed may vary with circumstances. For local applications complete shaving is best; but where it is desirable to save appearances a fringe of hair may be left all round the head, or with a single patch the hair may be close cut or shaved for an inch or more round it.

*Röntgen Rays.*—These have to be applied with great care and precision in order to avoid injuring the skin, and at the same time to get the full depilatory effect at one sitting. For this purpose the duration and strength of the application can be regulated by *Sabouraud's pastilles*, which are discs of paper thickly coated with an emulsion of platino-cyanide of barium. If the source of the rays is placed 15 cm. from the scalp, and the pastille exactly midway between them, *i.e.* 7½ cm. from the anti-cathode, the desired amount of influence is reached when the pastille acquires a particular fawn tint identical with a test colour supplied.

The application is made to a limited area of the scalp at one time, the rest being protected by sheet lead; but time is saved by treating on the same day five points, situated at least 5 inches from one another. After fifteen days the hair comes out with the slightest traction; and the patch must be constantly washed with soap and water till it is all removed, the diseased hairs falling last. The head remains bald for five or six weeks, when finer and coarser hairs begin to grow.

*Parasiticides.*—These must be regularly and constantly applied. The patch should be washed clean with soap and water night and morning, and the ointment or paste well rubbed in.

The following are some of those most commonly employed: mercurial applications, such as ung. hydrarg. ammon. or a stronger preparation of the same; oleate of mercury, from 3 to 10 per cent.; glycerinum acidi carbolici, or the same with a larger amount of carbolic acid; sulphur ointment; creosote; tincture of iodine; ung. chrysarobini; thymol or turpentine. Some of these may be combined together: sulphur 2 and carbolic acid 1 in 16 of lard or vaseline; iodine 1 and creosote or oil of cade 3 parts; caustic potash 9 grains, carbolic acid 24 grains, in ½ oz. each of lanolin and ol. cocoæ (Harrison); formalin in 40 per cent. solution, rubbed in vigorously with a brush ten minutes on alternate days for four days (Salter), is sometimes efficient, but it seems to be painful; sulphur ℥j, salicylic acid, β-naphthol and ammoniated mercury, of each 10 grains, and lanolin ℥j (Jamieson). Kerion may be treated with weak lotions of lead or boric acid, and gradually subsides; it should never be incised.

In order to test the effect of treatment the hairs must be examined from time to time, but no case can be considered cured until a good crop of hair has grown over the whole scalp, and even then a very careful search must be made for still active disease, which may be shown by a broken and twisted hair, or a small brown scaly spot.

During the treatment of a child with ringworm great care should be taken to prevent its spread to other children. The patient should use a separate brush and comb and towels. The head should be covered with a cap, which may be freshly lined with a piece of tissue paper every day, the old piece being destroyed.

*Tinea circinata* is easily cured by the use of ung. hydrarg. ammon., oleate of mercury, tincture of iodine, sulphurous acid in solution (1 part to 2 or 3 of water), a weak carbolic acid glycerine, hyposulphite of sodium (1 in 8), or some other not too strong parasiticide.

*Tinea marginata.*—Treatment of epidermophyton infection of the groins is usually not very difficult, but relapse will probably take place unless it be thoroughly carried out and unless the underclothing be disinfected. For most cases the following method proves successful: The infected regions are sponged twice daily with a lotion consisting of 2 per cent. of salicylic acid in 75 per cent. spirit; at night-time the following ointment is thoroughly rubbed in: ℞. Acidi benzoici gr. xxv, acidi salicylici gr. xv, paraffini mollis ℥ij, ol. cocois nuciferæ ad ℥j (Whitfield); in the morning either the ointment is again applied, or the following dusting powder used after sponging with the lotion: ℞. Acidi salicylici 4 parts, acidi borici 20 parts, talc. to 100 parts. It is preferable that the patient should wear a pair of short cotton pants under his ordinary ones during treatment, and even after apparent cure the use of the lotion and powder should be continued for a week or so. In resistant cases chrysarobin ointment may be used, or the pigmentum chrysarobini B.P.C., but great care must be taken not to irritate the scrotum. For eczematoid ringworm of the feet and hands the same methods may be employed, but on the feet the condition is often very intractable. *Tinea sycosis* may be treated also with Röntgen rays or by epilation.

In *onychomycosis* the nails should be scraped thin, softened with alkaline solutions, and soaked in lotions of sulphurous acid, sodium hyposulphite, or

mercury perchloride (2 grains to 1 ounce of water), or they may be frequently painted with carbolic acid. The better and quicker method is to remove the nails under an anæsthetic and dress the nail bed continuously with iodine until regrowth has occurred.

### TINEA VERSICOLOR

This is a common affection of the skin, produced by contagion, and fostered by warmth and moisture. It is more frequent in men, and especially in those who wear flannel underclothing. It is not often conveyed directly by contact — for instance, from husband to wife.

The disease begins as a small circular spot, of a yellowish-brown colour, which is slightly raised above the skin, and from which a few whitish scales can easily be detached by scraping with the finger nail or a scalpel. The patches extend and fresh ones form, so that soon a large part of the chest, where it is commonly seen first, is covered with a brown or brownish-yellow irregular patch, with a convex or scalloped margin; and on the healthy skin adjacent are numerous small isolated patches from $\frac{1}{4}$ to $\frac{1}{2}$ inch in diameter. The disease occurs only on covered parts, and is most abundant on the front and back of the chest and the abdomen. The scrapings examined in liquor potassæ under the microscope show epithelial plates with the specific fungus, *Microsporon furfur*, forming a network of branching mycelium threads, scattered among which are little groups of the relatively large conidia, like bunches of grapes. The affection does not cause much trouble beyond some itching, and is frequently ignored by the patient; but the great extent which the discoloration may sometimes reach has led to its being mistaken for Addison's disease and other pigment affections. The peculiar colour, the convex edge, and the ready desquamation of the surface should be quite distinctive, and the diagnosis is at once confirmed by the microscope

**Treatment.** It is quickly cured by rubbing in lotions of sodium hyposulphite (1 in 8) or of sulphurous acid (1 in 4), or finely powdered borax. It may, however, return if the same underclothing is used without thorough washing and disinfection.

### ERYTHRASMA

This disease is allied to tinea versicolor, but is much less common. It produces rough, brown scales, and occurs chiefly between the scrotum and thighs, in the axillæ and mammary folds. It is due to a fungus, the *Microsporon minutissimum*. It is more common in men than in women; it spreads very slowly, and lasts a long time. It must be treated like tinea versicolor.

### BLASTOMYCOSIS

In this disease there is an inflammation of the skin caused by the invasion of yeast, *blastomyces*. It is rare in this country, but is more common on the Continent, and in America.

The lesion is most often seen on the back of the hand, on the face and on the thigh, less often on the neck, other parts of the limbs, and scrotum. It begins as a papule which suppurates, and forms a crust, from under which pus or serum exudes. It gradually spreads so as to form an extensive patch, some inches in diameter, in which the skin is red, warty, with prominent vegetations, and discharging spontaneously or on pressure a sero-purulent fluid. The patch has a well-defined raised red margin. Histologically minute abscesses are seen in the rete Malpighi and in the cutis; and in the pus can be found the yeast organism, with its characteristic double contour.

The disease has some resemblance to verruca necrogenica, and to syphilitic lesions; and the diagnosis must be determined by the microscope. As a rule it does not affect the general health, but cases are recorded in which internal organs (lungs, lymph glands) were invaded.

**Treatment.**—Potassium iodide in large doses internally has a decided effect upon it; scraping, antiseptic applications and Röntgen rays may be required locally.

## DISEASES OF THE SKIN DUE TO INFECTION WITH PROTOZOA

The following are the known protozoal infections that may involve the skin:

| | |
|---|---|
| Syphilis . . . . | *Spironema pallidum* (p. 109). |
| Framboesia (yaws) . . | *Spironema pertenue* (p. 124). |
| Leishmaniasis (oriental sore) | *Leishmania tropica* (p. 161). |
| Venereal warts . . . | *Spirillum.* |
| Amœbiasis. . . . | *Entamœba histolytica.* |

Of these, syphilis and yaws are fully described elsewhere, and for an account of oriental sore ("Aleppo" or "Baghdad boil") reference should be made to special text-books. Amœbiasis of the skin was first described by Engman and Heithaus in a child suffering from amœbic dysentery, and must be excessively rare.

### VENEREAL WARTS

(*Condyloma vel verruca acuminata*)

Venereal warts are seen in both sexes. In men they occur chiefly in the furrow behind the glans penis, and on the corona and frenum, but may involve the whole prepuce and even the urethral orifice. In women they are seen on the vestibule, the fourchette, and the clitoris, and may spread over the whole vulva to the groins, anus, and perinæum. Rarely they are met with in the axillæ and on the scalp.

They consist of papillomatous vegetations, pink or brownish-red in colour, sessile and lobulated, or filiform and elongated. In women they may form enormous cauliflower-like masses, moist and foul-smelling. Being auto-inoculable, they tend to involve contiguous surfaces.

**Ætiology.**—Favre and Civatte have shown that the causal organism is almost certainly a spirillum, which is found in large numbers, both extra- and intra-cellularly. Lack of personal cleanliness, moisture, heat, and irritating discharges, which macerate the epidermis, favour their development. Gonorrhœa is a frequent though not essential predisposing cause, and pregnancy would seem to be a factor of importance.

**Anatomy.**—The vegetations consist of filiform outgrowths of young connective tissue, containing numerous large blood vessels, and covered by a thickened, actively growing Malpighian layer, the cells of which are in a state of rapid multiplication. The horny layer is thinned.

**Treatment.**—Scrupulous cleanliness, and the use of astringent lotions and a dusting powder, containing salicylic and boric acids, may cause their disappearance. In resistant cases the X-rays should be employed, or the vegetations may be removed with a curette.

## DISEASES OF THE SKIN DUE TO INFECTION WITH FILTER-PASSING ORGANISMS

It is probable that further research will show that several diseases of the skin of unknown ætiology are caused by a filter-passing virus—pityriasis rosea, for example. At present two only have been proved to be of this nature, namely, molluscum contagiosum and the common wart.

### MOLLUSCUM CONTAGIOSUM (*See Plate XLV., D*)

This name is given to small tumours on the skin, which are from $\frac{1}{16}$ to $\frac{1}{8}$ of $\frac{1}{2}$ inch in diameter, lenticular or hemispherical in shape, occasionally globular or pedunculated, somewhat irregular or nodulated on the surface, and of a yellowish-white colour. In the smaller tumours there is often a minute opening in the centre: in the larger there are several; and if the tumour be firmly squeezed, a little milky juice exudes from these apertures. Examined under the microscope, the juice is seen to consist of minute oval glistening bodies, the *molluscum corpuscles* or *bodies*. A vertical section through the tumour shows it to have a structure somewhat like that of a racemose gland. There are lobules separated by fibrous tissue; each lobule has externally a row of columnar cells, within this are more oval epidermic cells, and in the centre of each lobule is a collection of the glistening opaque molluscum bodies. The lobules in the larger tumours do not converge to a central duct or opening, but rather lie side by side, and open separately upon the surface.

The process appears to begin by a conversion of the prickle cells of the rete Malpighi into the molluscum bodies, which are stated to consist of keratin. The adjacent part of the rete enlarges downwards into the corium, and the septa between the apparent lobules are the fibrous remains of the papillæ. There is no necessary connection with sebaceous glands or hair follicles.

The growths are thus really benign epitheliomata, and are of interest in that they are caused by an infecting organism which is so minute that it passes through a Berkefeld filter, though not through the Pasteur-Chamberland. It may be inoculated by smearing the cheesy contents of the growth into an abrasion on the skin, or, better, by injecting intracutaneously a sterile saline filtrate, made by grinding up the tumours, removed with a curette, with saline solution and passing the resultant emulsion through a Berkefeld filter. After an incubation period of from twelve days to three weeks tiny molluscum growths make their appearance along the site of injection.

Molluscum contagiosum is common in birds, from which it may be communicated to man. It occurs both in children and adults, and the tumours are seen most commonly on the face, the arms or hands, the mammæ of women, and the genitals of men. They are apt to suppurate, and may disappear spontaneously; otherwise they may last indefinitely and multiply by auto-inoculation.

**Treatment.**—The tumours may be caused to disappear by expressing the contents and applying pure carbolic or iodine to the interior, or "by boring into them with a grooved needle dipped into melted silver nitrate, so that it becomes charged with a sheath of the salt" (Whitfield). A simpler method is to scrape them away with a small curette and paint the affected surface with weak tincture of iodine.

### WARTS

(*Verruca*)

There are several distinct varieties of verruca or wart, of entirely different ætiology. Thus the common wart (*V. vulgaris*) is due to a filter-passing virus, the venereal wart (*V. vel condyloma acuminata*) is probably caused by a spirillum (*see* p. 962), the seborrhœic wart (*V. seborrhœica*) is possibly a nævoid growth of late development, and the senile wart (*V. vel keratosis senilis*) is a pre-cancerous degeneration of the skin. The last two are often wrongly confused, and neither has any connection with the verruca vulgaris.

Common warts are excrescences from the skin, consisting of hypertrophied papillæ capped with horny epidermis. They vary in shape and size; thus they may be small and flat (*V. plana*), hemispherical, pointed, or pedunculated, and the larger ones may be lobulated or digitate. In colour the flat ones are pale yellow or pinkish, the larger ones darker and of a greyish-brown tint. They occur

most commonly on the backs of the hands, on the fingers, and on the face, particularly around the mouth, owing to the tendency of children to bite the original, or "mother-wart," situated on the hand. They also occur on the eyelids and scalp, and on the soles (*V. plantaris*). Large numbers of flat warts are not infrequently met with on the face in children, and sometimes in adults. In the former they are situated chiefly on the forehead, the temples, the cheeks and the chin, and children with seborrhœa and pityriasis of the scalp and face appear to be especially predisposed. In men both flat and pedunculated warts may occur on the beard region, and are spread by the shaving brush and razor. The nail-folds are sometimes involved, the warty growth often extending underneath the nail. *Plantar warts* are very common, and of importance owing to the intense pain they may cause; they are often mistaken for corns. They are usually situated over the heads of the metatarsals or on the heel. They are surrounded by a horny ring, in the centre of which the papillary vegetations can be observed through the transparent corneous layer.

**Ætiology.** Common warts are seen in children and adolescents more frequently than in adults. They are contagious and auto-inoculable. The infecting organism has been shown to be a filtrable virus (**7**); plane warts were reproduced by inoculating the juice of crushed common warts, filtered through a Berkefeld filter.

**Anatomy.**—There is an enormous hypertrophy and elongation of the papillæ, with thickening of all the epidermal layers. There is no inflammatory change in the dermis.

**Treatment.**—Warts of moderate or large size are easily removed by freezing with a stick of carbon-dioxide snow, after previously softening them with a fomentation. They may also be destroyed with the thermo- or galvano-cautery, by electrolysis, or, if very numerous, by ionisation with magnesium sulphate. Plane warts will often disappear with moderate doses of the X-rays, and also by the repeated application of a 1/1000 solution of biniodide of mercury in 75 per cent. spirit containing 2 per cent. of salicylic acid.

Plantar warts are sometimes difficult to cure, but, as a rule, large doses of X-rays — one-and-a-half to two pastilles, the surrounding normal skin being carefully protected, are successful. Internal treatment with magnesium sulphate, the green iodide of mercury, or increasing doses of arsenic is very unreliable.

## DISEASES OF THE SKIN DUE TO INFECTION WITH ANIMAL PARASITES

Various animal parasites may infest the human skin, and give rise to lesions or eruptions, which are, as a rule, characteristic. Lice, fleas, bugs and mosquitoes are examples of parasitic insects; several varieties of acarus, *e.g.*, the *Sarcoptes scabiei hominis*, the cause of human scabies, the *Leptus autumnalis* or "harvest-bug," the *Ixodes ricinus* or "wood-louse," the *Pediculoides ventricosus*, the cause of "grain-itch," and the varieties of Sarcoptes, parasitic on animals such as the dog, cat, sheep, and pig, may all invade human beings. Lastly, certain worms and larvæ may enter the skin, *e.g.*, the guinea-worm, the larva of *ancylostoma duodenale*, and the larva of certain dipterous insects—*larva migrans*. Here will be considered only the eruptions produced by lice, known as phtheiriasis or pediculosis, and human scabies.

The pediculi or lice which infest the human race are of three species: *Pediculus capitis*, or head louse; *P. corporis, vel vestimentorum*, or body louse; and *P. pubis*, or crab louse.

### PEDICULUS CAPITIS

The head louse is about 2 mm. long by 1 broad, and breeds amongst the hairs of the scalp. Its ova are found adherent to the hairs and are called nits. They

are about ½ mm. in length, whitish, somewhat conical in shape, with the apex always towards the scalp; and they are fixed to the hair by a cylindrical sheath of chitinous material extending some little distance beyond the apex. The irritation of the pediculus leads to constant scratching and pustular eczema, or contagious impetigo. This eruption is most common and severe in the occipital region, and the suboccipital glands are often enlarged as a consequence, and may suppurate.

The **Diagnosis** is not difficult. If the pediculi are not at once seen, the nits, which are readily distinguished on careful examination from scurf, will show at once that there are or have been pediculi. The position of the crusts at the back of the head is also strongly in favour of pediculi.

**Treatment.**—The insects can be destroyed by the use of ung. hydrargyri ammoniati, or better by lightly rubbing in paraffin oil, which rapidly kills the animals, or the hair may be soaked with a solution of carbolic acid (1 in 40) and then wrapped up in a towel for an hour. If there is much eczema or impetigo, the hair should be cut over it, and the crusts removed. The nits are not easily detached from the hairs; the cement is very resistant to acids, alkalies, and spirit, but dilute acetic acid (1 in 4) is said to soften it. They can always be slid off the hair, or may be sometimes combed off; but if very numerous, it is probably best to cut the hair.

### PEDICULUS VESTIMENTORUM (*See Plate XLVIII., B*).

This species is larger than the head louse, being from 2 to 3 mm. long, and from 1 to 1½ broad. It only occurs on the parts covered with clothes, and chiefly about the back and front of the chest, loins and abdomen. Occasionally the upper arms, thighs, and even legs may be attacked, but never the face or the hands. The body louse causes intense itching, or pruritus, which leads to proportionately violent scratching. All the lesions described under Prurigo (*see* p. 996) may occur, papules, blood crusts, scratch marks and the elongated scars which result from them, and after a time deep pigmentation. In its milder form it presents only scattered papules, blood crusts, and scratch marks over the upper part of the back and shoulders.

It occurs especially in old people, amongst the poorer classes, who have been allowed to lapse into conditions of filth and neglect. The disease has then been called *prurigo senilis*, and also *vagabonds' disease*.

Lice were a constant source of trouble amongst our soldiers in the trenches.

**Diagnosis.**—A prurigmous eruption of this kind over the back and shoulders should always suggest a search for the pediculus vestimentorum. It is commonly found in the "gathers" under the neck band of the shirt, or under the shelter of any edge projecting on the inner side; and it is recognised by its long oval shape and its grey colour, with a central dark red or black spot.

**Treatment.**—Ung. hydrarg. ammoniati or ung. staphysagriæ, smeared over the skin, will kill the pediculi. The clothes, in which the eggs are certainly incubating, should be completely changed; and they must be baked if they are to be worn again.

### PEDICULUS PUBIS

The crab louse is smaller than either of the other species, measuring from 1 to 1½ mm. long, and from 1 to 1½ broad. It has an almost square body, and six long legs, with claws by which it clings firmly to the hairs of the part. It is not only found in the pubic and axillary hair, and in that over the sternum, but is occasionally conveyed to the eyebrows, eyelashes, whiskers, or beard. The eggs are attached to the hairs close to the skin. Itching leads to scratching, and an eczematous rash is the result. In many cases bluish-grey macules (*maculæ cæruleæ, taches bleues, taches ombrées*) about the size of a finger nail are seen in the skin of the lower abdomen and thighs, and on the sides of the thorax, if the

axillary and thoracic hair is affected. They are absolutely characteristic of crab-louse infection, though formerly they were thought to be symptomatic of typhus and even typhoid fever. The pigment in these macules is derived from the salivary glands of the louse.

**Treatment.**—The unguentum hydrargyri B.P. is a favourite remedy among the public, and it is very effectual, but it is apt to cause pustular folliculitis or even acute mercurial dermatitis. A safer method is first to soak the infected parts for an hour with a solution of carbolic acid (1 in 40) applied on lint, and then to rub in an ointment containing yellow oxide of mercury, ammoniated mercury, or balsam of Peru for a few days. The pubic hair need never be shaved. It should be remembered that, although infection usually takes place from sexual intercourse, it may be acquired in other ways, as from dirty closet seats.

### SCABIES (*See Plate XLVIII., A*)

Scabies, or itch, is a multiform disease of the skin, consisting of papules, vesicles, pustules, and sometimes bullæ, due to the irritation of the itch acarus, *Sarcoptes hominis*.

The female acarus is oval in shape, $\frac{1}{70}$ inch in length, presents in front four little nipple-shaped processes provided with suckers on stalks, and behind four similar processes provided with long bristles. The male is smaller, has four suckers in front, two suckers and two bristles behind. The female after impregnation bores her way under the skin in an oblique direction, so that, as the superficial layers of the epidermis are detached by friction, she still remains at the same depth from the surface. As she proceeds she lays her eggs, one or two daily, it is said, and she may thus burrow through the skin in an irregular line for $\frac{1}{3}$ or $\frac{1}{2}$ inch. Such a burrow (*cuniculus*), or "run," may be recognised on the surface of the skin by the following features: At one end the epidermis is broken or frayed, and the free edges are dirty; at the other end is a minute white pointed elevation, in which the acarus lies; the burrow itself between these points is a sinuous black line. The whole burrow may be snipped off with a pair of scissors curved on the flat, or shaved off with a scalpel; and if it be then moistened with liq. potassæ and examined, there will be seen the female acarus, and behind her, filling the burrow, her eggs in every stage of incubation, with minute black spots of excremental matter among them. As the skin desquamates the most developed ova come to the surface, and are hatched. The male does not burrow, but remains on the surface, where he may be sometimes accidentally caught.

**Symptoms.**—As a result of the invasion of the acarus there is considerable itching, with consequent scratching, pus infection, and dermatitis of variable extent and character. The itching is mild or severe, but not generally so bad as that caused by pediculi, and the scratching rarely leaves scars or causes pigmentation. It is worse at night when the patient is warm in bed. The dermatitis consists of papules, vesicles, pustules, or even bullæ, which generally appear in the neighbourhood of the burrows, but also in parts more remote. Not infrequently a vesicle or pustule forms in the burrow itself. In addition to these lesions, patches of eczema, impetiginous crusts, and urticarial wheals are often present. There is thus very great variety in the lesions in different cases. In some the burrows are numerous, with few inflammatory lesions; in others vesicles and pustules are abundant, and burrows are with difficulty found. In some instances there are more papules, in others more pustules. The parts of the body especially liable to the attacks of the acarus are the skin between the fingers, the front or inner side of the wrist, the front of the forearm, the elbows, the ankle and foot, the axilla, the groin, and the genitals, the inner side of the thigh and the nates; and the eruption spreads beyond these parts on to the abdomen from the groin, or along the inner side of the leg or thigh. The back, shoulders, and chest are but little affected; and the face, neck, ears, and scalp nearly always

escape, except in infants at the breast, who may be infected from the mother The occupation of the patient may have influence upon the localisation. Hebra was in the habit of recognising the occupation of a cobbler by the predominance of itch lesions on the buttock. If the employment involves the immersion of the hands in materials (oily or otherwise) antagonistic to the acari, their presence in this typical situation will, of course, be prevented. In children the lesions are more widespread, the feet and ankles are commonly affected, and pustules are frequently present.

**Diagnosis.**—The position of the lesions is an important guide to diagnosis. An itching eruption of mixed papules, vesicles, and pustules, occurring mainly about the fingers and wrists, and also in the other situations mentioned, should lead to a careful search. If a burrow can be found, the minute white elevation at the cleaner end should be looked for, and its epidermis carefully scratched through with the point of a needle; the acarus may then be picked out, as it readily adheres to the surface of the needle. If this cannot be done, it is best to snip off the whole burrow, and to examine for ova or fragments of the acarus under the microscope. Finally, where there has been much inflammation, so that burrows cannot be found, the crusts may be removed, boiled in a solution of potash or soda, the fluid allowed to settle in a conical glass, and the sediment examined for fragments of acarus.

**Treatment.**—Itch is generally quickly cured by the free use of sulphur ointment. The patient should sit in a hot bath for half an hour, and scrub the skin with soap and a nail brush. He should then dry himself, and the ointment should be thoroughly rubbed in all over, especially over the hands, and flexures of the joints, and other parts most affected. Suitable clothing must then be put on over the ointment, and the patient may go to bed. In the morning the ointment may be washed off. The same measures should be carried out on the succeeding three or four nights. The disagreeable odour of sulphur ointment may be lessened by the addition of balsam of Peru (℥j to ℥j). For some skins sulphur ointment is too irritating, and requires dilution. Storax ointment (styracis prep., ℥ij; sp. meth., ℥iij; adipis, ℥j) is also a more pleasant, less irritating, and yet effectual preparation. Vlemingkx's solution may also be used, or sulphur baths (potassium sulphide, 4 ounces, in 30 gallons of water). It must be remembered that the itching may continue for some time after the acarus has been destroyed. In order to prevent the recurrence of the disease, it is necessary that new clothes should be worn, and that the old ones should be quite disinfected by baking before being worn again.

## GROUP II

# DISEASES CLASSIFIED ACCORDING TO THEIR MORPHOLOGY, NOT INCLUDED IN GROUP I

### THE ERYTHEMAS

Urticaria and some forms of erythema are essentially sensitisation phenomena, *i.e.*, they are manifestations of the allergic state (*see* p. 224). Recent observations have shown that Erythema multiforme, E. nodosum, E. scarlatiniforme, and even Lupus erythematosus must, like urticaria, be regarded as non-specific reactions, the toxin or antigen differing in different cases. The important work that has been done in connection with the tuberculides and trichophytides has made this clear. These eruptions occur in persons who have become sensitised

to tubercle and a trichophyton fungus respectively, when the actual infecting organisms or their toxins are circulating in the blood-stream.

Now there is little doubt that Erythema multiforme, E. nodosum and E. scarlatiniforme, particularly in their recurrent forms, are usually due to sensitisation having occurred to some strain of a *Streptococcus longus*, and the same is true of many cases of Lupus erythematosus; but Erythema multiforme probably (**8**), Erythema nodosum (**9**), and Erythema scarlatiniforme (**10**), may also be caused by sensitisation to a trichophyton, and Erythema nodosum, Lupus erythematosus and, perhaps, Erythema multiforme, may in some instances be tuberculides. Therefore, however unwilling one may be to ascribe eruptions with well-defined clinical characteristics to multiple causes, it must be accepted that these forms of erythema are syndromes, and not specific entities.

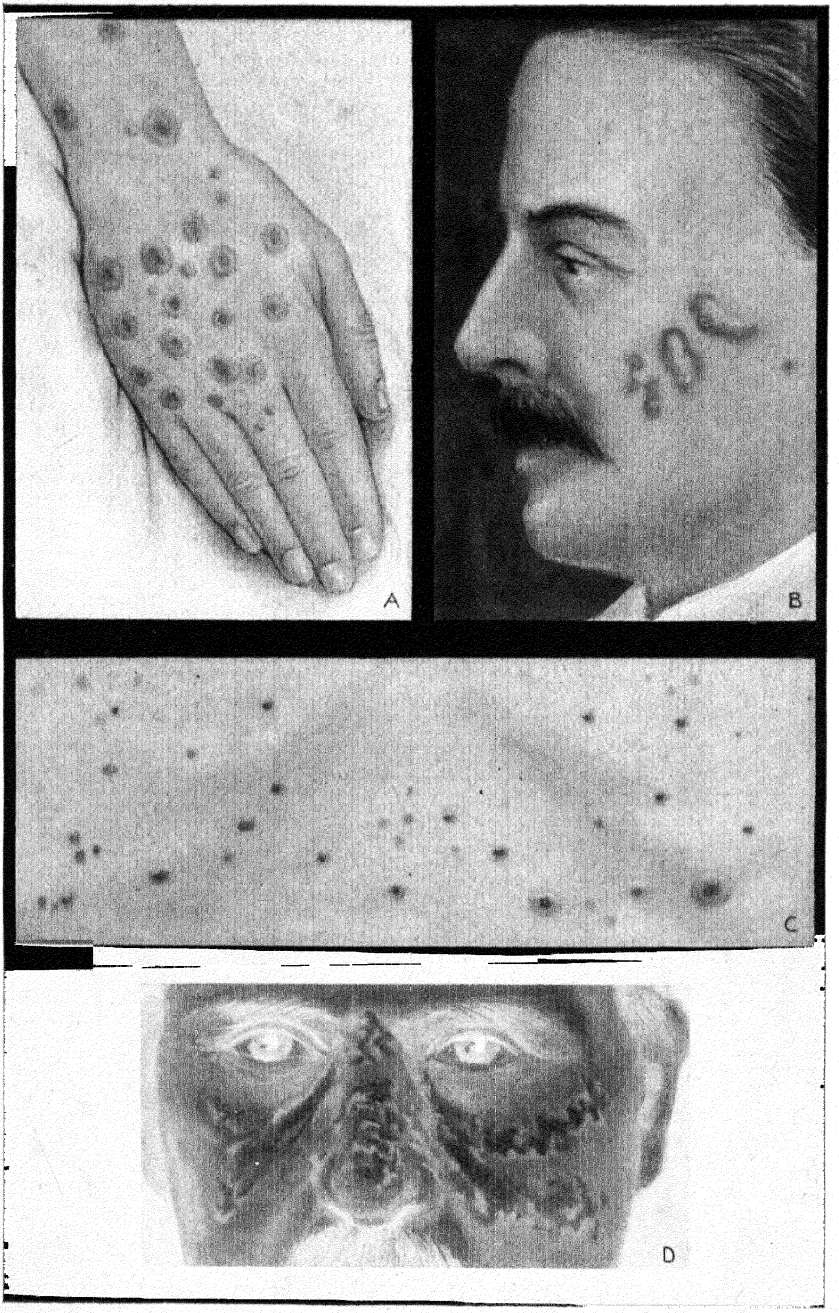
## ERYTHEMA MULTIFORME (*See Plate XLVII., A*)

Some authors are inclined to include under the heading of Erythema multiforme the ill defined erythemato-urticarial rashes that may result from sensitisation to food-substances (*e.g.*, pork, shell-fish), foreign serums ("serum rash"), various drugs, and many bacterial and protozoal toxins, but the classical form of the eruption known as *Erythema exudativum multiforme* is a clinical entity and should be separated from the above.

**Symptoms.** The eruption, which is often accompanied by sore throat, fever, pains in the joints and muscles, and gastro-intestinal disturbance, appears suddenly, at first almost invariably on the parts exposed to light, viz., the backs of the hands and wrists, the face and neck, and, in women, on the V-shaped area of the upper chest exposed by the cut of the blouse. It is bilaterally symmetrical, and later may involve the forearms, elbows, knees, ankles and feet; occasionally it is more or less generalised. The *mucous membranes* of the lips, cheeks, tongue, pharynx and vulva are frequently involved, particularly in the bullous form, and in recurrent cases. Moreover, the mucous membranes may alone be affected by periodical attacks for years without any cutaneous lesions developing, and many cases of so called recurrent stomatitis are really of this nature.

As the name suggests, there is considerable variety in the lesions produced. The most characteristic, seen most typically on the backs of the hands and wrists, are raised, flat, disc-like erythematous papules, bluish-red in the centre, bright red or pink at the periphery; on palpation such a lesion gives to the finger a peculiar sensation as of an india-rubber cap stretched over the mouth of a bottle full of water. The centres of the lesions are often white, owing to the exudation of fluid, which may be sufficient to produce a vesicle or bulla (*E. bullosum*), and in some cases large, pemphigoid bullæ, surrounded by an erythematous halo, occur; sometimes actual haemorrhage takes place in the centre of a papule (*Purpura urticans*). Moreover, the central parts may clear, leaving rings (*E. annulatum*), which by coalescence produce sinuous or scalloped patches (*E. gyratum*). Rarely a ring of erythema is surrounded by another right outside it, and this is followed by another farther out, while the first ring is beginning to fade. The different colours of the rings in various stages suggest the name *E. iris*. As many as four such rings may be seen at the same time, sometimes arranged concentrally around a central bulla (*Herpes iris*), which may occasionally be surrounded by one or more rings of smaller bullæ or vesicles. On the mucous membranes the lesions consist of flaccid bullæ, which are rapidly replaced by extremely painful erosions upon which a dirty white diphtheroid membrane forms, in some cases practically the entire buccal and pharyngeal mucous membranes are involved, and the pain and discomfort are such as to render eating almost impossible.

One of the most striking features of Erythema multiforme is its tendency to recurrence; these recurrences occur at varying intervals, sometimes annually—




A. Erythema Multiforme.—Note the distribution on the back of the hand, and the raised, discoid, erythematous lesions, pink at the periphery and violet in the centre.

B. Lupus Vulgaris.—Note the apple-jelly colour of the nodules, and the scarring left in areas that have spontaneously healed.

C. Urticaria Papulosa.—Note the urticarial wheals with their central papules, and the small papular, sometimes lichenified, lesions left when the surrounding wheals have subsided.

D. Lupus Erythematosus.—Note the distribution on the "butterfly-area" of the face, the persistent, irregular erythematous patches, with telangiectasia, and the superficial scarring.

[*To face p.* 969.

as a rule in the spring—sometimes every two or three months, and rarely a new attack may begin before the lesions of a previous one have disappeared. Osler long ago emphasised the close association that exists between Erythema multiforme, urticaria, purpura and Erythema nodosum, and their connection with visceral disease, such as endo- or myocarditis, nephritis, and gastro-intestinal disturbances. This association is easily explained, because the commonest cause of E. multiforme, E. nodosum, and purpura is an acute or chronic streptococcal infection, and urticaria may be due to sensitisation to the same organism. Moreover, although it is denied by some, there is no doubt that the common form of Erythema nodosum should be considered as the deep variety of Erythema multiforme, and the two may coexist in the same attack, or occasionally alternating outbreaks of one or the other occur. Apart from true Erythema nodosum, there may rarely be seen in association with the usual superficial lesions of E. multiforme circumscribed intradermic or subcutaneous nodules, doubtless caused by emboli of streptococci.

**Ætiology.**—As has been said, Erythema multiforme, like urticaria and some other forms of erythema, even in its most classical form, cannot be regarded as an absolutely specific reaction. The work of Landouzy would seem to prove that it, as well as Erythema nodosum, may be a toxi-tuberculide, but this must be rare. An Erythema multiforme type of trichophytide has also been described (**8**). These observations, though of great interest, are of little practical importance. All the evidence goes to show that in the great majority of cases Erythema multiforme is due to sensitisation having occurred to a strain of *Streptococcus longus*. Its association with acute rheumatism is too frequent to be a coincidence; it occurs in streptococcal septicæmia, and an attack may usher in a fatal streptococcal infective endocarditis. In a series of patients subject to recurrent attacks one or more foci of chronic streptococcal infection were invariably present, specific agglutinins and precipitins were present in the serum, injections of a vaccine prepared from the infecting strain of streptococcus often provoked focal reactions, *i.e.*, the appearance of a few typical lesions on the skin or mucous membranes, and removal of accessible foci of infection, combined with the administration of an autogenous streptococcal vaccine over a long period, brought about a cessation of the attacks (**18**). The usual sources or portals of entry of infection are the tonsils, nasopharynx, teeth, nasal sinuses, and intestines, but possibly others, such as the prostate and cervix uteri, may be responsible. Exposure to sunlight is undoubtedly a factor in predisposing to an outbreak of the eruption and in determining its localisation.

**Treatment.**—The patient should be put to bed, and the heart watched carefully for signs of endocarditis or myocarditis. Sodium salicylate or salicin, combined with alkalies, may be given, or quinine. Locally cooling lotions, such as lotio plumbi or lotio calaminæ, should be applied during the acute stage. In the bullous form of the eruption, when the mucous membrane of the mouth is involved, the pain and discomfort may be relieved by allowing the patient to suck the trochiscus krameriæ et cocainæ B.P. and the trochiscus acidi carbolici B.P.

When the attack has subsided a search should be made for some chronic focus of infection, such as the tonsils or teeth, and, if found, this should be dealt with. In recurrent cases a streptococcal vaccine prepared from the infecting focus should be administered over a long period. For the anæmia, following the attack, iron should be given until the blood returns to normal.

### ERYTHEMA NODOSUM

This consists of oval or circular solid flat elevations of the skin, from ½ inch to 1¼ inch in diameter, bright or dusky red in colour, gradually shading off into the surrounding skin, tender to the touch, and perhaps pitting slightly on pres-

sure. These occur most often over the whole length of both tibiæ, and not infrequently over both ulnæ. Though rare in other parts, they may be seen on the calf, on the thighs, over the scapulæ, and over the condyles of the humerus. They come out more or less in crops, last seven or ten days, and gradually subside with bruise-like staining. They may become soft and fluctuate, but never suppurate. They are most common in children and people under twenty years of age, and more frequent in girls than in boys. The onset is preceded by some malaise, pains in the joints, and slight pyrexia.

**Ætiology.**—As has already been pointed out, Erythema nodosum cannot now be regarded as a specific disease, but rather as a dermo-hypodermic reaction, similar to that which occurs in Erythema induratum, and, like it, in all probability always provoked by the embolism of infecting organisms in a sensitised person. As commonly met with, the eruption should be regarded as a hypodermic variety of Erythema multiforme, with which it may co-exist, and due to emboli of streptococci. Like E. multiforme, it may accompany acute rheumatism, and endocarditis may develop during an attack. A streptococcus has been cultivated from the blood (**11**).

Although, however, the eruption is, as a rule, presumably of streptococcal origin, it may be apparently a tuberculide, and Landouzy claims to have demonstrated a tubercle bacillus in a vessel of a nodule, and to have tuberculised a guinea-pig by injection of a portion of the same nodule. It has also been regarded as a syphilide and a lepride.

That it may be a form of trichophytide seems to have been definitely proved in cases of severe kerion. Bloch obtained focal reactions with injection of trichophytin, and Bruusgaard claims to have demonstrated spores in the nodules of his case, and to have cultivated the Trichophyton gypseum from the lesions, which was the fungus responsible for the original ringworm infection.

**Treatment.**—The patient must be put to bed, and the heart carefully watched. Sodium salicylate may be given, but, as with *E. multiforme*, the most important indication is to search for some chronic focus of infection, and to deal with this. Levinsohn has reported a case in which recurrent attacks occurred over a period of many years, and in which a cure resulted from the administration of a stock streptococcal vaccine.

## ERYTHEMA PERNIO

Pernio, or chilblain, is a superficial dermatitis, affecting the toes, sides of the foot, and the fingers, as a result of cold in people of defective circulation, and especially in children and females. There are patches of dusky redness, with itching, smarting, and pain, which come on frequently with the cold winter weather, and may only completely subside with the return of spring. In severe cases, or if irritated by friction or injury, they may vesicate or form indolent ulcers. They should be prevented, if possible, by warm clothing, sufficiently loose boots, and active exercise, such as running, skipping, dancing, and skating.

**Ætiology.**—Although cold, particularly damp cold, is the provoking cause, various conditions of ill-health may act as predisposing factors, such as malnutrition, chronic intestinal stasis, anæmia, chronic cardiac and renal disease, and tuberculosis.

**Treatment.**—The internal treatment must necessarily depend on the state of the general health; particularly is it of importance to guard against intestinal toxæmia and, if anæmia be present, to search for its cause. Regular physical exercise is of value, and should be insisted on. As regards drugs, in a few cases calcium salts seem almost specific; calcium lactate in 15-grain doses or the syr. calcii lactophosph. B.P. may be prescribed. In cases of malnutrition or tuberculosis, cod-liver oil should, of course, be administered. Locally, galvanic baths improve the circulation very effectively, and should be given for ten

minutes daily. Very good results are also obtained by immersing the affected parts in a mixture of hydrogen peroxide (15 to 20 vols.) and boiling water for a quarter of an hour every night, followed by the application of a stimulating preparation, such as iodex or a methyl-salicylate ointment.

### LUPUS ERYTHEMATOSUS (*See Plate XLVII., D*)

This disease occurs mostly in adults, especially between the ages of twenty and forty, is very rare in children, and is more common in women than in men. Its early recognition is of great importance, since it affects chiefly the face, and, as the lesions often result in scarring, it may cause permanent disfigurement. Various forms of the eruption are recognised, although they may all coexist in the same patient. In the majority of cases the disease begins as a red, injected, desquamating patch, with a well-defined, irregular, and slightly raised edge. If a piece of scale be removed, there will often be seen little horny plugs projecting from the under-surface; these fit into depressions in the epidermis, which, after removal of the scale, are recognised as small pits on the denuded surface of the patch. Dilated venules are usually visible, and in one variety the formation of telangiectases is very marked. As the disease spreads peripherally, involution often occurs in the centre, so that after a while there is a central, depressed, pale scar surrounded by a raised, red, scaly edge. The scar formation is not preceded by ulceration. In another variety there is no scale formation, but the disease appears as raised, œdematous patches of erythema, closely resembling, and, indeed, often indistinguishable from, those seen in *Erythema multiforme*. In this erythematous type, scarring is very slight, and may be entirely absent, the patches of erythema eventually disappearing without leaving any trace. Although the eruption is usually of limited extent, it may occur in an acute generalised form from the onset, or, more commonly, after a few chronic patches have been present for some time. In the acute form the recent patches either resemble those of *Erythema multiforme*, or they may be mistaken for eczema, although actual vesiculation is rarely seen.

**Distribution.**—This is important from the diagnostic standpoint. The commonest sites are the malar eminences, the nose, the ears, the scalp, the backs of the hands and fingers, and the vermilion border of the lips. The distribution tends to be symmetrical, and often patches occur on either cheek and across the nose, so that the outline of a butterfly or bat is simulated—a very characteristic feature. The disease may rarely begin on the scalp or on the fingers before the face is involved. On the scalp atrophy of the hair follicles occurs, and patches of permanent alopecia, usually traversed by telangiectases, result. The ears are affected along the edges, which become eroded, irregular in outline, and shrunken, and the inner surface of the auricle is also affected. The mucous membranes of the mouth, nose and conjunctiva may be attacked, but, except for the lips, they are usually spared.

The subjective symptoms may be slight, but there is often great irritation in the patches, and many patients complain of intense burning, particularly after meals or on exposure to sun or wind. The disease is not infrequently associated with rheumatoid arthritis of varying severity. In the acute generalised form there is pyrexia, sometimes of the septicæmic type, and there may be albuminuria; these cases may terminate fatally.

**Ætiology.**—As will be evident from the above description, the eruption tends to involve those situations in which circulatory stasis is apt to occur, namely the fingers and toes, the backs of the hands, the ears, and the rosaceous or "butterfly" area of the face, and in people with marked acroasphyxia the hands, feet, and ears may be the chief or only parts affected, whereas in those with a pre-existing rosacea the "butterfly" area is likely to be the site of predilection. Frequently, however, rosacea and a "chilblain-circulation" coexist, in which

case all the above sites may be involved in the same patient. Since the eruption is produced by circulatory toxins—perhaps, even, by an actual bacteriæmia—and is essentially chronic, it is obvious that the inflammatory reaction, which constitutes the lesions, is most likely to appear where there is vascular stasis; moreover, certain areas of skin evidently become sensitised to the toxins, so that recurrence may take place in the identical situations previously involved, when there is a recrudescence of the toxæmia after temporary disappearance of the eruption. As to the nature of the toxins or micro-organisms causing the disease there are now two predominant views; one, which obtains most favour on the Continent, is that it is a tuberculide, the other that it is a manifestation of chronic or acute streptococcal infection. Many now incline to the opinion that, like other forms of erythema, it is not a specific entity, but that both tuberculous and streptococcal infection (and possibly others) may be responsible in different cases.

In favour of its tuberculous origin in some cases, its occurrence in those with a strong family history of tuberculosis, its association with manifest tuberculous infection of glands, bones, joints, lungs, etc., and the claims that guinea-pigs have been tuberculised by inoculation of the affected tissue may be mentioned. On the other hand, no trace whatever of tuberculosis was found in some fatal cases of the disease (**12**). Convincing evidence has been produced that in some cases acute or chronic streptococcal infection is the cause (**13**). The primary sources may be the teeth, nasal sinuses, tonsils, and probably the prostate and female genital organs; in many cases there is a secondary and persistent intestinal infection with a *Streptococcus longus*, which may rarely be recovered from the fæces in almost pure culture. In fatal cases a *Streptococcus longus* has been obtained from the blood during life (**14**). Certainly the fatal cases reported have resembled clinically streptococcal septicæmia rather than general tuberculosis. It must be remembered that, as in chronic tuberculosis, cases of chronic streptococcal infection have as a rule both primary and secondary foci, and the removal of obvious and accessible sources of infection does not necessarily suffice to cure, owing to the existence of subsidiary foci in lymphatic glands, the intestines and elsewhere.

**Diagnosis.**—The conditions which are most likely to be mistaken for *Lupus erythematosus* are rosacea, *Erythema multiforme* and *Erythema pernio*, eczema, psoriasis, and *Lupus vulgaris*. The distribution, the persistence, the resistance to local treatment, and the tendency to superficial scarring, are the chief characteristics on which the diagnosis rests. From *Lupus vulgaris* the main points of distinction are that *Lupus erythematosus* very rarely begins in childhood, it is usually bilaterally symmetrical, it never ulcerates, and never destroys cartilage, it frequently involves the scalp, the scarring from it is superficial, it only rarely reacts to injections of tuberculin, and the characteristic apple-jelly nodules are absent.

**Treatment.**—The successful treatment of this chronic and disfiguring disease depends entirely on complete investigation. Evidence of latent tuberculosis and foci of streptococcal infection should be sought for. The teeth should always be radiographed, and, if there is evidence of apical infection, one affected tooth should first be extracted and cultured, and the patient's serum tested against the streptococcus isolated for the presence of specific agglutinins and precipitins. The tonsils should be carefully examined, and the possibility of a latent sinus infection considered: cases have been known to recover rapidly after the drainage of an unsuspected empyema of an antrum. The fæces should be cultured repeatedly for pathogenic strains of streptococci, and serological tests performed. A fractional test meal may reveal achlorhydria, which favours secondary intestinal infection, and an X-ray examination of the alimentary tract should be made for evidence of chronic appendicitis, stasis, etc. Lastly, the prostate and female pelvic organs should not be forgotten as possible sources of infection.

Apart from removal of any definite accessible infective foci, the patient's resistance must be raised in every possible way. In severe cases prolonged rest in bed out of doors is advisable; this alone will often result in great improvement, some or all of the eruption disappearing spontaneously. The administration of an autogenous vaccine prepared from the particular strain of streptococcus, that by serological tests has been proved to be pathogenic, is undoubtedly of great value, but must be continued, with intervals of rest, over a long period. Recently light-baths with a carbon-arc or mercury-vapour lamp have been shown to be of striking benefit, but it is advisable at first to protect the actual eruption from the rays, which may aggravate it, or even cause it to spread.

Internally iodine in increasing doses appears to do more good than any other drug that has yet been tried. It should be given as a 10 per cent. solution in rectified spirit, and should *not* be combined with iodide of potassium. Of this solution 5 drops daily in milk should be taken at first, the dose being increased gradually up to 40 drops in divided doses *per diem* if well tolerated. Very much larger doses have been given without ill effect, but instances of intolerance are met with. Cod-liver oil is often of great benefit, but is apt to upset the digestion. Quinine and salicin appear sometimes to be of value, especially in the more acute cases.

*Locally.*—In the acute and hyperæmic cases cooling lotions such as those of calamine and lead should be applied. In more chronic cases, without much infiltration, continuous compression with non-flexible collodion, to which 2 per cent. of salicylic acid may be added, sometimes results in improvement and even disappearance of the patches. Chronic patches of the fixed type may be treated more energetically. The scales should be removed with soft soap and spirit, or a salicylic acid plaster, and the patches may then be painted with tincture of iodine, while quinine is simultaneously administered internally (Hollander). Or the patches may be rubbed once a week with a glass rod dipped in a mixture of lactic acid 1 parts, carbolic acid 1 part. Repeated applications of solid carbon dioxide snow for fifteen seconds with moderate pressure, though causing a severe reaction, may bring about the disappearance of chronic patches. Ionisation with zinc or copper sulphate, scarification, and even cauterisation, have been used with varying results. The X-rays and the Finsen light are more likely to do harm than good. It should be remembered that local treatment, though of great value, does not prevent recurrences, and for this reason every effort should be made to locate the underlying infection responsible for the disease.

## ERYTHEMA AB IGNE

(*Livedo reticulata*)

This is redness with some infiltration and swelling which occurs on the front of the legs and sometimes the backs of the hands of those who are constantly in front of and close to a fire. It is commonly distributed in the form of a network, that is, broad bands crossing and interlacing, with intervening areas of paler or healthy skin. After a time it is accompanied by pigmentation, which becomes obvious when the blood is pressed out by the finger, and which persists for a long time, if the cause is removed and the hyperæmia consequently subsides. The pigmented condition is also known as *ephelis ab igne*.

A reticulated hyperæmia may be produced in other parts of the body by the prolonged application of hot poultices or hot-water bottles—*erythema a calore*.

The reddened bands of skin represent venous areas; and somewhat similar reticulated mottlings occur, which are independent of external irritants and are suggestive rather of stagnation of blood, or retarded circulation on the venous side. They are seen in young persons on the extensor surfaces of the forearms, and on the knees: in other subjects in association with mitral stenosis,

local venous obstruction, or the presence of cervical ribs; and in connection with Raynaud's disease (Parkes Weber).

To all these conditions, whether from external or internal causes, the names of *Livedo reticulata* and *Livedo annularis* have been given.

## ROSACEA

(*Acne Rosacea—Gutta Rosea*)

In the earliest stage of the disease there is periodic flushing of the face, occurring usually after meals or on exposure to heat and cold. At first the hyperæmia affects the nose and malar regions, but later it extends to the centre of the forehead and the chin. After a while the flushing tends to become permanent, although it varies in intensity at different times, and eventually telangiectases appear; they are particularly numerous in persons with a feeble peripheral circulation. In most cases the flushing is accompanied by seborrhœa, and sometimes by hyperidrosis, and secondary inflammation is very apt to occur around the pilo-sebaceous follicles, resulting in the formation of pustules and papules, which cause great disfigurement. Fresh outbreaks of these inflammatory lesions occur periodically, and in women they are apt to be most numerous just before the menstrual periods; an indigestible meal or indulgence in alcoholic liquor may invariably provoke an outbreak in certain cases. Although the disease is usually most evident on the nose and cheeks, it may affect chiefly the chin, particularly in women; in such patients, crops of indolent, red papules and pustules continually appear in this situation. This variety is said to be associated with ovarian or uterine disorders, but the association is probably not a direct one. In another variety of the disease the skin, instead of being greasy, is dry, and superficial, red, scaly patches, intermingled with small pustules, appear on the hyperæmic areas.

In long-standing cases of rosacea the condition known as rhinophyma may arise. It is seen most commonly in men of alcoholic habits, whose employment exposes them to cold winds. The nose becomes covered with large, lobulated or pendulous protuberances, separated by deep furrows; their colour is deep violet, and their surfaces traversed by large dilated veins, and pitted by the dilated follicles of the hypertrophied sebaceous glands.

The dyspeptic symptoms of patients with rosacea sometimes depend on chronic gastritis with hypochlorhydria. There is a feeling of fullness after meals, flatulence, and sometimes heart-burn and a sensation of nausea. The appetite is often poor, and the bowels constipated.

**Anatomy.**—In the early stages there is merely dilatation of the capillaries in the corium, particularly around the pilo-sebaceous follicles and the sweat-glands, with some small-celled infiltration. The papules are formed by chronic inflammation around the follicles. In rhinophyma there is enormous hypertrophy of the sebaceous glands, with new formation of fibrous tissue.

**Ætiology.** The primary erythema, which is the initial symptom of rosacea, probably always results from some disorder of the digestive system, although in women disease or functional disturbance of the sexual organs are thought to be important factors. It is true that rosacea, when present, is usually aggravated at the menstrual periods, during pregnancy, and at the menopause, but it is possible that their influence is an indirect one. The actual *cause* of the flushing of the central part of the face, which characterises the common type of rosacea, is not altogether clear; in some cases, at any rate in the early stage, it would appear to be a vasomotor reflex, since it may occur immediately after the swallowing of a hot drink, but the explanation of its persistence in chronic cases is not so apparent. It would seem probable that the formation and absorption of toxic vaso-dilator substances in the gastro-intestinal tract must occur, which presumably escape, unchanged by the liver, into the general circulation.

Investigation of patients with rosacea has revealed the following facts: (1) In a certain proportion of cases there is complete achlorhydria or marked hypochlorhydria, and full doses of hydrochloric acid after meals lead to striking improvement. In others the acid is normal, and occasionally there is hyperchlorhydria (*see* p. 407). (2) In most, if not in all cases, there is excessive fermentation in the intestines of carbohydrate with acid and gas formation, the fæces being hyperacid and containing an excess of streptococci and other acid-producing organisms such as *Bacillus welchii*. This, as has been suggested, is common, if not constant, in the seborrhœic state, with which rosacea is always associated. (3) In the type of rosacea, in which the flushing and secondary staphylococcal infection affects not only the central area of the face, but also the posterior parts of the cheeks and forehead, and even the ears and submaxillary regions, evidence of hepatic insufficiency, as revealed by the lævulose test, is present, and there may be actual cirrhosis of the liver.

From these facts the influence of the known predisposing causes of rosacea is easily understood. These are: (1) Abuse of gastric irritants such as alcohol, strong tea and coffee, pickles, curries, etc. (2) The presence of oral and naso-pharyngeal sepsis, which involve the constant swallowing of virulent bacteria, particularly streptococci. These are liable to set up an infective gastritis and, if they escape destruction by the gastric juice, as is bound to occur if there is a deficient secretion of hydrochloric acid, to cause intestinal fermentation. (3) Insufficient mastication, a deficient number of teeth, hasty and irregular meals. (4) An excessive carbohydrate dietary and in particular the taking of soft starchy foods, which cannot be masticated and predispose to bacterial fermentation. (5) Chronic intestinal stasis.

**Diagnosis.**—Rosacea may be distinguished from *Acne vulgaris* by the absence of comedones, and by the fact that it appears later in life. The two conditions, however, frequently coexist. In *Lupus erythematosus* there are usually well-defined patches, with raised borders and adherent scales, and superficial scarring is apt to result. Confusion with syphilitic eruptions should not arise.

**Treatment.**—The diet must be carefully revised, and all indigestible dishes avoided. Alcohol and strong coffee and tea are, of course, harmful, and should be forbidden. Oral, tonsillar, or naso-pharyngeal sepsis must be most carefully sought for, and dealt with if present. Examination of the gastric juice by the fractional method is always desirable, and in some cases an X-ray examination of the whole alimentary tract is indicated. Constipation should be corrected by the administration of liquid paraffin and saline aperients. In most cases the best results are obtained by giving an alkaline mixture half an hour before meals, and full doses of dilute hydrochloric acid (30 to 40 minims), either during or after the three principal meals of the day, until the gastric secretion becomes normal. Other drugs that are sometimes of value are bismuth and ichthyol; both should be given on an empty stomach. Locally, the main indication is to check seborrhœa and the resulting secondary infection. In mild cases calamine lotion containing 10 grains of resorcin and of boric acid to the ounce is sufficient, or the lotio sulphuris of the B.P. codex. In more advanced cases a paste containing sulphur, salicylic acid, and resorcin, with a little ichthyol, may be applied at night. Lassar's paste, containing 2 per cent. of yellow oxide of mercury, is of value if there is much pustulation, and in such cases an autogenous staphylococcal vaccine is often helpful. Dilated venules may be destroyed by electrolysis. In slight degrees of rhinophyma excellent cosmetic results may be obtained by removal of the hypertrophied tissue with the knife.

## URTICARIA

Urticaria (*urtica*, a nettle) has a close alliance with erythema. The eruption often comes out suddenly, and consists of firm, round, convex, or lenticular

elevations of the skin from ¼ inch to 1 inch in diameter, at first pink, and soon becoming white in the centre. These are called pomphi or wheals. They are scattered or closely crowded over the part affected, and are not symmetrical. They may arise very rapidly, and subside in a few hours or a day (*U. acuta*), or they last longer, or recur frequently (*U. chronica*). Sometimes the elevations are quite small (*U. papulata*), in rare cases as large as a walnut or hen's egg (*U. gigas*). Rarely a vesicle or bulla may form on the surface of the wheal (*U. bullosa*). Urticaria is accompanied with intense itching, so that the patient cannot forbear from scratching himself, and thus, no doubt, the lesion is considerably aggravated.

**Ætiology.**—The urticarial wheal is one of the commonest cutaneous reactions towards toxic substances, either when applied locally to the skin, or when absorbed from within. In some cases an urticarial eruption is a manifestation of anaphylactic susceptibility towards some foreign protein, and such susceptibility may be congenital or acquired during life. A tendency to urticaria, asthma, hay fever, and certain forms of eczema is often hereditary and appears in several members of the same family, and all these conditions are reactions which indicate an undue sensitiveness on the part of the patient to protein substances. Foreign proteins capable of inducing such reactions may be contained in food substances (*e.g.* eggs, cereals, shell fish, fruit), in plants (*e.g.* the pollens which cause hay fever, the nettle and other poisonous herbs), of animal origin (*e.g.* asthma from cats or horses, urticaria from stinging insects, fleas and bugs), or of bacterial origin (*e.g.* asthma or urticaria due to chronic infection). An erythematous and urticarial rash also occurs as part of an anaphylactic reaction after the injection of foreign sera (serum rash), and as the result of the exhibition of certain drugs. The causes, therefore, of urticaria are very numerous and often obscure. They may be classified as follows :—

*External.*—The commonest external causes of urticaria are the bites or stings of certain insects, particularly fleas, lice, bugs, and mosquitoes, and it should be remarked that certain persons are much more susceptible to such insects than others. Thus, a flea bite may produce in one a small erythematous macule with a central purpuric spot, and in another a large urticarial swelling. Contact with the hairs of some caterpillars, with certain jelly fish, and with plants, such as the nettle, is also a possible cause.

*Internal. Food Substances.*—A very large number of these are capable of provoking an attack of urticaria in different persons, the commonest being fish, crustacea, bivalves, pork and sausage, mushrooms, nuts, cereals, certain fruits and eggs. In this connection it should be noted (1) that susceptibility to a given food may apparently be congenital, or it may be acquired, *i.e.* the person may become sensitised during life ; in some cases the susceptibility is so great that the merest trace of the noxious food is sufficient to provoke an attack of urticaria ; (2) that the toxic effect may depend on incomplete digestion, whereby the food substance in question is absorbed unchanged ; this explains why a large number of oysters, for example, may provoke an attack, whereas a small number may not ; moreover, in cases of achlorhydria the administration of hydrochloric acid may prevent an attack even when a food, which usually causes urticaria, is taken ; (3) that the provoking cause may be some toxic substance produced by decomposition of the food before it is eaten (*e.g.* bad fish or meat), or by its incomplete digestion, or by excessive bacterial putrefaction in the intestines ; (4) that inflammatory conditions of the gastro-intestinal tract may predispose to urticaria by allowing the absorption of toxic substances which are not absorbed in health.

*Drugs.*—The balsams, such as copaiba, opium, quinine, salicylates, potassium iodide, veronal, medinal, arsenic and arseno-benzol, and others.

*Therapeutic sera.*

*Bacterial Infection.*—Chronic recurring attacks of urticaria may depend on

some focal infection. Thus, they may be due to oral sepsis, to infected tonsils, to chronic appendicitis, to cholecystitis, and to *Bacillus coli* infection of the urinary tract.

*Animal Parasites.*—Intestinal worms, hydatids, and parasitic protozoa.

In some individuals direct mechanical irritation will cause a local infiltration to take place almost at once, so that the scratch of a pen or the nail upon the skin is sufficient to cause a linear elevation; and thus figures or letters can be traced upon the skin lasting for some minutes (*factitious urticaria, dermographism* or *autographism*).

In some cases, according to Wright, the coagulation time of the blood is prolonged.

*Angeio-neurotic œdema* (*see* p. 383) is allied to urticaria.

**Treatment.**—This entirely depends on the cause. Not uncommonly cases are met with in which drastic internal treatment has been given, when the eruption is really due to the bites of fleas, bugs, lice, or mosquitoes. In acute cases clearly due to ingesta an emetic may be given, if the patient is seen early enough, followed by the administration of saline aperients and large doses of alkalies, such as sodium bicarbonate, combined with calcium lactate. The subcutaneous injection of liq. adrenalini hydrochlor. 10 to 20 minims, repeated, if necessary, may give immediate relief, and, as a hypnotic, chloral with or without a bromide should be given.

In chronic cases the treatment must depend on a careful examination of (1) the diet; (2) the whole alimentary tract for evidence of achlorhydria, intestinal stasis, worms, abnormal bacteria, etc.; (3) the urine for albumen, excess of indican, and for bacilluria; (4) the teeth and gums, tonsils, appendix, gall bladder, etc., for evidence of chronic sepsis; (5) the blood for diminished coagulability and high eosinophilia or other abnormality.

If focal infection is found to be the cause, vaccination may be necessary for a while after treatment or removal of the septic focus. If the coagulation time of the blood is prolonged, acid fruits should be forbidden, milk should be given freely, and calcium salts prescribed. If the secretion of hydrochloric acid by the stomach is low, the acid. hydrochloric dil. B.P. should be given in doses of 30 to 40 minims, well diluted, with meals. Local measures for relief of itching consist of frequent tepid alkaline baths, followed by sponging with a spirit lotion containing phenol (℥ss or ℥j to ℥viij), and the application of a starch dusting powder.

### URTICARIA PAPULOSA (*See Plate XLVII., C*)

(*Lichen Urticatus, Strophulus*)

Papular urticaria is a very common eruption in childhood, but it is as yet uncertain whether it should be considered merely as a variety of ordinary urticaria, which is relatively rare in infants, or whether it is a distinct disease, the cause of which is still obscure. The eruption consists of bright red urticarial lesions in the centre of which is a small indurated papule, which persists after the surrounding erythema and œdema have subsided, so that a number of these small, shotty, brownish papules may be seen mixed with recent urticarial wheals. The rash causes intense irritation, and, as it is apt to be most profuse in the evening, the child often lies awake at night scratching and rubbing himself: during the day-time itching may be absent or slight. As a result of scratching the papules become covered with small blood-crusts, and secondary infection with pyogenic cocci occurs, resulting in the formation of pustules, impetiginous lesions, and sometimes ecthymatous sores. The eruption may then imitate that of scabies very closely. Sometimes exudation may be so great that actual vesicles are formed, or very rarely bullæ, and this vesicular form of papular urticaria is not infrequently mistaken for chicken-pox.

In chronic cases the papules, conical at first, are apt to become flattened, shiny, and angular, and they frequently have a central pit; they may thus resemble the papules of *Lichen planus* very closely. Indeed, cases that have been thought to be examples of *Lichen planus infantum* have later proved to be really *Urticaria papulosa*, although true *Lichen planus* does very occasionally occur in young children. In some children acute attacks of papular urticaria occur occasionally, usually in summer-time, and between the attacks there is absolute freedom from the eruption; in others the disease is chronic, acute exacerbations appearing from time to time. It is usually, however, most severe in hot weather. As a rule it does not continue beyond the age of seven or eight years.

**Ætiology.**—The eruption usually makes its first appearance during the first two years of life. The actual cause is uncertain. It does not seem to depend on intolerance of any particular food substances, although in many cases an excessive carbohydrate dietary is certainly a predisposing cause, since restriction of sweet and starchy foods often leads to a cessation of the attacks. In some children an outbreak may invariably occur after eating certain raw fruits.

**Diagnosis.**—The disease has to be distinguished from scabies, Hebra's prurigo, varicella, and *Lichen planus*. From scabies the diagnosis may be difficult, but burrows can almost invariably be found in children with scabies, if carefully sought for. Hebra's prurigo chiefly affects the lower extremities, avoids the flexure surfaces, and is usually accompanied by inguinal adenitis. Careful observation should always prevent confusion between bullous *Urticaria papulosa* and chicken-pox: *Lichen planus* is extremely rare in children, and the papules are not preceded by urticarial wheals.

**Treatment.**—In an acute attack purgatives should be given, such as small doses of calomel followed by sodium or magnesium sulphate, and the diet should be restricted to milk and water for a day or two. In chronic cases, or in those subject to repeated recurrent attacks, the amount of carbohydrate food should be diminished, and anything which upsets the digestion, such as raw fruits, forbidden. Regular saline aperients are valuable, such as mist. alba ℥j to ℥ij every morning, and small doses of grey powder may be given at night. Calcium seems to be specific in some cases, and useless in others. It may be prescribed either as the lactate, combined with bicarbonate of soda and magnesia, or as syr. calcii lactophosphat. B.P. Small doses of arsenic, combined with syr. ferri phosph. co. B.P.C., and given for a considerable time, are often useful in chronic cases. It may be advisable during an acute attack to ensure sleep by means of hypnotics, and a combination of chloral grs. iij, pot. brom. grs. iv, aq. menth. pip. ℥j, should be given for a few nights at bedtime. A great deal can be done to relieve itching by local measures. A warm bath, to which one or two ounces of liq. picis carbonis may be added, should be taken morning and evening, and the skin afterwards powdered with warmed starch powder. The child should wear non-irritating material next the skin day and night, and it is essential that it should not be overclad. If septic infection of the skin has resulted from scratching, boric baths and a boric or weak mercurial ointment will usually rapidly overcome it.

### URTICARIA PIGMENTOSA

This is a rare disease, which should probably not be considered as a variety of urticaria. The eruption consists of round or oval macules and papules, the colour of which varies from light fawn to dark brown. They tend, when numerous, to lie with their long axes in the lines of cleavage. The pathognomonic sign of the disease is that the lesions, when vigorously rubbed or scratched, become reddened, swollen, and form urticarial wheals. The majority of cases are accompanied by itching, and dermographism is frequently seen. The disease usually

begins in early infancy, and often disappears or becomes much less evident at puberty; it may, however, persist indefinitely, and its first appearance may be delayed until puberty or even adult life. Most of the lesions are situated on the trunk and buttocks, but the limbs and head may be involved. Histologically there is œdema of the cutis, as in ordinary urticaria, increased pigmentation in the deeper layers of the epidermis, and a dense infiltration of *mast cells*, which is absolutely characteristic of the disease.

**Ætiology.**—This is unknown. The disease is much commoner in males than in females, and may be familial. The most logical view is that it is a nævoid condition.

**Treatment.**—Beyond the relief of itching, no method of treatment has hitherto had the slightest effect.

## DERMATITIS VENENATA AND TOXIC ERUPTIONS

Many forms of dermatitis can be traced to the direct application of poisonous materials or to their internal administration. Such, for instance, in a mild form is the familiar urticaria of the stinging nettle (*Urtica dioica*), and in a much more severe form the erythematous rash produced by contact with the leaves of the *Primula obconica*, and with the juice of plants of the order *Anacardiaceæ*, to which *Rhus toxicodendron* and the Indian marking-nut belong. The results of the presence of toxins and allied poisons in the blood are seen in the erythematous eruptions of the exanthems, in the occasional eruptions of pyæmia and septicæmia, in eruptions after vaccination and in the erythematous rashes which sometimes follow the injection of diphtherial and other antitoxins. *Uræmic dermatitis* is a toxic form which is occasionally seen in chronic Bright's disease towards the end of the illness (*see* p. 626). It often begins as papules or larger elevated patches of red inflamed skin, which ultimately coalesce, so that the whole body may be covered with red thickened skin. Subsequently desquamation takes place, and some cases have a close resemblance to exfoliative dermatitis. Lastly, we have the poisonous effects of certain drugs when given in undue quantity when unduly retained by inadequate elimination by the kidneys, or in cases of idiosyncrasy.

### ERUPTIONS CAUSED BY DRUGS INGESTED

The eruptions produced by drugs are erythematous, urticarial, vesicular, bullous, purpuric, or in some other form; the first four varieties are more common, and especially the first—namely, erythema. The following are the most important:

**Antipyrin.**—A red, papular or morbilliform eruption over the greater part of the body, sometimes with itching and subsequent desquamation. Purpura has also been seen, and sometimes characteristic pigmented erythematous patches.

**Arsenic.**—Urticaria, erysipelatoid rash, or small papules. Herpes zoster has occurred during the use of arsenic. The long-continued use of arsenic has caused a general pigmentation of the skin, and in psoriasis the healed patches sometimes become very deeply stained. *Keratosis*, or thickening of the horny layer of the epidermis, especially affecting the soles of the feet and palms of the hands, also occurs, and was seen in the accidental poisoning of beer-drinkers by arsenic in 1900.

**Borax and Boric Acid.**—Inflammations of the skin have occasionally followed the internal use of borax, as well as its application to the surface, and to internal cavities. The eruptions are erythematous, and sometimes bullous, or even hæmorrhagic.

**Bromides.**—An acneiform eruption is common as the result of the use of

bromides in epilepsy; the pustules are usually discrete, and occur on the face, chest, back, or scalp, and around the hair follicles on the thigh. More extensive lesions occur in exceptional cases in children on the face and limbs; these are large, oval or circular, much-raised patches of deep red colour, covered with a number of pustular points, or the thick scab which follows their rupture. The substance of the patch is soft; it mostly subsides, and the scab is detached, without leaving any scar, but only a rather persistent stain. The lesions often begin some days after the bromide has been stopped, and their appearance is favoured by any disease of the kidneys which hinders elimination of the drug. Arsenic internally is said to promote their cure, and if given with the bromide may prevent their occurrence. Erythematous, papular, and bullous eruptions are also met with.

**Chloral.**—Erythematous eruptions, diffuse redness or red papules, and occasionally purpura. They occur mostly after long-continued use of the drug.

**Copaiba.**—Erythema, consisting of bright red, roundish or irregular patches, slightly raised above the surface, here and there confluent, somewhat like measles, covering the arms, legs, trunk, and face. Purpura, vesicles, and urticaria are occasionally present. Desquamation may occur after a persistent eruption.

**Cubebs** seems occasionally to produce a similar rash.

**Iodides.**—The eruptions are erythematous, pustular, vesicular, bullous, nodular, or purpuric (*see* p. 542). The erythema is papular, and occurs over the trunk, face, and limbs. Pustules are seen like those of the bromide rash, but smaller in size when discrete; and the confluent forms are less common, and tend to be more bullous. Sometimes large bullæ occur, with a very narrow areola around each, and clear serous contents. Like the bromide eruption, it may be delayed for some days after the drug has been stopped, and is more likely to appear if the kidneys are diseased. The addition of arsenic or aromatic spirits of ammonia to an iodide mixture, or taking the dose in half a tumblerful of water, may be tried to prevent its occurrence.

**Quinine.**—Erythematous rashes are most common, either diffuse or papular; an urticarial form is next most frequent; both of these produce severe itching, and erythema may be followed by extensive desquamation. Purpuric, vesicular, and bullous rashes are less often seen.

Other drugs that have more or less frequently caused rashes, mostly of an erythematous or urticarial type, are belladonna, cannabis indica, potassium chlorate, chloroform (inhalation), cod-liver oil, digitalis, iodoform, mercury, morphia, opium, phenacetin, phosphoric acid, salicylic acid, santonin, strychnia, stramonium, sulphonal, tar, terebene, and turpentine.

The **Treatment** should be the withdrawal of the drug and the use of astringent lotions, such as those of subacetate of lead, oxide of zinc, or calamine.

### ERUPTIONS CAUSED BY DRUGS APPLIED EXTERNALLY

Many substances are applied externally in order to cause erythema and vesication for their therapeutic effects, such as cantharides, capsicum, mustard, croton oil, and turpentine. Belladonna, iodine, sulphur, mercury ointments, arnica, chrysarobin, and pyrogallic acid are also likely to cause irritation if too long applied to sensitive skins.

An important and frequent cause of dermatitis of the face and neck, and sometimes of other parts of the body, is the use of certain lotions and dyes for the hair. Of these the most important is paraphenylendiamine, which is contained in several proprietary hair dyes and is also used to dye furs. It may produce a most violent dermatitis, which usually begins on the face, ears, and neck, and is apt to cause intense irritation and swelling of the eyelids; in severe cases the eruption may spread on to the chest, back, arms, and even to the lower limbs,

and there may be fever, a marked leucocytosis with eosinophilia, and sometimes albuminuria. Numerous other substances, many of which are used in various trades and occupations, will in certain individuals provoke a dermatitis. Of these may be mentioned many aniline dyes, salts of chromic and picric acid, formalin, photographic developers, such as metol, alkalies and acids, and antiseptics, such as corrosive sublimate, biniodide, lysol, and iodoform. In these cases of dermatitis from external irritants (*Dermatitis venenata*) there may be an innate susceptibility of the individual affected, or such susceptibility may be acquired from frequent exposure to the irritant in question. A person, once he has become susceptible, is liable to a recurrence of the dermatitis even after exposure to a minute quantity of the toxic substance, although previously he may have freely exposed himself to it without ill effect.

## TRAUMATIC AND SOLAR DERMATITIS

Apart from surgical conditions, *traumatic dermatitis* may be recognised in the marks of scratching, such as are seen in pruritus, jaundice, scabies, and phtheiriasis, to which the reader is referred. These effects are in part due to repeated infections by pus organisms.

Another form of traumatic dermatitis is that intentionally produced in order to *feign* disease, an event most common among young women, who may use nitric acid, mustard, cantharides, iodine, or other irritant. The site of the lesion is generally the breast or a limb, at least a part accessible to the right hand; the lesion is generally redness with or without vesication or pustulation; but it may be continued until ulceration is produced.

*Solar dermatitis* (formerly called *eczema solare*) is well known to follow unwonted exposure to the rays of the sun as reflected from the cricket field, the river or sea, and especially from Alpine snowfields. There is intense redness and swelling, with formation of vesicles and bullæ, accompanied by itching and smarting pain, and followed by free desquamation and pigmentation. Bowles believed that this was due rather to the light rays (*actinic*) than to the heat rays, and his contention appears to be borne out by the effects of light used in the treatment of lupus, etc. Of analogous origin is the severe and persistent dermatitis which results from prolonged exposure to the *Röntgen rays*.

## ECZEMA (*See Plate XLVI., D*)

A great deal of confusion still exists as to the precise significance of the word *eczema* (ἐκζέω = I boil over), and there are some who would abolish it altogether and substitute the term *dermatitis*. If, however, we regard eczema as a form of reaction of the skin, possessing well-defined clinical and histological features, and not as a disease *sui generis*, the difficulty is solved, and the disputes, that have occurred among dermatologists from the time of Willan and Bateman as to what is and what is not eczema, now appear trivial, and certainly serve no useful purpose.

Eczema is an inflammatory process involving the epidermis and dermis, and, unlike eruptions such as urticaria or lichen planus, possesses no single characteristic lesion, but consists rather of a number of lesions which differ in the various stages through which the inflammatory process passes.

Although the evolution of these different stages varies in different cases, it may be traced as follows: The earliest, or *erythematous stage*, is represented by a patch of erythema, the borders of which are usually ill defined, accompanied by a certain amount of œdema of the affected part, and associated with a sense of tension, burning, or itching. Occasionally the inflammatory process progresses no farther, its subsidence being followed by a fine desquamation, after which the skin rapidly regains its normal appearance. Usually, however, a number of

small superficial vesicles appear on the erythematous area, and this, the *vesicular stage*, is one of the most characteristic phases of the eczematous process. By confluence of the vesicles actual bullæ may be produced. In situations where the epidermis is naturally thick, as on the palms and soles, the vesicles appear deeply imbedded in the skin, and may dry up without breaking, but as a rule they soon burst, either spontaneously or as the result of scratching, and discharge a clear serous fluid, which is albuminous and stiffens linen, thus giving rise to the *weeping stage*. The fluid, after flowing for a little, dries up into yellowish crusts which adhere to the surface until detached by accident or lifted by discharge underneath (*encrusted stage*). As the inflammation subsides, the eczematous area becomes paler, the discharge ceases, and healing takes place by a regrowth of epidermis. This may occur spontaneously under the crust, which may remain long after recovery is advanced. For some time, however, the formation of the new horny layer is imperfect (*parakeratosis*), so that it remains thin and transparent, and is cast off as large flakes or papery scales (*stage of desquamation*). Eventually under favourable circumstances the skin regains its normal appearance.

As a result of secondary infection of the eczematous surface with pyogenic cocci the secretion may become purulent, and the crusts formed are then opaque and greenish-yellowish or orange-coloured, as in true impetigo (*impetiginised eczema*).

Lastly, in cases in which the eczematous process becomes chronic, the persistence of the inflammation and constant scratching and rubbing of the parts by the patient lead to thickening of the skin, with hypertrophy of the epidermis and infiltration of the dermis. This change, known as *lichenification*, will be described under the heading of Prurigo.

Locally, eczema gives rise to severe itching (*pruritus*), smarting, or burning, and in certain positions to pain on movement. The general condition of the patient, even in cases with an extensive eruption, may be but little affected. In acute cases there is often slight fever, and the digestive system may show signs of derangement, such as a coated tongue, foul breath, anorexia, and excessive thirst. In chronic cases the insomnia resulting from the constant itching, which usually reaches its acme of intensity at night-time, may lead to severe mental depression and irritability, thus seriously interfering with the proper performance of the patient's duties.

**Ætiology.**—Since eczema is merely a form of cutaneous reaction towards a great variety of irritants, which may reach the skin either by direct external application or through the blood stream by absorption from within, it is clear that a detailed exposition of all its possible causes is impracticable. A tentative classification of the different varieties of eczema, based as far as possible on their causation, may however be attempted.

*Eczema due to External Factors.*—Eczematous dermatitis may be produced by exposure of the skin to both physical and chemical irritants :—

*Physical.*—Mechanical irritation of the skin by friction, pressure, or constant rubbing and scratching, not infrequently results in local eczematisation. As examples may be quoted truss-dermatitis, lichen simplex, and Whitfield's "cross-legged" eczema, which occurs sometimes on the outer side of the legs just above the external malleolus in those who habitually sit with one ankle crossed over the other knee. Other physical causes are exposure to great heat, *e.g.* stokers' or blacksmiths' eczema, and to extreme cold, particularly cold winds, and to actinic rays (*eczema solare*).

*Chemical.*—A very large number of chemical substances are capable of provoking an acute or chronic eczematous dermatitis when brought into contact with the skin, and many of them are employed in various trades and professions. Thus the constant use of highly alkaline soaps and washing soda is a very frequent cause ("washerwomen's eczema"); exposure to salt and brine may produce

"butchers' eczema," to metol "photographers' eczema," to antiseptics (particularly formalin, perchloride and biniodide of mercury, lysol) "surgeons' eczema." Grocers and sweet manufacturers are liable to eczema from handling sugar. Aniline dyes, picric and chromic acids, hair dyes (particularly those containing paraphenylendiamine), hair lotions, mouth washes and tooth pastes, turpentine, sulphur, etc., are also common causes.

In "bakers' eczema" the skin of the parts exposed to the flour may be sensitive to some or all of the proteins contained in wheat. An important point to remember is that in many instances the skin of certain parts may become susceptible to an irritant, whereas other parts remain immune. Eczema due to these external irritants, as well as that produced by certain plants, has already been referred to under the heading of Toxic Dermatitis. Although in these cases the provoking cause of the dermatitis is an external one, other factors must often be taken into account. There is no doubt that the general constitutional condition in some way influences the liability to occupational eczema, and the development of susceptibility to a given irritant may correspond to the onset of ill-health; dyspepsia, associated with oral sepsis and constipation, would seem in particular to be a predisposing cause, and it is probable that incomplete digestion and assimilation, and inefficient excretion of toxic waste products, render the skin more susceptible to external irritation. In some cases, at any rate, when the general health is improved, immunity to the irritant causing the dermatitis is completely or partially recovered.

The seborrhœic state, which will be referred to later, is another important predisposing factor to the development of eczematisation, and its prevalence among our soldiers in France accounted, partly at any rate, for the severity of the secondary dermatitis that so frequently accompanied scabies. Moreover, seborrhœic persons were found to be more susceptible to mustard-gas dermatitis, and they are very liable to eczema after even a moderate use of sulphur ointment for the treatment of scabies.

Lastly, in ichthyosis, and in persons with very dry skins, there is naturally an abnormal tendency to eczema both from exposure to cold wind and from the use of alkaline soaps, soda, and other chemicals which are fat solvents.

*Eczema due to Pyogenic Organisms.*—Unna at one time held the view that eczema was due to infection of the skin with a specific organism—the morococcus—by which term he was probably describing more than one variety of staphylococcus; but Sabouraud, as a result of careful research, insisted that the primary vesicle of eczema was non-microbic. At the present time it is generally admitted that both staphylococci—particularly the *S. aureus*—and streptococci are capable under certain conditions of producing an eczematous reaction in the skin.

*Dermatitis Infectiosa Eczematoides.*—Under this title Engman, and later Fordyce and Sutton, have described a form of eczematoid dermatitis which usually results either from trauma, a localised infected sore, or from suppurative conditions, such as furunculosis, wounds, chronic otitis media, discharging sinuses, etc. It is not uncommonly seen after an attack of scabies with secondary infection. The earliest lesion is generally a vesicle or pustule, and the eruption occurs in circumscribed patches of varying size, which spread by peripheral extension. The epidermis at the margin of the patches is often raised, forming a scaly frayed edge. The condition appears to depend on the fact that sensitisation to the *Staphylococcus aureus* has occurred owing to some preceding suppurative process, and this organism is usually found in the lesions in pure cultures. Positive cutaneous reactions to the staphylococcus are often obtained, and Sutton has pointed out that in about 25 per cent. of cases urticarial lesions may accompany the eruption.

*Streptococcal Dermatitis*—This form of eczematous dermatitis is most commonly seen in folds, around orifices, or in the neighbourhood of infected wounds,

or septic ulcers A chronic fissure in the skin is often present, *e g.* behind the ears, between the toes, in the groins, at the angles of the mouth (*la perlèche*), the nose, eyes, or anus, which acts as a chronic focus of infection. The affected area is reddened, œdematous, and shiny, and at the margin there is often a sharply-defined scaly edge, or there may be some vesiculation Many cases of so-called varicose eczema or eczema rubrum are really of this nature, although the milder forms of the former are due to the staphylococcus. It should be remembered that chronic or relapsing lymphangitis, leading sometimes to elephantiasis, may accompany streptococcal infection of the skin.

*Eczema of Internal Origin.*—Although our knowledge of eczema due to causes other than external irritation has increased considerably during recent years, it is still far from being complete. One fact, however, has been definitely established, namely, that eczema, like urticaria and asthma, may be, particularly in children, a manifestation of protein-sensitisation (*see* Toxic Idiopathies, p. 224). In these cases the influence of heredity is often very evident, and a tendency to asthma, hay fever, urticaria, or eczema, may be transmitted through many generations and may occur in several members of one family. Most persons with this inherited tendency are susceptible to more than one substance, and they may react differently towards the various proteins to which they are sensitive. Thus a horse-asthmatic may develop urticaria should he eat shell-fish, whereas other food substances, such as certain cereals, may provoke in him an attack of eczema. It is in children that eczema due to food sensitisation is most frequently seen, for in adult life tolerance of most foods has usually been acquired. Cereals, such as oats and wheat, would seem particularly prone to cause eczema, but other foods, such as cows' milk, cabbage, pork and beef, are also capable of provoking it in susceptible persons

Apart from food sensitisation, certain cases of eczema, mostly in adults, appear to depend on *focal infection*, and it is probable that in some instances, at any rate, the eczema is a manifestation of bacterial sensitisation. Oral sepsis (*see* p 392) particularly is an exciting cause, and numerous cases have now been recorded in which eczema of long standing has disappeared rapidly after removal of infected teeth. The association of *gout* and eczema has probably been much exaggerated, and, although the two conditions may certainly co-exist, it is rare to see a case of true tophaceous gout in a dermatological clinic. Concerning the connection between eczema and *renal disease* little is definitely known, but in uræmia a generalised eczematous and exfoliative dermatitis may arise ; in such cases the amount of urea in the blood is always very high and the eruption is of grave significance, death usually occurring within a few days of its appearance. *Glycosuria* may cause a localised eczema around the vulva, or prepuce, due to irritation by the saccharine urine, but, apart from this, eczema is not uncommon in diabetics. Moreover, an excessive intake or defective assimilation and digestion of carbohydrate are certainly predisposing causes of eczema both in children and adults ; but whether this depends directly on hyperglycæmia is not yet clear. Several observers have emphasised *nervous shock or strain* as a provocative cause of eczema, and in some persons an acute outbreak may certainly follow a severe shock or period of worry, but the association is probably an indirect one. *Intestinal toxæmia*, associated with excessive putrefaction and indicanuria, is apparently responsible for one variety of eczema, the eruption affecting chiefly the parts of the skin exposed to light, namely, the face, ears, neck, and the backs of the hands ; it is possible that in these cases some toxin is absorbed from the intestines which sensitises the skin to sunlight.

The term *seborrhœic eczema* has unfortunately been confused with seborrhœic dermatitis; the latter is a parasitic infection, in which, perhaps, the *Staphylococcus albus*, bottle bacillus and the microbacillus are all concerned, and can be readily cured, as a rule, by applications containing sulphur and salicylic acid. Seborrhœic eczema, however, is probably primarily non-microbic, although,

owing to the rapidity with which oozing takes place, secondary infection occurs very quickly. It begins as discrete erythematous patches, usually in situations where the sebaceous glands are numerous and large, *e.g.* the eyebrows, centre of the forehead, nose, ears, scalp, beard region, neck and joint flexures. Within a few hours vesiculation and weeping may take place, and the condition spreads rapidly. The sebaceous glands are hyperactive, and in the acute stage, even in cold weather, droplets of highly acid sweat may be seen on the nose, forehead and upper lip. There is often an accompanying nasopharyngeal catarrh. The urine is usually very acid, and the ammonia ratio high. In early cases, as a rule, the administration of large doses of alkali, even without local treatment, will rapidly cut short the attack. Very large doses may, however, have to be given before the urine turns alkaline. This form of eczema was extremely common during the war among our soldiers in France, and was usually wrongly diagnosed as impetigo or seborrhœic dermatitis; since the war it has become much less frequent.

**Morbid Anatomy.**—The histological changes met with in the skin in eczema vary according to the stage of the disease. In acute eczema there is great dilatation of the capillaries in the papillæ with œdema and some leucocytic infiltration. The transitional epithelium becomes œdematous, the prickle cells are separated and disintegrated by the serous exudation (*spongiosis*), and thus vesicles are formed. As a result of these changes in the epidermis the process of keratinisation is interfered with. In the more chronic stages there is marked thickening of the interpapillary processes (*acanthosis*), and more and more infiltration of the corium, with connective tissue proliferation; owing to the imperfect keratinisation (*parakeratosis*), scales consisting of clumps of nucleated cells are formed, and periodically shed. In old patches the thickening of the skin may be very great.

**Treatment.**—Since the possible causes of eczematous inflammation of the skin are so numerous, it follows that successful treatment must depend, as a rule, on accurate diagnosis. Thus many patients have been treated for years for "gouty eczema" of the hands and feet when they were really suffering from eczematoid ringworm, and in recent years recurrent attacks of acute eczema, due to the use of certain proprietary hair dyes, have become increasingly common, and have often been regarded as of constitutional origin. It cannot be too strongly insisted upon that in every case of eczema the possibility of an external irritant being responsible should first be considered. The distribution of the eruption and the history of its onset will usually help one in determining this point, but, although the cause may obviously be an external one, its discovery may be a matter of great difficulty.

Whatever the cause, the local treatment depends on the stage of the inflammatory process. Thus in acute cases with intense hyperæmia, vesiculation, or weeping, astringent lotions repeatedly applied should be used; when the eczematous surface is drying up, a protective paste or cream should be substituted for the lotion, and in the chronic scaly stage ointments may be employed. The following are suitable lotions in the acute stage: (1) Liq. plumbi subacetat., 1 dr.; Spirit. vini rect., 1 oz.; Aq. dest., ad 8 oz., to which may be added aluminium acetate in a strength of from 1 to 3 per cent.; this should be applied on a single layer of lint, which must be kept continually moist. (2) ℞ Calaminæ præpt., Zinc oxide, āā, 2 dr.; Glycerini, 1½ dr.; Spirit. vini rect., 1½ oz.; Aq. dest., ad 8 oz. (3) Lotio nigra and Aq. calcis, equal parts. (4) Ichthyol, 3 per cent in water. In some cases a calamine liniment is preferable to a lotion, *e.g.* ℞ Pulv. tragacanth., 1 dr.; Phenolis, Glycerini, āā, 10 mins.; Zinc oxidi, Calamine præpt., āā, 1 oz.; Ol. olivæ, 4 oz.; Aq. q.s., ad ℥j.

As a paste the well-known formula of Lassar is invaluable: ℞ Zinci oxidi, Pulv. amyli, āā, 2 dr.; Acidi salicylici, 10 gr.; Paraffini mollis alb., ad 1 oz.; this may be modified by omitting the salicylic acid, which sometimes proves

irritating, and by adding a little lanoline. This paste or a zinc cream, *e.g.* R Zinci oxidi, ½ oz.; Ol. olivæ vel amygdalæ dulc., ½ oz.; Adip. lanæ. anhyd., 1 dr.; Aq. calcis, 3 dr., may be used alone or as a base for the incorporation of ichthyol, wood or coal tar, sulphur, resorcin and ammoniated mercury, etc. In subacute or chronic cases, in which these stimulating substances are indicated, the tolerance of the skin towards them must be tested gradually by prescribing them at first in small quantities.

In very chronic patches in which lichenification has occurred crude coal tar, pyrogallol, eugallol (the monoacetate of pyrogallol), and even chrysarobin may be employed. Crude coal tar may be painted on neat or incorporated either with the pigmentum caseini B.P.C. or a zinc paste (*e.g.* crude coal tar, 2 parts; zinc oxide, 2 parts; corn starch, 16 parts; petrolatum, 16 parts); it should never be employed when secondary pyogenic infection has occurred If used neat, the eczematous surface having been first cleansed with soap and water, the tar is applied as a uniform coating and allowed to dry. The applications may be repeated daily for several days if well tolerated. Eugallol is a dark liquid containing 33·3 per cent. of acetone, and should be applied once every few days. Chrysarobin is best employed in an ointment, and its effects must be carefully watched.

The X-rays afford one of the most valuable methods of treating these chronic infiltrated patches. Either a full pastille dose may be given and repeated, if necessary, in a month's time, or fractional doses of a third of a pastille at intervals of ten days. In suitable cases rapid relief from itching is obtained by the use of the rays, and resolution of the thickened patches takes place in a few weeks.

The question of washing must always be considered in the management of a case of eczema, whatever be the cause. Soap should never be used in the acute or subacute stages, but in chronic cases a superfatted soap, with or without the addition of tar or ichthyol, is sometimes of value Water should be avoided altogether in acute cases, but in subacute and chronic eczema sponging or bathing with a 1 per cent. salt solution seldom does harm, and is often beneficial, for, as Whitfield suggests, the irritant effect of plain water probably depends on the fact that it is hypotonic.

*Internal Treatment.*—Apart from local applications, the treatment of eczema must obviously depend on the cause, and on the state of health of each individual patient. As already indicated, even in cases in which an external irritant is the provoking cause, internal treatment, particularly of the digestive tract, is often required, and may materially hasten recovery. *The diet* is of importance, particularly in some infantile cases, and the rules to be observed differ according to the type of eczema and the state of the patient's digestion. Alcohol and excess of tea and coffee should be forbidden in all cases. In acute cases and in some chronic ones, associated with intense itching, benefit may accrue from fasting for from two to six days with the administration of saline purgatives in the early morning and of large draughts of water containing alkaline diuretics throughout the day. In seborrhœic cases the carbohydrate intake should be reduced and a diabetic diet prescribed for a while. On the other hand, in patients with marked indicanuria, in whom the secretion of hydrochloric acid by the stomach is often low, the protein intake should be diminished and a lacto-vegetarian *régime* instituted. In some infantile cases it is advisable to restrict the amount of carbohydrate, and in others it will be found that an excess of milk is being taken.

In old people in whom the skin is abnormally dry and the general nutrition poor, and in ichthyotic persons, remarkable improvement is often derived from the administration of cod-liver oil and thyroid gland. The former may be combined with malt or iron, and the latter should be given at first in small doses, which should be increased according to tolerance. The importance of treating oral sepsis can hardly be exaggerated, for cases of eczema which have resisted all other measures sometimes yield rapidly after the removal of teeth with root

abscesses or the treatment of pyorrhœa alveolaris. Lastly, in patients with physical or nervous exhaustion a complete rest, followed by change of surroundings, should be ordered whenever possible.

Certain varieties of eczema may now be considered separately, and the special treatment suitable for each indicated briefly : –

*Dermatitis Infectiosa Eczematoides. Locally.*—In acute cases astringent lotions, such as lotio plumbi containing aluminium acetate, or cold boric compresses, should be employed alternately with a dusting-powder of talc and zinc oxide combined with boric acid (10 per cent.) or salicylic acid (1 to 2 per cent.). In subacute or chronic cases Lassar's paste containing ammoniated mercury (2 to 5 per cent.) is often successful, and the patches may be painted occasionally with a 2 to 5 per cent. solution of silver nitrate in spirit. ætheris nitrosi. *Internally* a mixture containing sodium bicarbonate and sodium citrate, 30 grains of each, should be given before meals. In resistant cases an autogenous staphylococcal vaccine in doses of 250 to 2,000 million at weekly intervals is often of great value.

*Streptococcal Dermatitis.*—In this form of eczematous dermatitis, as has been pointed out, there is usually present some chronic focus of infection, such as an ulcer, a discharging sinus, or a fissure either at one of the muco-cutaneous junctions, as at the angle of the mouth (*perlèche*) or nose, or in a fold, as behind the ears or in the groins. It is essential that this infective focus be completely healed, or recurrence of the dermatitis will inevitably take place ; and to obtain this result the lesion, as well as the surrounding infected area, should be painted daily with a 2 to 5 per cent. solution of silver nitrate in distilled water or spirit. ætheris nitrosi ; after this a paste or ointment containing a mercurial should be applied, *e.g.* ℞ Hydrarg. oxid. flav., gr. x ; Past. zinci co. B.P.C., ad ℥j ; or the Ung. hydrarg. nitratis dilut. B.P. In cases in which there is an acute spreading dermatitis the affected area should first be dressed with the lead-aluminium lotion until the surface has become comparatively dry.

*Eczema due to Protein-sensitisation.*—The successful treatment of these cases usually depends on the discovery of the provoking cause. For this purpose the cutaneous reactions to various food and other proteins may be tested by the method described in the section on Toxic Idiopathies (*see* p. 224). In cases of food-sensitisation the offending protein should, as far as possible, be omitted from the dietary, or one may attempt to desensitise the patient either by giving minute and increasing doses of the toxic food-substance by mouth, or by injecting small quantities of the protein in solution subcutaneously or intramuscularly. In America eczema due to poison-ivy (*Rhus toxicodendron*) is now being treated with success by intramuscular injections of a tincture prepared from a fluid extract of the plant. It should be remembered that in the case of food proteins toxic symptoms may depend on their non-digestion, and in some cases of achylia gastrica the administration of hydrochloric acid may enable the patient to take with impunity articles of food which otherwise prove harmful. In cases of eczema and of urticaria in which the eruption is thought to depend on sensitisation to some toxic protein absorbed from the intestines, but in which its actual nature cannot be determined, the method described by Danysz is sometimes strikingly successful ; this consists of injecting a vaccine prepared from fæcal bacteria killed at 70° C. in small, increasing doses every three or four days. This method is doubtless analogous to non-specific protein therapy.

*Seborrhœic Eczema.*—The local treatment of seborrhœic eczema depends on the acuteness of the inflammatory process. In the early acute stage calamine lotion or liniment or lead lotion should be applied ; in the dry, subacute, or chronic stage Lassar's paste, to which sometimes a little ammoniated mercury may be added, is the best application. The internal treatment is of great importance. In the acute stage the diet should be very light, carbohydrate and fat being reduced to a minimum. Large quantities of water flavoured with lemon or orange juice should be taken, and the following alkaline mixture should

be prescribed three or four times daily on an empty stomach: ℞ Sod. bicarb., ʒj to ʒij; Potass. citrat., ʒss to ʒj; Aq. menth. pip., ad ʒj. The bowels should be kept loose with morning saline aperients. The alkaline mixture should be given until the urine is strongly alkaline; the dosage may then be reduced, but it is advisable to continue the administration of alkali for several weeks, as relapse often occurs when it is discontinued. In patients subject to recurrent attacks the diet must be very carefully revised. Sweets, jam, marmalade, cakes, pastry, and starchy puddings should be forbidden absolutely, and bread, potatoes and sugar should be taken sparingly. Meat, fish, eggs in moderation, fresh and cooked fruit and green vegetables *ad lib.* should form the chief articles of diet in these patients.

## ERYTHEMATO-SQUAMOUS ERUPTIONS

### PITYRIASIS ROSEA (*See Plate XLVI., B*)

*Pityriasis rosea* (Gibert) is chiefly of importance in that it is so frequently wrongly diagnosed, and yet the eruption is, as a rule, so distinctive that its recognition should rarely be difficult. In probably the majority of cases a single patch of considerable size precedes the generalised outbreak of the eruption by several days, but its appearance may pass unnoticed. This "herald patch," or *plaque primitive*, is larger than the subsequent lesions, and is usually seen on the neck, trunk or proximal parts of the limbs. It is more or less circular, scaly, and has a well-defined border; it is often mistaken for a patch of ringworm or of seborrhœic dermatitis. The generalised eruption appears with comparative suddenness, like that of an eruptive fever, first on the upper thorax, and then successively on the neck, the upper arms, the abdomen and back, and on the thighs. A few patches may be seen on the forearms, the face, and lower parts of the legs, and occasionally almost the entire skin, even the scalp, may be profusely covered. The lesions begin as small, pink papules, which enlarge by peripheral extension to form the characteristic circular or oval patches. A typical patch consists of an outer, raised pink border, and a central, fawn-coloured portion, which presents a wrinkled appearance; between the two is a fine collarette of scales, the free edges of which lie towards the centre. As each patch enlarges the centre may fade almost completely, so that the appearance of rings is assumed, the rosy tint of the ring contrasting with the slightly yellowish tint of the enclosed area. A very characteristic feature of the fully developed eruption is the way in which the oval patches tend to run obliquely round the chest and flanks, their long axes lying roughly parallel to the direction of the ribs. The outbreak of the disease may be accompanied by slight malaise, with sore throat and mild fever, but usually constitutional disturbance is not present. Itching may be entirely absent, slight, or so severe that the patient cannot sleep. The lymphatic glands may be enlarged, particularly the posterior cervical group.

**Ætiology.**—The disease should probably be classed among the exanthems, although the actual cause is unknown. It is perhaps commonest in early adult life, but may be met with even in old age. Although cases occur all the year round, it is certainly most prevalent in early spring. Second attacks are very rare; more than one case may be met with in the same family, but there is little evidence that the disease is contagious.

**Diagnosis.** So characteristic is a typical lesion, with its pink border, its collarette of scales, and wrinkled yellowish centre, and so constant the distribution and arrangement of the patches, that the disease can usually be diagnosed at a glance by one familiar with it, and yet errors are often made. It may be confounded in its early stage with tinea circinata or with seborrhœic dermatitis (pityriasis circinata); in its developed stage with a secondary syphilide, or even psoriasis. Tinea circinata yields a fungus on microscopical examination

of the scales. Seborrhœic dermatitis consists of greasy, figurate patches, with follicular papules, and occurs chiefly on the mid-line of the chest and back, and often on the scalp and face. Pityriasis rosea should never be mistaken for a roseolar syphilide, since the latter is never scaly, and from the papular syphilide it can be easily distinguished, since the papules of syphilis are darker in colour, are markedly infiltrated, are situate on the distal portions of the limbs as well as on the face and scalp, and are accompanied by other signs of syphilitic infection, such as sore throat, mucous patches, and marked generalised adenitis; moreover, a primary sore can usually be discovered. In psoriasis the scales are larger and present the well-known silvery appearance when gently scratched, and the eruption is commonly present on the elbows, knees and scalp.

**Treatment.** The eruption disappears spontaneously in from three to eight weeks. If irritated, however, by unsuitable applications, such as sulphur or mercury, the duration is lengthened. The patient should take a daily bath, to which two or three tablespoonfuls of Condy's fluid may well be added, after which the skin should be dusted over with the pulv. acidi salicylici comp. B.P.C., or a weak salicylic ointment may be used. Whitfield recommends the application of the following lotion twice daily: ℞ Pulv. calamin præp., ℥ij; Pulv. zinci oxidi, ℥ij; Sp. vini rect., ℥j; Liq. picis carb., ℥jss; Aq. rosæ, ad ℥viij. It is doubtful whether internal treatment has any influence on the course of the disease.

## PSORIASIS (*See Plate XLVI., C*)

This disease consists in the formation of raised red patches covered with thick silvery-white adherent scales. In a great number of instances the lesions appear first on the knee over the patella, ligamentum patellæ and tubercle of the tibia, and on the elbow over the olecranon. It begins with papules, which enlarge into large flat plaques; quite early the papule is seen to be covered with an opaque scale, and with its enlargement in size the scale becomes thicker, especially in the centre, and silvery white in appearance. The scale is rather firmly adherent, and co-extensive with the red plaque, so that the red colour can often only be seen at the edge. If the scale is removed, it leaves a shining, moist-looking, but actually dry bright red surface, in which examination with a lens will show a number of deeper red points, the hyperæmic papillæ. The patches are at first round or oval, and enlarge to ½ inch, 1 inch, or more in diameter; fresh patches come out near the first or in other parts of the body. If a patch becomes very large, it may heal in the centre, and thus form a ring; coalescence with other rings will produce serpiginous or gyrate figures. The patches may spread sufficiently to cover large areas of the body continuously, so that the original shape of the spot cannot be detected. The old names given to indicate these different stages have little more than a descriptive value, such as *P. punctata, P. guttata, P. nummularis, P. circinata, P. gyrata, P. diffusa, P. universalis.*

Next to the knees and elbows, the adjacent extensor surfaces of the leg and forearm are most commonly affected, and then the thighs, back, loins, chest, and abdomen; and in all regions a very striking symmetry is observed. The scalp and face are usually attacked when the eruption is at all widespread, but the palms and soles are often spared. The nails are not infrequently involved: they become variously altered, opaque, thickened, pitted, furrowed transversely, or immensely thickened and discoloured.

The amount of scale varies in different instances or in the same case at different times. In *P. rupioides* the scales are heaped into small conical masses, each on its circular base.

The eruption is usually dry, but may become moist in the flexures, or as the result of irritant applications, and in seborrhœic persons the patches tend to be greasy. Itching is variable, but is not, as a rule, severe. Although it is often

stated that psoriasis occurs in persons in perfect health, this is seldom or never true.

The disease breaks out spontaneously, often in early childhood, and even if not treated subsides after three or four months, to recur again after a quite uncertain interval. Sometimes the recurrence is twice a year ("spring and fall," as it is often expressed), or a period of years may intervene. In other cases a slight amount of eruption persists, and extensions take place from time to time. During recovery pigment stains mark the situation of the patches, especially after the use of arsenic.

**Ætiology.**– Although the actual cause of psoriasis is still unknown, a consideration of the evolution and course of the disease points to it being due to a systemic infection with a micro-organism of low virulence, against which little immunity is developed. As Civatte has insisted, a comparison of the histological features of psoriasis and of the psoriasiform type of seborrhœic dermatitis, which may clinically resemble each other very closely, suggests that the lesions of the former are inflammatory reactions produced in the skin by a blood-borne toxin or micro-organism, while those of the latter are due, as, indeed, is known, to an external microbic infection. It has been said that psoriasis is a disease of healthy people, but this is hardly accurate, for a careful investigation of the majority of cases shows that the patients present symptoms, sometimes vague, sometimes well defined, suggesting an underlying chronic infection. The association of rheumatism—using the word in its widest sense—with psoriasis has been long recognised, and the eruption coexists with chronic rheumatoid arthritis more frequently than can, perhaps, be explained by mere coincidence. This association (*psoriasis arthropathique* of the French) has been especially studied, and it has been suggested that both the psoriasis and the joint affection are dependent rather on some metabolic disturbance than on an infection (**15**). There would seem, however, no reason to differentiate the arthritis from the chronic infective type. Apart from definite arthritic changes, a large proportion of patients with psoriasis complain of rheumatic pains in the muscles and joints, particularly in damp weather or after exertion, and in men impotence is a curiously frequent symptom.

One striking clinical fact, which must presumably have some bearing on the ætiology of the disease, is that an acute outbreak of the eruption often takes place after an attack of tonsillitis; in such cases the whole body may be covered within a few days, the outbreak resembling, in its suddenness, that of one of the exanthemata. This would suggest that the throat may be the portal of entry of the infection in some cases.

The eruption known as keratodermia blennorrhagica, which is seen rarely as a complication of gonorrhœal arthritis, resembles psoriasis very closely both clinically and histologically. Either the eruption may be psoriasis, modified by a co-existent gonorrhœal infection, which is unlikely, or psoriasis may be caused by an organism closely related to the gonococcus (**16**). From a consideration of these facts it would appear likely that psoriasis will eventually be shown to be due to infection with a specific organism, perhaps a species of the streptococcal group, and that the throat in a certain number of cases is the primary source of invasion.

Several cases have been published in which psoriasis appeared first at the site of vaccination, but it is uncertain whether this indicates a local inoculation of the virus of the disease, or whether the trauma produced by vaccination induces a localised outbreak of the eruption in a person with latent psoriasis. That trauma of the skin frequently does determine the localisation is well known, but the same is true of lichen planus, the syphilides, and some other eruptions. Heredity is certainly a factor of importance, although this has been denied by some authors. A familial incidence is far too common to be accounted for by mere chance. Psoriasis may occur at almost any age from two years upwards,

but appears most commonly in adolescence. In women it may disappear during successive pregnancies, recurring in the intervals, but, on the other hand, it may be much aggravated during pregnancy; not infrequently at the time of the menopause localised persistent patches arise in the groins and perineum, and on the hands and feet, which are most resistant to treatment. These facts indicate that the endocrine glands have some influence on the course of the disease, and in a certain number of cases the internal administration of thyroid gland has a remarkably beneficial effect.

**Pathology.**—It is an inflammation of the papillæ and corium, with increase of the stratum mucosum, downgrowth of the same between the papillæ, which appear correspondingly enlarged, and increase of the horny layers (*keratosis*). The silvery appearance of the scales is due to the inclusion of minute air globules. The eosinophil leucocytes in the blood are often increased.

**Diagnosis.**—In most cases it cannot be mistaken. Patches of dry *eczema* may resemble it, but the edges are not so sharp, and the scales are not so thick and silvery. In psoriasis of the scalp the scales are often yellow, and look like crusts of impetigo; but psoriasis spreads beyond the scalp on to the forehead or neck, and there is usually psoriasis of some other parts of the body which will be distinctive. *Lichen planus* and *pityriasis rubra* must be distinguished by the descriptions given. Patches of *tinea circinata* may look like psoriasis, but their small number, want of symmetry, small scales, and the results of microscopic examination will show their nature. *Lupus erythematosus* is recognised by its position on the face, the greater thickening of the skin, the sebaceous plugs and the scars, and *scaly syphilides* by the small size of the lesion, the slight scaliness, the browner colour, and the concomitant symptoms.

**Treatment.**—Although in the majority of cases the eruption can be temporarily cleared by suitable external treatment, this should never be relied upon alone, and a careful systematic investigation should be made in each individual case in order to discover some defect in the general health. In plethoric persons in whom the eruption is apt to be extensive and irritable, and in whom the urine is hyperacid, great benefit often results from the internal administration for a while of alkalies combined with salicin (*e.g.* salicin, gr. xv; sod. bicarb., ℥ss—℥j; potass. cit., ℥ss; aq., ad 1 ounce t.d.s. a.c.). The same mixture is also indicated in those cases in which the eruption breaks out acutely after an attack of tonsillitis. In patients with excessive intestinal putrefaction and marked indicanuria, constipation should be overcome by morning saline aperients, liquid paraffin, and regular physical exercises, and creosote in increasing doses of from 5 to 20 minims may be tried. Arsenic is almost specific in some cases, but is absolutely without effect in others. It must be given in increasing doses, beginning with 3 or 5 minims of the liquor and stopping at 10 or 15 minims according to tolerance; the dose should be raised gradually, and the drug should always be given well diluted immediately after meals. The risk of chronic arsenical poisoning should not be forgotten. If anæmia is present, iron should be combined with the arsenic. Iodide of potassium in very large doses is advocated by some, but its action is very uncertain. It should be remembered that an underlying syphilitic infection may apparently provoke an outbreak of psoriasis in those subject to it, and in these cases active antisyphilitic treatment may bring about a rapid disappearance of the psoriasis. When the disease is associated with rheumatoid arthritis or with other rheumatic conditions, such as sciatica or fibrositis, a diligent search should be made for foci of infection, and when found these should be removed, and a vaccine prepared from the infecting organisms should be given over a long period. The method of treatment introduced by Danysz, which consists in injecting bacterial emulsions (killed at 70° C.), prepared from fæcal organisms, in increasing doses at intervals of two or three days, is sometimes strikingly successful, but, on the other hand, often fails absolutely, and it does not prevent recurrences. It is, however, worthy of trial in obstinate

cases. In patients with symptoms of hypothyroidism, and in women in whom the disease appears about the time of the menopause, thyroid gland preparations should be given persistently, but cautiously, over a long period. As regards diet, no fixed rules can be laid down. Some patients can keep free from the eruption if they remain vegetarians; in others restriction of carbohydrate is beneficial. Alcohol in excess almost invariably aggravates the disease, and is likely to cause itching.

The external treatment, like the internal, necessarily differs in different cases. If the eruption be extensive, the patient should be advised to submit himself entirely to treatment until he is absolutely free. Chrysarobin is by far the most effective application, but it stains linen and is very irritating, particularly to the skin of the flexures. It is best applied as an ointment (ung. chrysarobin comp. B.P.C.). It should never be applied to the face or scalp. Next to chrysarobin, pyrogallic acid and the various tars (ung. ol. cadini, ung. picis liquidæ, ung. picis carbonis) are the most useful agents. For the face and scalp an ointment containing ammoniated mercury, salicylic acid, and resorcin is usually effective. Whatever preparations are used, the scales must be first removed by means of a daily alkaline bath, friction with a tar soap, and inunction with an ointment containing salicylic acid.

## THE ERYTHRODERMIAS

By the term *erythrodermia* is understood a more or less *generalised* inflammation of the skin, characterised by *redness*, *scaliness* and a tendency to *persistence*. The term is an artificial one, for under it are grouped a number of conditions which, although presenting the above clinical characteristics, are ætiologically quite distinct. It should be observed that (1) the *redness* is essentially of inflammatory origin, and is usually accompanied either by thickening, or by thinning and retraction of the skin; (2) the *scaliness* is an important feature, and the scales may be small and powdery, or so large that literally sheets of them are exfoliated daily; (3) the eruption tends to involve large areas, and often affects the whole integument; the nails and hair may be shed; (4) the majority of erythrodermias are *chronic*, and the severe forms persist till death.

The classification adopted by Darier is the most convenient: thus we have: (1) *Primary erythrodermias*, which develop on healthy skin; (2) *Erythrodermic dermatoses*, in which, owing to the generalisation of one of the erythemato-squamous eruptions described above, erythrodermia results; (3) *Secondary erythrodermias*, in which erythrodermia is a complication, often fatal, of some preceding eruption; (4) *Congenital erythrodermia*, occurring in the newly-born.

The primary erythrodermias alone require description here, and are classified as follows:

A. *Acute Primary Erythrodermia.*—This form includes one clinical entity, namely, *erythema scarlatiniforme recurrens*, and is also a rare form of toxic eruption caused in sensitised persons by the absorption of certain drugs, particularly mercury, quinine, chloral, belladonna, opium and picric acid.

B. *Subacute Primary Erythrodermia.*—This is merely a more chronic and serious form of the preceding. It is seen particularly in alcoholics and those with chronic renal disease, may be accompanied by progressive wasting, diarrhœa and fever, and is fatal in a considerable proportion of cases. Salvarsan dermatitis (see p. 123) may be included under this heading.

C. *Chronic Primary Erythrodermia.*—(1) *Pityriasis rubra;* (2) *Premycosic erythrodermia;* (3) *Leukæmic erythrodermia.*

### PITYRIASIS RUBRA

It is clear from what has been said that the clinical syndrome of generalised exfoliative dermatitis may result from various causes, and at one time the term

*pityriasis rubra* was employed indiscriminately in all cases of chronic generalised erythrodermia. It is best, however, to confine it to a group of cases in which the erythrodermia is *primary*, *i.e.*, does not supervene on any previous disease of the skin, and *persistent*, and which invariably end fatally. The cause is entirely unknown, and *post-mortem* examinations have failed to throw any light on the ætiology. Such cases were described by Hebra, and Jadassohn, who collected eighteen, pointed out that a considerable proportion die of tuberculosis. Typical tubercles with giant-cells and tubercle-bacilli were found in sections of the skin of one case. It is probable, however, that the tuberculous infection, if it be present, is co-existent with, and not a cause of, the erythrodermia.

**Symptoms.**—The eruption begins as erythematous areas, with slight scaliness but without infiltration, as a rule, in the joint flexures. It extends and in time involves the skin of the entire body. The scales are small on the face, but larger on the trunk and limbs, detached at the margins, and frequently and abundantly shed, so that the bed is filled with dry, papery flakes, amounting to a pint or two in twenty-four hours. There is no exudation of serum, and the skin feels dry and hot, but in some cases there occur periodically profuse sweats, which leave the patient temporarily exhausted. For a long time the subjective symptoms are usually slight; the patients complain of feeling cold, and shivering fits are common, but the appetite is good, at times excessive, and nutrition is well maintained. Itching may be slight or even absent, but is sometimes intense and paroxysmal, causing insomnia. The temperature is apt to be subnormal, but pyrexial attacks are common towards the end. In time definite infiltration of the skin occurs, so that free movement is difficult, the hair of the scalp becomes thinned, the lanugo hair disappears, and the nails are affected. After the eruption has persisted for several months, secondary sclerosis and atrophy of the skin occur. The epidermis is thinned, and the whole skin feels hard and inelastic, fitting tightly over bony surfaces and joints. Its colour tends to become darker and more livid, and increased pigmentation, particularly in the natural folds, is noticeable. In rare cases the skin takes on a characteristic bluish-brown hue, which is seen in no other condition. Gradually the patient's strength begins to fail, he wastes, his sweats increase, his mental state becomes affected, so that he is childish and is a prey to delusions, albuminuria and diarrhœa may be present, and the secondary infective complications—cellulitis, deep abscesses, bronchitis, hypostatic pneumonia, etc.—provoke a pyrexial response of varying degree. Hæmorrhages into the skin and mucous membranes are often seen terminally, and their occurrence in the suprarenal capsules may be the apparent cause of death.

**Pathology.**—It is remarkable what little evidence there is of organic disease *post-mortem* in many cases of this obscure condition. Jadassohn's experience that death usually occurs from tuberculosis has been mentioned, but no signs of active tuberculosis have been found in some characteristic cases. On section the skin in the early stages shows the appearances of a superficial inflammation with parakeratosis. In later stages there is atrophic change, the papillæ and interpapillary processes may be obliterated, the pilo-sebaceous follicles and sweat glands have disappeared, and there is cicatricial contraction of the newly-formed connective tissue, with deposit of pigment in the corium.

### PREMYCOSIC ERYTHRODERMIA

The various prodromal eruptions that may precede the appearance of the characteristic tumours of mycosis fungoides are mentioned in the description of that disease (see p. 1019). One of these is an erythrodermia, beginning as intensely itching, red patches, and becoming generalised. The skin is of a vivid red colour ("*homme rouge*"), more livid in the flexures and lower limbs; there is much less desquamation, and the scales are smaller than in pityriasis rubra, and here and

there may be small islets of healthy skin, which appear depressed below the surface of the surrounding inflamed and infiltrated integument. The nails are not usually affected, except that they are worn and shiny from constant scratching. The itching is intense, much more so, as a rule, than in pityriasis rubra. The subcutaneous lymphatic glands are enlarged and easily palpable. Eventually, often after many years, the nodular tumours of mycosis fungoides arise, but in some cases the erythrodermia may spontaneously disappear and later recur, or death may result from cachexia or an intercurrent infection before the appearance of tumours.

*The histological appearances* of premycosic erythrodermia are characteristic. There is a dense infiltration, composed of lymphoid cells, lying in the papillary zone and sharply limited below; the papillæ are broadened and elongated; the interpapillary processes are thinned and often bifid; the horny layer is thickened and in places parakeratotic; and here and there lying in the malpighian layer are seen little nests of lymphocytes, which are pathognomonic of mycosis fungoides.

## LEUKÆMIC ERYTHRODERMIA

Leukæmia, like Hodgkin's disease, may be accompanied by various cutaneous manifestations. In acute leukæmia, which is usually of the myeloid rather than the lymphoid type, the lesions may be divided into (1) leukæmic tumours; (2) dermatoses of various kinds. Of the latter purpura is by far the commonest, but erythematous rashes, papules, vesicles, hæmorrhagic bullæ, and erythrodermia may occur. In chronic myeloid leukæmia tumours in the skin are very rare; cutaneous and subcutaneous hæmorrhages are seen, usually towards the end, and pruritus has been described. In chronic lymphoid leukæmia tumours are not uncommon, and may be present before the occurrence of leucocytosis, although a differential count usually shows a relative lymphocytosis. Apart from tumours, there are other cutaneous manifestations—the *leucemides* of Audry and Germès; they are polymorphic, and usually accompanied by pruritus. They take the form of *urticaria*, which may be of the papular or vesicular type, *erythemata*, simple or bullous, imitating erythema multiforme or even dermatitis herpetiformis, and *prurigo*, recalling that seen in Hodgkin's disease. It is probable that these various types of eruption, and the erythrodermia to be described, are due to toxins liberated from disintegrated lymphocytes.

*Leukæmic erythrodermia* is not very rare, and different forms of it have been distinguished. Thus some are relatively acute and febrile, corresponding to the group of subacute primary erythrodermias; others are essentially chronic, and simulate pityriasis rubra; others again are partial, and resemble psoriasis. The commonest variety clinically resembles premycosic erythrodermia very closely, but the itching is usually less intense. Sequeira and Panton have described a series of cases under the term *lymphoblastic erythrodermia*, characterised by a moderate leucocytosis, 8,000 to 30,000 per cubic millimetre, with an increase of the small lymphocytes up to about 75 per cent. There seems, however, no reason to separate these cases from leukæmic erythrodermia. In the differential diagnosis between premycosic and leukæmic erythrodermia the following points should be noted: (1) The blood picture; (2) the histology, that of premycosic erythrodermia being characteristic; (3) The X-rays have little or no effect on leukæmic erythrodermia, but their action on mycosis fungoides is striking, though temporary.

## PITYRIASIS RUBRA PILARIS

This rare disease should, perhaps, be considered with psoriasis, which it closely resembles. The primary lesion is an acuminate, red follicular papule, with a broken hair in its centre, and surrounded by a horny collar, which dips into the follicle. These papules are seen best on the hairy parts of the limbs, especially

on the extensor surface of the forearm and on the dorsal parts of the fingers. Apart from the primary follicular lesions, red, scaly patches, resembling psoriasis, appear on various parts of the body, and they may extend until a generalised erythrodermia results. The palms, soles, and face are particularly affected. The evolution of the disease varies. The onset is usually sudden, and the eruption spreads rapidly until the whole skin is involved. The erythrodermia may remain generalised for several months, and then disappear slowly or rapidly until the skin returns completely to normal; or the greater part of the eruption may clear, but certain areas, usually the palms, soles, and face remain affected sometimes for years. Two or more attacks may occur in the same patient. The general health is but little affected, but there may be slight fever at the onset, and during the erythrodermic stage of the disease. The inflammation and thickening of the skin give rise to a sensation of heat and burning, and there is great discomfort from cracks on the palms and soles, and from the ectropion, which may result from involvement of the face.

**Ætiology.**—The course and evolution of the eruption suggest that it is caused by a specific infective organism, the nature of which is entirely unknown.

**Treatment.**—The local treatment is like that of psoriasis, except that chrysarobin should not be employed. Oil of cade is, perhaps, the most effective application. Internally, the administration of arsenic in increasing doses, and of thyroid seem to be of benefit, but the disease often seems to run a definite course without apparently being influenced by any form of treatment.

## PAPULAR ERUPTIONS

The term lichen has been long in use to signify any sort of papular eruption, but is now generally restricted to two diseases: one falls in the present group, *lichen ruber planus*, or *lichen planus*; the other is a tuberculide, *lichen scrofulosorum* (*see* p. 953).

### LICHEN PLANUS

*L. planus* consists of raised flat patches of a dull red or almost violet colour, and a surface smooth, shining, or covered with quite small scales. The patches arise from the aggregation of papules, which are at first discrete, and then become continuous by the growth of fresh papules in the intervening spaces. The papules are flat, shining, square or polygonal, and sometimes have a minute depression in the centre. Wickham pointed out that white opalescent points or striæ forming a fine network are visible under a lens, especially if the surface has been moistened with oil or water. The eruption is more or less symmetrical, and appears first on the wrists and forearms, and on the inner sides of the knees, and then on the extensor surface of the arms or legs, the ankle, foot, the flank, hip, and lower part of the abdomen. It also tends to appear on parts subject to pressure—for instance, the waist, the legs where the garter presses, and the palms and soles; but in these last two situations there is only a general thickening of the epidermis, with white spots where the horny layer is cracking. Sometimes the papules have a linear distribution along the course of the nerves. The rash on the skin is often associated with white spots on the tongue and inner side of the cheeks. The head and face are very rarely affected. The itching may be slight, moderate, or intense. When the lesions subside they leave a very persistent stain. On the lower extremities, and in connection with varicose veins, the papules may grow to a great size (*L. hypertrophicus*).

**Ætiology.**—In many cases no cause can be discovered; in others worry, anxiety, insufficiency of food or defective digestion have preceded the disease. It is most common between the ages of twenty and fifty, and rarely attacks children.

**Pathology.**—The inflammatory process in *L. planus* begins round a sweat duct in the upper part of the corium; there is a dense growth of connective tissue cells, which are, according to some, of granulomatous type. This is followed by increase of the cells of the stratum mucosum, and thickening of the horny layer. There is irregular thickening of the eleidine, which causes the appearance of white striæ, above mentioned.

**Treatment.**—The treatment is not unlike that of psoriasis. Internally arsenic should be given steadily in full or increasing doses for a considerable time; it is most successful in chronic cases and less certain in acute; perchloride of mercury and salicin are also recommended. The local treatment consists in the use of tarry preparations, such as ung. picis liq., ung. creosoti, liq. picis carbonis, thymol, and carbolic acid. If there is much hyperæmia, more soothing applications, such as lead or zinc lotions, may be desirable for a time. The general health also requires attention, by bodily and mental rest, nutritious food, the usual tonics, and, perhaps, change of air. Resistant patches may be treated with X-rays.

## PRURIGO

This is a papular disease accompanied with severe itching (*prurio*, I itch). The papules are at first not so much visible as palpable, having the colour of the skin, and only later becoming pink and red. They are not collected in groups, but scattered. As they are accompanied by severe itching, they are soon scratched, the heads of the papules are removed, and small blood scabs are the result. More violent scratching leads to enlargement of the papillæ; the skin in the regions affected becomes darker in colour, thickened and rough, the natural furrows are deepened, the surface is covered with mealy scales, the downy hair is destroyed, and when the hand is passed over the skin it feels like a nail brush or like rough brown paper. This change, called *lichenification*, is not peculiar to prurigo, but occurs in most complaints attended with itching, such as eczema, psoriasis, and lichen, and may arise locally in healthy skins from the pressure of clothes, or from constant friction. Besides the excoriation of the papillæ of prurigo, more extensive scratch marks, abrasions, and scars may be caused, and other secondary lesions, such as eczematous patches, urticaria, pustules, enlargement of the femoral, axillary, or elbow glands, and, finally, more or less deep pigmentation of the skin.

The parts first affected are the extensor surfaces of the legs and arms, especially the former. The chest, back and front, the abdomen, and gluteal regions are all affected; even a few papules may appear on the face; but the flexures of the elbow and knee, the axillæ, the genitals, the ankles, wrists, palms, and soles are always spared.

**Ætiology.**—It is more common in males than in females, and among the poorer classes. Different statements are made as to the influence of cold, but it is certain that winter cold does cause in some people a pruriginous condition, especially of the legs (*P. hyemalis*). Prurigo commonly begins in infancy, and has sometimes been preceded by an urticaria (*urticaria papulosa* or *lichen urticatus*); it continues, unless vigorously treated, for the rest of life.

**Varieties.**—*P. mitis*, *P. gravis*, *P. ferox*, have been described; they appear to differ only in intensity. Hebra regarded as a special and incurable form the very intense cases which he saw in Vienna, but there seem to be cases intermediate between those and the milder forms commonly seen in England.

**Pathology.**—The early anatomical change is an exudation into the papilla of leucocytes or serum, the former collecting about the vessels; there is also infiltration and imperfect vesicular formation in the epidermis. It still remains doubtful whether the papules precede the itching, or *vice versâ*.

**Diagnosis.**—It must be distinguished from *pruritus*, which means the sensation of itching, from whatever cause arising, and which, if intense, is accom-

panied by all the lesions, scratch marks, blood crusts, scars and pigmentation that occur in prurigo. This is seen notably as a result of pediculi, and in a doubtful case these insects should be looked for (*see* Phtheiriasis). Prurigo is, however, distinguished by the age of the patient, the long history of the disease, the absence of any cause, and the distribution of the lesions.

**Treatment.**—This must consist of the frequent use of warm water, the Turkish bath, or alkaline baths, and the thorough munction of soap and emollient ointment. Tar and sulphur preparations are also of value. Whatever method is employed must be continued daily and perseveringly. The following may be used: spermaceti ointment alone or with the addition of cod-liver oil, or tar ointment or $\beta$-naphthol; equal parts of soft soap and spirit or a fluid glycerine soap; sulphur ointment or Vlemingkx's solution (containing sulphides of calcium); carbolic acid (2 to 5 grains to aq. 1 ounce); menthol (5 to 10 grains in dilute alcohol 1 ounce, or as a soap); solution of coal-tar (1 drachm to 8 ounces); or tar baths (brushing the surface all over with tar, and then remaining in a hot bath for three or four hours); or baths medicated with sulphurated potash, creolin or izal. Internally help may be obtained from arsenic, carbolic acid, cannabis indica, and antipyrin. Nutritious food, cod-liver oil, wine, etc., should also be given.

## PRURITUS

The subjective sensation of the skin, itching or pruritus, is experienced in a great number of diseases of the skin as well as some other conditions of ill-health, among which may be mentioned jaundice, diabetes, and Hodgkin's disease. The cutaneous disorders are (1) the various forms of erythema and dermatitis, especially prurigo, urticaria, eczema, psoriasis and lichen; (2) the presence of the animal parasites, *Acarus scabiei*, and pediculi; (3) to a less extent the vegetable parasites, or tineæ; (4) some local conditions such as *pruritus vulvæ* and *pruritus ani*. The former may be due to the irritation of saccharine urine in diabetes, and the latter to piles or hæmorrhoids; both may be due to threadworms in children. A general pruritus is seen in its most severe form in phtheiriasis, in prurigo and in jaundice; and the violent scratching which it induces may be followed by all the secondary lesions described in the accounts of those disorders.

**Treatment.**—This must aim at removing the cause, for instance, curing the lesion of the skin or destroying the animal or vegetable parasites. Where the removal of the cause can only be slow or is difficult, or where the cause does not lie primarily in the skin, itching may be directly treated. For this alkaline baths ($\frac{1}{4}$ to $\frac{1}{2}$ lb. of sodium bicarbonate in 30 gallons of warm water), tar lotions, or lotions containing liquor carbonis detergens (℥ij to aq. ℥viij) should be tried. Lumbar puncture has given relief in some cases, as in lichen planus and in prurigo. For the pruritus vulvæ of diabetes, pending the diminution of the sugar, a lotion of $\frac{1}{2}$ ounce of fresh yeast in a pint and a half of water may be applied locally and used as a vaginal injection. In pruritus ani a mercurial ointment, or an ointment made by rubbing a quantity of quinine sulphate into vaseline, is very useful. Some have discovered streptococci in swabs from the anal region, and have had good results from the use of autogenous vaccines. Lavage of the colon is recommended.

## PARAKERATOSIS VARIEGATA

This is a condition somewhat resembling a lichen, consisting of small flat papules, each covered with a fine adherent scale, which can be scratched off without bleeding. When the scale is removed the papules are yellowish red on the body or bluish red on the extremities. The papules run together in such a way as to include healthy areas of skin in a sort of meshwork, and thus give a

retiform or marbled appearance to the skin. The whole of the surface may be affected, except the face, scalp, palms, and soles. It occurs especially in otherwise healthy adult males. It is chronic in its course, but subject to remissions and exacerbations, unaccompanied by subjective symptoms, and very resistant to treatment. Histologically it is an inflammation of the subepidermal layer of the cutis, with dilated vessels, œdema, and cell infiltration, as well as some œdema and thickening of the epidermis.

### GRANULOMA ANNULARE

This somewhat rare condition consists of a ring or rings of small raised nodules, enclosing an area of healthy or slightly reddened skin, and occurring most often on the backs of the fingers, hands or wrists, less often on the feet, ankle, neck, elbows and buttocks. The ring varies in diameter from $\frac{1}{2}$ inch to 1 inch or 2 inches, and the nodular part is about $\frac{1}{16}$ inch in breadth. In colour the nodules are at first white and waxy, later pink or even bluish. Histologically the epidermis is either normal or slightly thickened, and in the cutis occur groups of cells, chiefly mononuclear, with connective tissue corpuscles and epithelioid cells, which surround the sweat glands and vessels or are scattered in the substance of the cutis. Here and there may be areas of necrosis, but no giant cells or tubercle bacilli. There are few or no subjective symptoms, and the lesions, though persisting for a long time, spontaneously recover or yield to treatment. They occur especially in children and young persons under twenty-one (60 per cent. of cases collected by E. G. Little).

**Treatment.**—Salicylic acid in ointment or plaster, or ichthyol or resorcin ointments should be applied, and, if these fail, one or more doses of the X-rays.

## VESICULAR ERUPTIONS

The name herpes has been given to certain vesicular diseases, but it is not easy to give a definition that will cover all. One may say that the vesicles of herpes are generally smaller than those of pemphigus, are seated upon an inflamed base, and terminate by scabbing. The diseases for which the name is still retained are *Herpes zoster* or *zona*, *Herpes facialis*, and *Herpes genitalis*.

### HERPES ZOSTER
(*Zona, Shingles*)

This is an eruption of vesicles, arranged in groups, which generally correspond in position to the distribution of a cutaneous nerve. The name zona, or girdle, is taken from the most common or intercostal variety, in which the groups of vesicles extend from the spine round one-half of the body to the middle line in front. The eruption is preceded sometimes by pain, tingling, or smarting, and it may be a little malaise or slight pyrexia; then appear groups of closely set papules, forming red patches, 1 or 2 inches in diameter; and upon these the vesicles quickly arise, with thin walls, clear contents, not very tense, and, when numerous, acquiring a polygonal form from mutual compression. The patches do not all appear simultaneously—for instance, one may form first near the spine, then later one in the axilla, and later again one near the sternum; some patches also—that is, the later ones—may fail to produce any vesicles, the process, as it were, subsiding early or aborting. After a time the contents of the vesicles become opaque or milky, and the vesicle dries into a scab which drops off, leaving a red stain. The milkiness may amount to the formation of pus, and the superficial layer of the skin may be destroyed, so that scars result. Scars may form in each group, but not from every vesicle of a group. Quite rarely extensive sloughing of the skin takes place, leaving deep ulcers, which heal slowly. Although the patches are obviously related to nerves, on the trunk

they form a band from 2 to 4 inches broad, the direction of which is more horizontal than the true course of the ribs; and the vesicles may transgress the middle line both in front and behind. Occasionally vesicles occur, even abundantly, in parts of the body remote from the nerves primarily concerned (*aberrant vesicles*, or *generalised herpes*).

*H. frontalis* occupies the area of the supraorbital nerve on the forehead, scalp, upper eyelid, and side of the nose. Herpes of the ear and neighbouring parts may arise from implication of the sensory branches of the fifth nerve, of the facial (*nervus intermedius*) and of the ninth and tenth nerves and their corresponding ganglia. *H. cervicalis* lies over the neck, clavicle, and deltoid; *H. brachialis* follows the course of the nerves of the arm; and other similar groupings on the abdomen, thigh, and leg are occasionally seen. An intercostal zona may be accompanied by herpes of the inner side of the arm (intercosto-humeral nerve), or a gluteal by an anterior crural, representing posterior and anterior branches of the lumbar nerves. The eruption is nearly always unilateral, and its bilateral occurrence has very rarely been recorded. An important feature of the *Herpes zoster* is that the lymphatic glands corresponding to the affected area are enlarged and tender even before the eruption appears.

The duration of the eruption is from four to ten days, but the disease does not always end here. Especially in old people, neuralgic pain in the course of the affected nerve may continue for months or years, and be a source of serious trouble; and in a few cases motor nerve fibres are involved as well as the sensory, and paresis of the corresponding muscles has been observed, most often in those supplied by the facial, sometimes in those supplied by the third, motor fifth or sixth cranial nerves, and by the nerves to the deltoid and abdominal muscles. Frontal herpes may be accompanied or followed by conjunctivitis, ulceration of the cornea, or iritis.

**Pathology.** The eruption itself is an inflammation of the papillæ and corium, followed by effusion into the layers of the *Stratum mucosum;* and the close relation to cutaneous nerves is confirmed by the lesions which have been found in them. The most frequent is inflammation of the ganglion on the posterior root of the spinal nerve, and of the nerve below it; others are inflammation of the sensory root above the ganglion, peripheral neuritis, neuroma, and hæmorrhage into the Gasserian ganglion. While sometimes due to local causes (disease of the ribs), it more often presents all the characters of an acute infectious disease, and this is supported by the occurrence of aberrant vesicles. Some have regarded it as a *posterior poliomyelitis*, corresponding to the more familiar anterior poliomyelitis (*see* p. 97). *Herpes zoster* also occurred in some of the cases of arsenical poisoning among beer-drinkers in Manchester in the year 1900, and may be caused by arsenic taken by the mouth for medicinal purposes, or by the injections of arsenical preparations such as salvarsan. It also occurs as a result of syphilitic meningitis around the posterior roots, and in tabes recurrent attacks of zoster, usually accompanied by severe lightning pains in the affected area, are sometimes observed. Recent observations and experimental research, however, indicate that in the majority of cases *Herpes zoster* is due to infection of the posterior root ganglia with a *streptococcus longus*, which obtains entry into the blood-stream usually through the tonsils or teeth.

The possibility of a relation between *Herpes zoster* and varicella has been already referred to (*see* p. 44). The facts alleged are that in the same individual (1) an attack of herpes has been followed by an eruption of varicella, (2) that the eruption of varicella has been followed by *Herpes zoster*, (3) that the two eruptions have occurred at the same time, (4) that an eruption of shingles in one person has been followed after an interval of some days by varicella in children or others who have been in his company.

Until a sufficient number of cases have been carefully observed and recorded or until the organisms are known, it is idle to speculate on the identity of the

virus or of the organisms in the two complaints. But it should be remembered that the vesicles of *Herpes zoster* are not always confined to the course of the nerve or nerves presumably involved, and that the vesicles of varicella may be few in number, and possibly grouped: in other words, the diagnosis must be very carefully established before conclusions are drawn.

**Diagnosis.**—This depends on the unilateral group of vesicles, corresponding to the distribution of a nerve; but frontal herpes, in the early stage of redness and infiltration, may closely resemble erysipelas.

The **Prognosis** is favourable, but the probability of scarring, and the tendency to troublesome neuralgia in elderly patients, must be remembered.

**Treatment.**—Nothing will check the disease; but we should try to protect the vesicles from injury and from rubbing by the clothes, and to allay any irritation, tingling, etc. This may be done by applying powdered zinc oxide with starch powder, to which a little pulvis opii may be added if the pain is severe, and covering it with cotton-wool. When the vesicles burst, or the skin ulcerates, zinc ointment or boric ointment may be applied. For the severe pains afterwards, arsenic, antipyrin (in 10-grain doses), phenacetin (10 gr.), quinine, or sodium salicylate should be given. The last may be introduced by ionisation. But morphia, either internally or subcutaneously, is often required. Menthol may be rubbed in; and blisters over the origin of the nerve and the continuous galvanic current also give good results.

### HERPES FACIALIS

*(Herpes labialis)*

This occurs as groups of vesicles forming rapidly upon an inflamed base. The contents are clear at first, then turbid, and afterwards dry into a scab, which falls off, leaving scarcely a mark. It affects the lips, the alæ of the nose, and the adjacent cheeks, is usually bilateral, and lasts from five to ten days. It mostly occurs in association with some acute febrile disease, especially with croupous pneumonia, of which it is sometimes considered diagnostic; but this is not so, as it happens in ordinary catarrh and bronchitis. It occurs in diphtheria and in relapsing fever, and one sometimes sees it without any other recognisable disease other than a sharp pyrexial attack, with high temperature and rigor. It not infrequently recurs in the same person, particularly in those who are subject to repeated attacks of naso-pharyngeal catarrh. It may also in some persons apparently be produced by eating certain articles of food, such as cheese.

**Treatment.** A threatened attack may sometimes be aborted by sponging with a 1 per cent. solution of menthol in alcohol. When the vesicles have appeared a calamine-zinc lotion, or a mild antiseptic powder, should be applied. Recurrent attacks may sometimes be prevented by giving periodically a prophylactic anticatarrhal vaccine, or by the removal of septic tonsils, or the correction of some abnormal condition of the nasal passages. Chronic constipation would seem to be a predisposing cause in certain cases, and relief of this has been known to lead to a cessation of the attacks.

### HERPES GENITALIS

*(Herpes preputialis)*

This closely resembles the preceding. A vesicle or a group of vesicles on an erythematous base forms on the inner side of the prepuce, less often on the outside, on the glans, in the meatus or even in the urethra. But it is seen sometimes on the labia, nymphæ, and pubes in women. It is often preceded by some local disease, such as gonorrhœa, soft chancre, or stricture of the urethra, and it is of importance, because the vesicles rupture early, and form small ulcers, which may be mistaken for chancres. Like *Herpes facialis*, it is apt to recur. The

treatment is to keep the parts thoroughly clean, and to apply lead lotion on strips of lint, or dust starch and zinc oxide over the vesicles, and separate the parts with lint. Iodoform or lotio nigra on lint may be used where a sore has formed.

### CHEIROPOMPHOLYX

*(Pompholyx, Dysidrosis)*

**Definition.**—A bilaterally symmetrical vesicular or bullous affection of the hands and feet, frequently accompanied by hyperidrosis.

**Symptoms.**—The word *cheiropompholyx* (from χείρ, a hand, and πομφόλυξ, a bubble) should be reserved for an eruption which appears spontaneously and often periodically in certain persons. A dermatitis bearing very close, if not identical, clinical appearances to those of pompholyx, may be produced by various irritant applications and by a ringworm fungus (epidermophyton), but this must be differentiated from the disease to be described.

An attack of pompholyx usually begins with a sensation of itching, burning, or tingling in the hands, and within a few hours the eruption appears as small translucent vesicles situated usually on the sides of the fingers, in the interdigital clefts, and in the palms. These vesicles are deep-seated, unaccompanied by any signs of inflammation, and have been compared to boiled sago grains. In mild cases they may remain discrete, but frequently they coalesce, forming large bullæ projecting beyond the skin. The fluid in the vesicles is clear, alkaline, and contains albumin. The lesions may dry up spontaneously, the overlying skin being shed in dry flakes; in this way the skin of the whole palm may peel off, leaving a pink new skin underneath, which gradually assumes the normal appearance. On the fingers, however, the larger vesicles and bullæ are apt to be ruptured, exposing a raw, moist, tender surface beneath. Secondary infection of the lesions is liable to occur, particularly in uncleanly persons, or in those whose resistance is low; the vesicles then become purulent, and a spreading lymphangitis of the arms with adenitis and fever may result. A peculiarly offensive odour, resembling that of atrophic rhinitis, occurs in these infected cases: this probably results from the digestion of keratin by staphylococci which, growing in the alkaline fluid of the vesicles, liberate proteolytic enzymes.

The eruption may be confined to the hands, but not uncommonly the toes and the soles of the feet are affected, and here secondary infection is the rule. In some cases the disease is accompanied by eczema of other parts of the body.

The duration of an attack varies from a few days to several weeks, but recurrences are common, particularly in spring and summer. Patients subject to the disease are usually in a poor state of health, and are frequently neurotic; the symptoms associated with chronic intestinal toxæmia of the putrefactive type are often evident, namely, pigmentation of the skin, clammy sweat, foul breath, anorexia, and headaches. There is sometimes hypochlorhydria, and the urine contains a considerable quantity of indican in the majority of cases.

**Anatomy.**—The histology resembles that of eczema in that the vesicles are formed in the prickle-cell layer. In the corium the papillary vessels are dilated, and there is some inflammatory cell infiltration around them. Thus the vesicles have not, as was once thought, any direct relation to the sweat ducts.

**Ætiology.**—The disease is commonest in early adult life, and is seen, perhaps, more often in women than in men. It is certainly a manifestation of toxæmia, and the frequency with which indicanuria and other symptoms of excessive intestinal putrefaction are present in patients subject to the disease suggests that the toxin may be some decomposition product of protein. Those with sedentary occupations may lose their tendency to the attacks in adopting an out-of-door life with vigorous exercise. The disease occurs most frequently in hot weather, and some persons are liable to one or more attacks every summer.

**Diagnosis.**—True pompholyx must be distinguished from eczematous

dermatitis produced by external irritants, and from eczematoid ringworm of the extremities. Differentiation from these two conditions may be difficult, particularly from the latter. It is essential in doubtful cases that scales and the roofs of the vesicles should be examined microscopically, with their under-surface upward, in liquor potassæ.

**Treatment.**—If, as is usually the case, there is evidence of excessive intestinal putrefaction, the diet should be revised. Patients liable to severe recurrent attacks may well become vegetarians temporarily, and in any case fruit and green vegetables should be taken rather than the heavier meats and sweets. Regular aperients are advisable for a while, such as liquid paraffin, sodium sulphate, and occasionally calomel in small doses. If hypochlorhydria is present, dilute hydrochloric acid should be given in 30 to 40 minim doses, well diluted, after meals, as this will materially aid the digestion of protein. Sometimes the so-called intestinal antiseptics, *e.g.* dimol, kerol, seem to do good. Locally in the early stages sponging with a 2 per cent. solution of salicylic acid in spirit stops the irritation at once, and then either the pulv. acidi salicylici comp. B.P.C. or pasta zinci comp. B.P.C. may be applied. In cases in which the skin has become raw a lotion containing boric acid, calamine, and glycerine, should be used, and those in which secondary infection has taken place should be treated with weak antiseptic lotions locally, rest in bed, low diet, and tonics.

## BULLOUS ERUPTIONS

### PEMPHIGUS

The term pemphigus (πεμφιξ, a blister) has in the past been somewhat loosely applied to a number of bullous eruptions which ætiologically have nothing in common. Thus *P. neonatorum* and *P. contagiosus tropicus* are merely varieties of *Impetigo contagiosa*, and are therefore due to superficial infection of the skin with a *Streptococcus pyogenes longus; P. syphiliticus* is a bullous syphilide most commonly seen on the palms and soles of infants with congenital syphilis; and *P. leprosus* is a bullous form of leprosy.

At the present time the word pemphigus is confined to the following conditions: (1) *P. acutus malignus*. This is a rare acute infective disease, usually fatal, which occurs in those who handle dead meat, and is probably due to infection with a diplococcus. It should no longer be included in the pemphigus group. (2) *P. vulgaris*. (3) *P. vegetans*. (4) *P. foliaceus*. These are probably merely varieties of the same disease—*true pemphigus*.

Closely allied in all probability to true pemphigus is *Dermatitis herpetiformis*, of which *Hydroa gestationis* is doubtless a variety occurring in pregnant women.

### PEMPHIGUS ACUTUS MALIGNUS

This disease, which is excessively rare, has been chiefly observed among butchers and farm-hands, and in most cases its onset has been preceded by local wounds on the hands or arms, through which presumably the infecting organism obtained entry. As in true pemphigus, the lesions are large bullæ, arising usually directly from the skin without any preceding erythema; they contain at first clear or slightly blood-stained serum, but later this becomes puriform from secondary infection. New bullæ arise on all parts of the body, and the mucous membranes are usually involved; the skin later becomes excoriated and covered with crusts and fœtid discharge. There is high fever, albuminuria, vomiting, and diarrhœa, and in from two to three weeks the patient usually passes into coma and dies. A few cases have, however, recovered.

The diplococcus, isolated from the bullæ by Demme, Bulloch, and others, is probably the causal organism.

**Treatment.**—The skin should be dressed with mild antiseptic applications, and, if possible, boric acid baths should be given. Quinine is the only drug that seems to have any beneficial effect, and should be given in increasing doses.

### PEMPHIGUS VULGARIS

**Ætiology.**—Although true pemphigus may occur from childhood to old age, it is, perhaps, most frequently seen between the ages of forty and fifty. The two sexes are about equally affected. At present the cause of the disease is absolutely unknown.

**Pathology.**—The bullæ are formed by the effusion of fluid, which collects sometimes beneath the horny layer, and sometimes beneath the whole epidermis. The fluid at first contains considerable numbers of eosinophil corpuscles, and later becomes turbid owing to the leucocytosis which results from secondary staphylococcal contamination. In the papillary layer of the skin there is inflammation with infiltration of leucocytes, many of which are eosinophil, and eosinophilia in the blood is often very pronounced. *Post-mortem* examinations have failed to reveal any constant morbid changes in the viscera.

**Symptoms.**—The disease usually runs a somewhat chronic course. The eruption may be preceded in some people by chilliness, nausea, or pyrexia; then the bullæ appear at one or other part of the body, small at first, gradually increasing in size, tense, hemispherical, with clear yellow or slightly turbid contents. The fluid is an albuminous serum, and the turbidity is due to the presence of leucocytes. Around the bleb the skin is at first quite normal, but a narrow pink areola is acquired later, and increases in proportion to the opacity of the fluid. After a few days the fluid is absorbed, or the bleb ruptures, and shrinks down on to its base. From this it is subsequently shed; it leaves a mark which is injected and afterwards slightly stained but rarely or never scarred. Sometimes the bleb contains pus or blood, and after its rupture the base may be covered with yellow lymph, or may slough.

The number of bullæ in any case is very variable. There may be but few in one part, or isolated bullæ in different parts of the body; or the whole surface may be thickly covered by blebs, which come out in successive crops, lasting only a few days each, but keeping up the disease for weeks and months. Nearly every part of the body may be affected, but the hairy scalp least of all. The disease may involve the mucous membrane of the mouth, nose, pharynx, larynx, and vulva, producing raw tender areas, sometimes covered by a diphtheroid membrane; the conjunctiva, too, may be attacked, resulting in contraction ("essential shrinking"), which may lead to obliteration of the conjunctival sac and ultimate blindness.

The amount of constitutional disturbance varies according to the extent of the eruption. When this is limited the general health is usually maintained, but in generalised cases there is great pain, prostration, moderate pyrexia, and sometimes vomiting and diarrhœa. The patient presents a pitiable appearance, and, when the buccal mucous membrane is involved even liquid food causes great pain; insomnia is a constant symptom, and the unfortunate sufferer, utterly exhausted, eventually lapses into the tremulous, maudlin state which heralds the approach of death.

**Diagnosis.**—The essential lesion of true pemphigus is the bulla, and the diagnosis is not usually difficult. The bullous variety of *Erythema multiforme* may lead to confusion, but the sites of predilection in this condition, the acute course, and the presence of the typical erythematous patches, should suffice to distinguish it from true pemphigus. *Dermatitis herpetiformis* in its bullous phase may be difficult to diagnose from pemphigus, but eventually the appearance of papules and small vesicles, the herpetiform grouping of the lesions, and the intense itching, will make the diagnosis clear. *Epidermolysis bullosa*

may be distinguished by the localisation of the bullæ over points of pressure and friction and by the history. Confusion with bullous impetigo should not occur.

**Prognosis.**—Probably pemphigus is invariably fatal. The patients may, however, survive for years, and may pass through periods of freedom from the eruption. In thirty cases analysed by Highman a fatal issue resulted in every one.

**Treatment.**—Arsenic and, to a less extent, quinine seem to control the disease in some cases, but eventually lose their effect. Improvement has also been observed after blood transfusion and injections of salvarsan. The local treatment consists in keeping the skin as clean and comfortable as possible by means of boric baths, mild antiseptic creams and powders, etc. Lozenges containing cocaine and carbolic acid relieve the pain when the oral mucosa is involved. In the terminal stages morphia should be given freely.

### PEMPHIGUS FOLIACEUS

This is a very rare and fatal form of pemphigus, in which the whole surface of the body is gradually involved. The blebs are flaccid and flat, never tense and hemispherical. Their contents are turbid, and when these escape an inflamed excoriated surface is left; to this the remains of the bullæ adhere, forming thin flakes, the under-surface of which is moist with an offensive secretion. If the flakes are removed there remains a red, raw, secreting surface, not unlike that of *Eczema rubrum*. When the whole surface is affected it is mostly covered with the adherent epidermis, with raw patches at intervals; then also the occurrence of blebs is not easy to observe, as they form under the existing epidermis and soon rupture. The course is slow, with remissions and relapses, it may be with healing of the skin in parts; but eventually the disease is fatal by exhaustion or intercurrent disease.

**Treatment.**—Drugs have no influence; immersion in a warm bath gives temporary relief.

### PEMPHIGUS VEGETANS

is another rare variety, in which the mouth is first affected; then bullæ of ordinary type form on the skin, ulcerate, and remain unhealed for a long time. The characteristic feature is that in moist situations, like the axillæ, groins, and gluteal folds, fungating papillary growths form on the site of the ruptured blebs, project a quarter to half an inch above the surface, and secrete an offensive muco-purulent fluid. Severe prostration ensues, and the cases end fatally. Some relief may be obtained by local antiseptic applications.

### EPIDERMOLYSIS BULLOSA

This is a very rare disease in which the skin is abnormally susceptible to trauma, so that even trivial mechanical injury results in the formation of bullæ. These bullæ are usually formed in the deepest part of the rete, so that practically the whole epidermis is lifted from the corium by the effused fluid. The disease is frequently familial; in a family described by Bonajati thirty-one persons out of sixty-three were affected in five generations. The onset may be in infancy or delayed until childhood, and the affection may become milder or even disappear in later life. The lesions naturally occur chiefly on sites most exposed to trauma, such as the hands and feet and over bony prominences; they may also involve the nails and the mucous membranes. The teeth are often lost early. In adult life the skin may become curiously atrophic. No treatment is of any avail, except that protection from trauma should be ensured as far as possible. The bullæ, when formed, should be dressed with a mild antiseptic paste.

### DERMATITIS HERPETIFORMIS

Dühring, who introduced this term, gives the following definition of the disorder to which he applies it: "An inflammatory, superficially seated, multiform herpetiform eruption, characterised mainly by erythematous, vesicular, pustular, and bullous lesions, occurring usually in varied combinations, accompanied by burning and itching, pursuing usually a chronic course with a tendency to relapse and recur." This includes cases hitherto called *hydroa*, and probably some of the cases recorded as pemphigus. It covers *Herpes gestationis* or *Pemphigus gestationis*, a bullous eruption which occurs during pregnancy, affecting more or less the whole of the body, disappearing with delivery, and recurring generally in future pregnancies. *D. herpetiformis* thus presents the most varied lesions over the body at the same time, in one place patches of erythema, in another urticarious wheals, and in a third bullæ like those of pemphigus; and there may be pyrexia with it. The disease is very little amenable to treatment; the lesions may spontaneously heal at one part, and break out in another. The itching is exceedingly troublesome; but the patient's health and nutrition are generally maintained, and many cases ultimately recover.

The lesion is an acute inflammation of the papillary layer of the corium with the formation of vesicles directly beneath the epidermis and the extravasation of enormous numbers of polymorphonuclear and eosinophil leucocytes. The proportion of eosinophil corpuscles in the blood is also largely increased, up to 10 or 15, or even 30, per cent. of the leucocytes.

**Ætiology.**—The eruption is probably due to "sensitisation of groups of cutaneous nerve-endings to some bacterial protein from the bowel" (**17**). In favour of this view is the fact that a very abnormal intestinal flora has been found in patients suffering from the disease, and that injections of autogenous vaccines prepared from certain organisms cultivated from the fæces, even in small doses, have produced a marked reaction, with exacerbation of the itching and the outbreak of new lesions, and that the long-continued administration of such a vaccine has led to striking improvement, and apparently in a few cases to actual cure.

**Treatment.**—Arsenic, as a rule, is of great value in controlling the eruption, but is in time liable to lose its effect. The diet should be simple and restricted. In severe cases prolonged starvation is justifiable. Some observers have claimed good results from auto-serotherapy. Intravenous or intramuscular injections of peptone certainly do good, but more efficacious are non-specific proteins, such as "Aolan" or "Cibalbumin," and autogenous fæcal vaccines prepared according to the method of Danyz. In some of the cases investigated there has been marked hypochlorhydria or even achlorhydria, and improvement has followed the administration of full doses of hydrochloric acid after meals. Locally, weak sulphur ointments or pastes appear to give most relief.

## HYPERKERATOSES OF THE SKIN

### CALLOSITIES AND CORNS

These are produced by friction and pressure.

A *callosity* consists of a hypertrophy of the horny layer of the epidermis, and is familiar on the ball of the great toe, the heel, the hands of the working man, of oarsmen and others, the tips of the fingers of those who play the violin, etc.

The *corn* (clavus) is a local thickening of the epidermis resulting in a conical downgrowth, which presses upon the subjacent papillæ, causes their atrophy, and sets up inflammation and hypertrophy in the surrounding papillæ. Corns are common, as is well known, on the toes, especially the outer side of the little toe, the dorsum and the sides of the other toes. The pain of the ordinary corn is largely due to the little plug being driven down on the cutis beneath, but spon-

taneous shooting pain is often present. When the corn lies between the toes and is kept constantly moist, the thickening is less marked; but the inflammation is more obvious, and the part is often extremely tender (*soft corn*). Occasionally corns will inflame and ulcerate, or a cyst or bursa forms under the corn, constituting a *bunion*.

**Treatment.**—Corns may be cured, and almost entirely prevented, by the use of properly shaped boots. The sole should be as large as, or slightly larger than, the sole of the foot as it shapes itself in the standing position with the weight of the body upon it. If the boot sole is narrower than the sole of the foot, the upper leather will be in close contact with the edge of the foot in any movement, and constant friction will be the result. The inner edge of the sole should be straight, and pointed boots should be strictly avoided. If corns have formed, they must be treated by soaking in hot water, and shaving with a sharp knife or razor, when the dry white plug will be met with and can be removed. A corn plaster may then be worn, or the toe may be simply strapped with a good linen plaster, by which, with properly constructed boots, the friction will be reduced to a minimum. Soft corns may also be carefully shaved, and pressure removed by cotton wool between the toes, or by a turn or two of narrow strapping below the corn. The thickened epidermis may also be removed by the application of salicylic acid, either as a 2 per cent. ointment, or as plaster, or in solution in collodion (℥j to ʒj). The tender part may be benefited by the use of alum or tannic acid lotions. But in all cases a sufficiently broad-toed boot, with a wide sole and a low heel, is the one requirement for permanent relief.

### KERATOSIS

Keratosis, or increase of the horny layer of the epidermis, is an essential part of many of the forms of dermatitis already described, such as psoriasis, chronic eczema, and pityriasis rubra pilaris. Increase of the horny layer also results from arsenical poisoning. A rare congenital disease, in which enormous hypertrophy of the horny layer of the palms and soles occurs, is known as *keratosis* or *tylosis palmaris et plantaris*. It may occur through several generations and in members of the same family.

### KERATOSIS PILARIS

This consists of small papules, the size of a pin's head, which occur mostly on the extensor surfaces of the limbs, and are formed by accumulations of epidermis at the mouths of the hair follicles. The hair may pierce the centre, or more often it is coiled up in the centre and broken off. The papules are often brown or black from adherent dirt.

The **Treatment** is similar to that of ichthyosis.

### KERATOSIS FOLLICULARIS

This rare condition, often described as *Darier's disease*, is also one in which papules connected with the hair follicles are capped by horny masses; and these horny masses descend as plugs into the entrance of the hair follicle. The lesions occur especially in the groin and hypogastric region, but also on the scalp, face, and other hairy parts. The disease has so far proved incurable.

### CORNU CUTANEUM

Horny growths, sometimes several inches in length, and generally twisted or bent, have in rare cases been seen. They are, as a rule, solitary. They consist of accumulated epidermic layers on a base of hypertrophied papillæ.

The **Treatment** consists of removal and cauterisation of the base.

### ICHTHYOSIS

In this disease the skin is dry and rough from thickening of the epidermis. It is presumed to be congenital, though it is not seen until some weeks or months after birth, and it frequently runs in families. In its mildest form (*xerodermia*, dry skin) the skin is rough, dry, and dirty-looking, especially over the extensor surfaces of the legs and arms. In more pronounced forms (*ichthyosis simplex*) the whole of the body is more or less affected, the limbs most, the scalp, face, palms, soles, genital organs, and flexures of the joints least. The skin is not reddened; but it is covered with thin epidermic scales, whose shape is more or less determined by the folds of the skin, and on the extremities inclines to a polygonal or diamond shape; and along these lines the scale is partly detached, while within them it is adherent. Still a certain amount of shedding is constantly taking place. There is an absence of perspiration, but the sebaceous secretion is mixed with the epidermic scale, and with the adherent dirt contributes to give a grey or greenish dirty appearance to the whole of the skin, or its most affected parts. The health is not injured by it, but the growth of the patient is sometimes stunted; and eczema is a common occurrence. A very considerable proportion of ichthyotic subjects suffer from asthma or hay fever, and the eczema, from which they are so liable to suffer, is often not due to external irritants, but is of the anaphylactic type.

There is a gradation between this and *ichthyosis hystrix* (porcupine skin); but the condition known under this name is often localised or unilateral, and in some cases follows the track of cutaneous nerves or, at any rate, occupies linear tracks, mostly parallel with the length of the limbs, and transverse on the trunk (*I. hystrix linearis*). It consists of thick green or greenish-black plates or masses of hypertrophied epidermis, of square or polygonal shape, rising $\frac{1}{4}$ or $\frac{1}{3}$ inch above the skin, closely fitting together, like a mosaic pattern. Under the epidermic masses the papillæ are hypertrophied. Microscopic examination shows that in all forms the accumulated masses consist of aggregated epidermic scales.

In ichthyosis hystrix the papillæ are hypertrophied, and the horny layers of the epidermis dip down into the interpapillary spaces. In ichthyosis simplex the cutis is unaffected.

**Treatment.**—Complete cure cannot be effected, but considerable relief can be obtained. The scales should first be removed by hot baths, alkaline baths, sulphur baths, friction with soap, etc., and then some emollient application should be kept constantly applied, such as glycerine of starch, vaseline, lanolin, cold cream, or olive oil; to these may be added resorcin (10 to 30 grains to the ounce) and salicylic acid (5 to 7 grains). Internally thyroid extract should be given in small doses over a long period. On the cessation of the treatment the former condition will return. For the smaller growths of ichthyosis hystrix Crocker recommended removing the horny caps and painting the base with a saturated solution of salicylic acid in alcohol. Radium has been found to be useful for *I. linearis*.

## ATROPHIC AND DYSTROPHIC CONDITIONS OF THE SKIN

### SCLERODERMIA

Sclerodermia may be diffused or circumscribed. In *diffused sclerodermia* there is a general hardening or induration of the skin, which begins most commonly about the face, neck, shoulders, chest, and arms, and may gradually extend to the lower part of the body. A similar condition may begin in the fingers, forming *sclerodactylia* or *acrosclerodermia* (*see* p. 381). There is at first no change in colour, but the skin is hard, rigid, inelastic, and cannot be pinched up into folds. As it progresses the movements of the limbs are hindered, the joints are

more or less fixed, the chest is limited in its respiratory movements, and if the face is affected it loses its power of expression; the mouth can be opened with difficulty, but the eyelids often retain their mobility. Subsequently the skin becomes shiny and glossy, irregular patches of pigment appear, and here and there are areas of vascular dilatation, giving a pink or violet colour. The secretions of sweat and sebum are diminished. The course of the disease is slow, and it extends over years, eventually, in many cases, subsiding entirely. During this time the patient's health is practically unaffected, but rheumatism and cardiac troubles have been noted as occasional complications. In the skin itself eczema, erythema, and ulceration may occur.

In some cases, the disease begins with more thickening or œdema of the skin, and this, according to Crocker, tends to result in an atrophied, rigid, tight condition, which is much less liable to spontaneous recovery than the simply indurated forms.

**Ætiology.** The diffused form is commonly associated with Raynaud's disease. Localised forms have occasionally followed injury, and more general conditions have been attributed to chills, to worry, and to the specific fevers. In recent years it has been regarded by various authors as due to disorders, whether of excess or defect, in the internal secretions, of the thyroid gland especially, but also of the suprarenals and of the pituitary body.

The disease occurs in young adults and middle-aged persons, less frequently in children, and hitherto not under the age of thirteen months (*see* Sclerema Neonatorum). It is more frequent in women than in men, but little is known of its causes. (*Vide* Ætiology of Vitiligo.)

In *circumscribed scleroderma*, or *morphœa*, there is an unsymmetrical patch of 2, 3, or more inches in diameter, frequently corresponding to the distribution of a nerve. For instance, a patch may occur over the distribution of the supraorbital nerve on the forehead; the trunk near the breast and the limbs are also common places for the eruption. The patches are irregular in shape, or may be in the form of bands round or along a limb. They are of a dead white ivory colour, surrounded by a violet or pink zone of dilated vessels. The skin is smooth and dry, and may often be pinched up; it may be level with the healthy skin, or below or above it. The disease lasts several years, and then subsides and disappears, or it may extend into the diffused form, or persist in an atrophic condition. Circumscribed sclerodermia is also more common in women than in men, and can sometimes be referred to local irritation as a cause.

**Anatomy.** – The epidermis is unaffected except for some pigment in the rete; there is a considerable overgrowth of connective tissue in the corium and subcutaneous tissues; the deeper vessels are surrounded by numerous leucocytes, and the superficial vessels are often contracted and empty. Leucocytes also surround and may obstruct the sweat gland ducts, and the muscular fibres of the skin are hypertrophied.

**Treatment.** – Very little can really be done in this disease. The patient should be kept warm at all times, and tonic remedies should be given. Locally emollient applications and friction, and shampooing to restore the circulation in the skin, and galvanism may be employed. The use of thyroid extract and the application of X-rays have been followed by improvement.

### SCLEREMA NEONATORUM

This is a peculiar induration of the skin, which is either congenital or appears shortly after birth in feeble infants with deficient circulation. It may begin in the lower extremities and spread to the rest of the body, or it occurs in scattered patches on the thighs, buttocks, trunk, arms, and cheeks. The affected parts feel quite hard and firm, suggesting that the subcutaneous tissue has been frozen. The patches have a well-defined edge, are slightly raised above the surface, and

sometimes have a bluish-red colour. They only pit after very prolonged pressure. The children are cold and drowsy, with small pulse and feeble respiration. They often die from collapse or diarrhœa, but occasionally recover. The cause of the change is not well understood.

It may be confounded with a true *œdema*, which occurs in similar circumstances. Œdema affects mostly the dependent parts; the skin is blue and mottled, can be pinched up from underlying structures, and pits readily on pressure.

**Treatment.**—The child should be kept warm and efficiently fed, by a nasal tube, if necessary.

Besides senile atrophy, in which the skin becomes dry, inelastic, wrinkled and often pigmented, the following conditions may be described as atrophy of the skin.

### ATROPHODERMIA NEURITICA

This, the "glossy skin" of Paget, follows upon neuritis and other lesions of the nervous system. It is especially well seen in the fingers, of which the skin becomes smooth, shining, dry, the colour pink or red, the whole finger tapering, and the nails curved longitudinally and transversely. With this is a severe and persistent burning pain.

### STRIÆ ET MACULÆ ATROPHICÆ

(*Atrophodermia striata et maculata*)

Striæ atrophicæ are translucent, scar-like lines in parts of the body which have undergone considerable distension, such as the abdomen after pregnancy (*lineæ gravidarum*, *lineæ albicantes*), the breasts after lactation, the abdomen, thighs, legs, and arms after extreme anasarca, and the shoulders, breasts, and thighs, from obesity or the presence of more localised fatty tumours. The lines are from 1 to 3 or 4 inches in length, tapering to a point at each end; they are slightly depressed below the surface of the healthy skin, but in the event of œdema or anasarca occurring in the part they project beyond it. A similar change may occur in the skin without any preceding distension, and this most commonly during some prolonged and prostrating illness, such as typhoid fever. It is then seen mostly about the buttocks, thighs, knees (*lineæ patellares*), and ankles; and the atrophic lines may be associated with circular spots, or *maculæ*, varying from $\frac{1}{6}$ inch to $\frac{1}{2}$ inch in diameter. In all these cases the skin is really atrophied, and the elastic tissue disappears; the epidermis is thinned, the papillæ are small or absent, and the subcutaneous tissue and glands are atrophied. But an early vascular, or inflammatory, or even hypertrophied, condition has been observed in the cases not related to distension; and in them the action of toxins is invoked as an explanation.

### XERODERMIA PIGMENTOSA

(*Kaposi's Disease*)

This is a remarkable and rare disease, which consists of combined atrophy of the skin, increased pigmentation, telangeiectasis and later the growth of malignant tumours. It occurs equally in males and females, and has a tendency to affect members of the same family without being actually hereditary. It begins in childhood, and generally in the summer-time, with pigment spots, or with erythematous spots, which soon fade into pigment. These form over the face, neck, scalp in the temporal region, outer side of the arm and forearm, and back of the hand. The pigment spots afterwards become atrophic, and patches of white, depressed, shrunken skin form among them. These white spots are slightly contracted, and difficult to pinch up; and subsequently sufficient tightening of the skin may occur to depress the eyelids, and set up conjunctivitis. On the atrophic area there occur pink spots of dilated vessels, which gradually enlarge.

The disease may remain stationary for a long time, and may never spread to other parts of the body; but eventually warty growths develop out of either the dilated vessels or the pigment spots, and these subsequently grow into tumours of an epitheliomatous nature. These fungate, discharge or bleed; and, other tumours forming in remote parts of the body, the patient is carried off by exhaustion.

**Treatment** can do little for this disease. As it is thought that exposure to the sun has something to do with the development of the disease, it is advisable to protect the face by thick red veils or by local applications. New growths may be excised as they arise, or be treated with radium or X-rays.

Kaposi described a *xerodermia albidum* (atrophodermia albida, Crocker) affecting the leg from the thigh downwards, and sometimes the arm down to the hand, in which the skin is atrophied and then stretched. It begins in early childhood and remains stationary.

## ALTERATIONS OF PIGMENT

Increase of pigmentation is a frequent result of intense or persistent hyperæmia, through which, no doubt, there is extravasation of hæmoglobin, but the links between this and the increase of the pigment naturally in the deepest layers of the epidermis are still obscure. The most familiar instance is exposure to the sun or to the wind; but in the foregoing sections it will have been noticed how frequently pigmentation is said to follow upon the different forms of dermatitis —for instance, eczema, erythema, pemphigus, lichen, and psoriasis; to these may be added erysipelas, syphilitic eruptions and ulcerations, and especially old-standing ulcers from varicose veins in the lower extremities. The application of blisters and mustard plasters is often also followed by staining, a fact which should make one careful how one orders these counter-irritants to the neck or arms of ladies. Another common traumatic cause of increased pigmentation is the scratching which is indulged in to relieve pruritus, especially that which results from severe prurigo, or from the presence of pediculi.

Some disorders of the skin in which hyperæmia is not a marked feature are also accompanied by pigmentation, such as sclerodermia, Kaposi's xerodermia, and leucodermia, which will be described presently. As a result of internal disease, we see pigmentation of an extreme form in Addison's disease, to a less extent in some cases of lymphadenoma, in the carcinomatous cachexia, in malaria, in Graves' disease, in rheumatoid arthritis, and in some cases of tuberculosis, of diabetes, and of cirrhosis of the liver. Interference with the solar plexus has been suggested for its origin in lymphadenoma and in Addison's disease, but toxic causes are highly probable in many of the above and in some other disorders, such as chloasma uterinum; and pigmentation is well known as a result of the internal use of arsenic. It is not common to employ any special name, but the terms *melanodermia*, *melasma* (melasma suprarenale) and *chloasma* (from χλοάζω, I am pale green) have been used in different instances. In all the cases which are due to a removable cause, the pigmentation will, in its absence, eventually disappear; on the other hand, it persists in incurable cases like Addison's disease, and increased pigmentation coming on in old age does not, of course, undergo any improvement. Local collections of pigment occur, as pigment moles and pigmented warts. The special forms to be here described are lentigo or ephelis, chloasma uterinum, and ochronosis.

Deficiencies of pigmentation are seen in albinism and leucodermia.

### LENTIGO

(*Ephelis, Freckles*)

Yellow, orange, or yellowish-brown maculæ appear on the face, neck, forearms, and backs of the hands, from exposure to the sun under certain conditions.

They are most marked during the summer-time, and fade or entirely disappear during the winter; they are first seen about the age of late childhood, and rarely in advanced life; and they affect especially people with fair hair and blue eyes (xanthochroic type). Of a similar kind are the mottled patches of pigment (*ephelis ab igne*) which are associated with erythema ab igne (*see* p. 973).

**Treatment.**—As a rule freckles are better left alone: they can only be removed with the epidermis in which the pigment lies. If urgently desired, this may be attempted by the method mentioned under Chloasma Uterinum.

### CHLOASMA UTERINUM

The pregnant state, as is well known, is commonly accompanied by an increased pigmentation of the nipples, axillæ, and the line between the umbilicus and the pubes. In some women, in these circumstances, a broad band of pigment forms on each side of the forehead, just below, but not touching, the margin of the scalp. It is narrower in the middle line, widens out as it reaches the temple, and may extend over the zygoma on to the cheek; it is continuous, or broken into separate small patches. The colour is yellow or brown. With it may be associated the familiar dark ring round the eyes. This frontal chloasma sometimes recurs with each successive pregnancy, and disappears with delivery. It may be due to other uterine disturbances *-e.g.* dysmenorrhœa -and sometimes no cause can be traced.

**Treatment.** -Mercury perchloride has been most used locally, in a solution of the strength of 1 or 2 grains to an ounce of almond emulsion, applied twice daily until the skin is reddened. Zinc ointment may then be applied. Solutions of citric acid, carbolic acid, and other mild caustics, by which the epidermis is removed, and with it the pigment, have also been used. But the colour is likely to recur. Crocker recommended salicylic acid paste, or plaster, or a saturated solution of the acid in alcohol kept on for some hours.

### OCHRONOSIS

Virchow gave this name to a rare condition, in which there is a black pigmentation of the skin, cartilages, and sclerotics; but whereas in these cases the cartilages have been constantly stained, the skin has been affected in only a few instances. The face is of coal-black or dark brown colour, darker than that of Addison's disease; the hands may present bluish-black areas, and patches have been seen on the mucous membrane of the lips. A black patch is seen in the sclerotics on each side of the cornea, midway between it and the canthus. The change in the cartilages is clinically observable in the ears, which have a bluish-grey colour, due to the blackened cartilage being seen through the thin skin; but *post mortem* the rib cartilages and the intervertebral, sterno-clavicular, laryngeal, and tracheal cartilages have been found of jet-black or inky black colour.

Some tendons have been smoky brown in colour, and the cardiac valves and chordæ tendineæ discoloured in patches. The pigment is deposited in the matrix of the cartilages and in the fibrous tissue of the corium of the skin.

Some of the cases have been associated with *alkaptonuria* (*see* p. 608), and a few with carboluria after the constant application of carbolic acid to chronic ulcers for many years.

### ALBINISM

This is a congenital deficiency of colour, not only in the skin, but also in the hair and in the iris and choroid. It is at once recognised by the white hair and the pink eyes; and there is commonly intolerance of light (*photophobia*), and it may be nystagmus, from the want of pigment in the fundus of the eye. It occurs

in dark races as well as in the pale-faced, and in various animals—cats, mice, and others.

### VITILIGO

(*Leucodermia*)

Vitiligo is an affection of the skin characterised by the depigmentation of certain areas and increased pigmentation of the zones immediately surrounding them. It is acquired, and must be distinguished from congenital leuco-melanodermia, which is nævoid in origin. The depigmented areas are milk-white in colour, oval or irregular in shape, and of varying size; their margins are sharply demarcated from the neighbouring hyperpigmented parts, the colour of which is usually of greater intensity at the edges of the white patches, fading off gradually at the periphery into the normal skin. The hairs covering the white patches may be also depigmented, or of normal colour.

The *Distribution* tends to be markedly symmetrical, and the backs of the hands, wrists, forearms, face and neck, the loins and genital organs are most frequently affected. The *evolution* appears to vary in different cases. The onset may be sudden or gradual, and the pigmentary change may slowly extend or remain stationary for years. Sunlight renders it more obvious by causing increased pigmentation of the dark areas, and thus accentuating the contrast. Partial recovery is common, but complete restoration of the skin to normal is rare. Sometimes the depigmentation becomes almost generalised.

**Ætiology.** Vitiligo is rare in very young children, and is seen chiefly in youth and middle age; it is commoner in the female sex. It is associated with Graves' disease, alopecia areata, scleroderma, or circumscribed prurigo in a considerable percentage of cases, and also with organic diseases of the nervous system, such as tabes, syphilitic meningo-myelitis, subacute combined degeneration of the cord, and involvement of the spinal cord or nerve-roots secondary to tuberculous caries, osteoarthritis of the vertebral joints, and spina bifida. A familial incidence is not uncommon, as in alopecia areata. There would appear to be no doubt that both these conditions and scleroderma are dependent on involvement of the sympathetic nervous system; this view, which is supported by experimental evidence, explains the frequency of their association with Graves' disease, and the influence of shock and worry as provoking factors. The sympathetic may be involved by organic central nervous disease, or more commonly by acute or chronic infection. There is considerable clinical evidence to show that chronic focal streptococcal infection is the most frequent cause of all three conditions.

**Anatomy.**—Histological examination shows absence of pigment in the white patches, whereas there is excess in the epidermis and dermis of the surrounding dark areas.

**Treatment.** This must necessarily depend on complete investigation of each individual case. A search should be made for evidence of organic nervous disease, of syphilis, and of chronic focal infection. It has been shown that repeated exposures of the affected skin over a long period to sunlight or the carbon arc light rays will gradually restore pigment to the white areas.

## AFFECTIONS OF THE SWEAT GLANDS

### ANOMALIES OF SECRETION

**Anidrosis,** or deficiency of perspiration, is seen in fevers, in diabetes and in some diseases of the skin—*e.g.* ichthyosis and pityriasis rubra.

**Hyperidrosis,** or excess of perspiration, may be general or local. General sweating results from dilatation of cutaneous blood vessels, as after exercise, or from emotional causes. Sometimes, on the other hand, it occurs with contracted

vessels, as in conditions of collapse or fear. General perspiration has been mentioned in connection with malaria, phthisis, pyæmia, and the crisis of acute illnesses. Local excess of sweating occurs from emotional causes and in rickets. A very troublesome form of excessive sweating is found about the hands and feet, axillæ, and genitals in some persons without any adequate cause. Some such sufferers are in deficient general health, but others are perfectly well.

The local application of belladonna liniment or the use of atropine of belladonna internally should be tried. A drop dose of liquor atropinæ will sometimes stop the sweating of phthisis for two or three successive nights. Profuse local sweating of the axillæ, and palms of the hands, has been successfully treated by the Röntgen rays. It may be also treated by the methods used for the next complaint.

**Bromidrosis.**—This is often associated with hyperidrosis—that is, the sweat is both offensive and excessive. It affects chiefly the feet and axillæ, and the odour is probably due to the decomposition of the fatty sebaceous material which is secreted with the sweat. It is not uncommon in young men or young women of the domestic class, and it may be quite independent of the general health. Thin has described a bacterium in connection with it—*Bacterium fœtidum*. It is essential to wash the feet thoroughly and frequently, and use astringents and antiseptics. The socks may be dusted inside with finely powdered boric acid, and should be frequently changed, or with a mixture of salicylic acid 3 parts, starch powder 10, and talc 87 parts; or the feet may be painted with a 5 per cent. solution of chromic acid, or smeared with a salicylic ointment of 2 per cent. strength, or an ichthyol ointment of 5 per cent. strength. It has been also recommended to spread glycerine over the soles of the feet, and over the toes, before the socks are put on in the morning, and to repeat this every morning as long as required. A cure may take place in three days (Benians).

**Chromidrosis,** or coloured sweat, is a rare affection, and is perhaps sometimes due to indican. But the possibility of its being feigned should always be remembered.

**Hæmatidrosis,** or sweating of blood, also quite rarely occurs, mostly in highly neurotic people.

**Uridrosis** is the name given to some cases in which the sweat has crystallised on the surface, and the crystals have been found to contain urea and salts.

### MILIARIA

This name is given to rashes determined by profuse secretion of sweat, such that it is unable to escape by the ducts, and either raises small vesicles in the epidermis or sets up a local inflammation.

*Miliaria crystallina* or *Sudamina*.—In this form there are small transparent vesicles, not larger than a pin's head, due to the elevation of the most superficial layer of the epidermis by accumulated sweat. They are found most abundantly on the chest and abdomen, but from their perfect transparency may be better felt than seen. The vesicles dry up, and leave a few branny scales, the remains of the detached epidermis. They are most common in phthisis and in enteric fever.

In *miliaria rubra*, there are vesicles produced in the same way, but accompanied by inflammation. They are surrounded by a red areola, and contain a yellow, turbid alkaline fluid or actual pus. It is not uncommon to see these in the course of rheumatic fever.

Sweating in infants, as a result of wrapping them too closely in binders or napkins, produces a papular form of miliaria (formerly called *strophulus* or *red gum*); and the prickly heat of hot countries (formerly *lichen tropicus*) is regarded as *miliaria papulosa*. It affects chiefly the trunk and thighs, is accompanied by severe itching, and does not materially influence the general health. (*Vide* p. 945.)

## AFFECTIONS OF THE SEBACEOUS GLANDS

### MILIUM

This is a small bright, white, round tumour, the size of a pin's head or slightly larger, which results from complete obstruction of the duct of a sebaceous gland. Such little tumours are seen on the forehead, eyelids, cheeks, and genitals. Occurring in children, they were formerly called *strophulus albidus*. Sometimes they are met with in great numbers on thin cicatrices, especially those of lupus. The contents are cholesterin and fatty material. They can be treated by puncturing the skin and squeezing out the contents.

### ADENOMA SEBACEUM

This is really a mixed tumour, consisting partly of an overgrowth of the sebaceous glands and partly of vascular new growth (angioma). The tumours are of small size, being seldom larger than a pea. They occur chiefly on the nose, especially the alæ, on the adjacent portions of the cheeks, on the centre of the forehead and upper lip, and more rarely as isolated lesions on the scalp and neck. As a rule their distribution is symmetrical. Their colour varies from brown to light red, according to whether the sebaceous or the vascular overgrowth predominates. Frequently sebaceous adenomas are associated with pigmented moles, telangiectases, and patches of pigment on other parts of the body, and they occur most frequently, but not always, in persons of defective mental development, epileptics, and idiots. Though of congenital origin, the tumours do not as a rule appear until some years after birth.

**Treatment.**—The tumours can be removed by the cautery or electrolysis, and the larger ones may, if necessary, be excised or curetted out.

### SEBACEOUS CYSTS

(*Wens*)

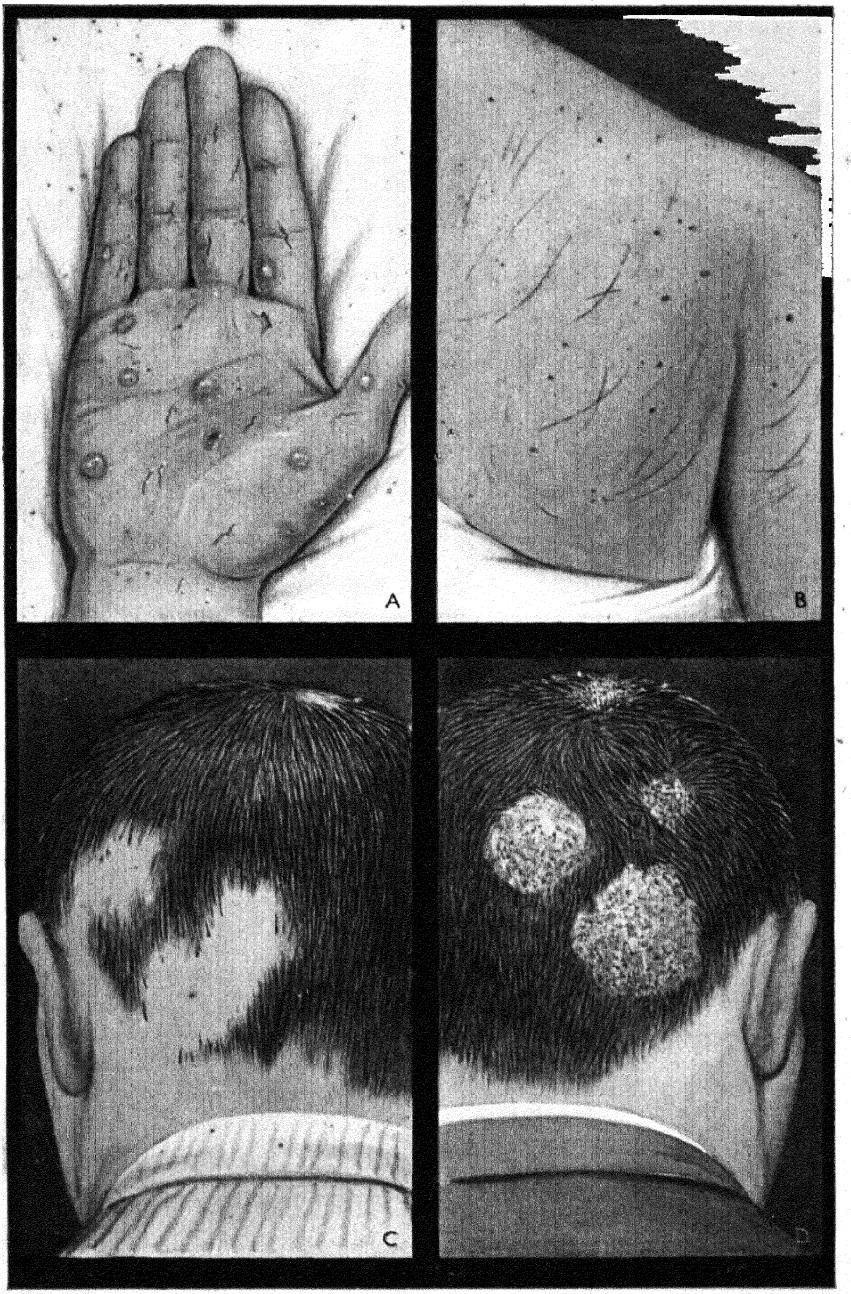
These are retention cysts arising from obstruction to the ducts of the sebaceous follicles. They are most common on the scalp, but occur on the eyebrows, face, or neck, and more rarely on the trunk or limbs. In size they vary from that of a pea to that of a nut or an orange. They are hemispherical, or more globular, uniform and smooth. The skin looks thin, and often presents well-marked vessels ramifying over it. The contents are semi-fluid or pasty, and consist of animal fats, albumin, epidermic cells, cholesterin, and earthy salts enclosed in a capsule made up of layers of epithelial cells and fibrous tissue.

The **Treatment** consists in incising the cyst, squeezing out the contents, and tearing out, or dissecting out, the cyst wall entirely.

## AFFECTIONS OF THE HAIR AND HAIR FOLLICLES

The hair may be developed to an excessive degree as a congenital phenomenon; this is very rare, and is called *hirsuties*. Deficiency or falling of the hair, known as *alopecia*, is, on the other hand, exceedingly common. The following three conditions are quite rare: *trichorrhexis nodosa*, in which some of the hairs are found to present little nodules or thickenings due to the splitting up of the cortical fibres; *monilethrix*, in which the hair looks beaded, and breaks readily at the internodes, so that it is only 2 or 3 inches long all over the head; and *lepothrix*, in which the hairs of the axilla, scrotum and perineum are brittle, and present irregular masses on and around them, consisting of clumps of bacteria enmeshed in a homogeneous viscid substance.

The most common *change of colour* in the hair is that known as *canities*: the




A. Scabies. Note the numerous sinuous "runs" or burrows, and the vesicles, which have become secondarily infected with pyogenic cocci.

B. Pediculosis Corporis.—Shows the scapular and posterior axillary regions, one of the sites of election in this condition. Note the linear scratch-marks and blood crusts.

C. Alopecia Areata. Note the smoothness of the baldness, their depression below the general surface of the hairy scalp owing to atrophy and fall of the hairs, the absence of scales, and the "exclamation-mark" hairs, lying at the periphery of the patches and indicating active spread of the alopecia.

D. Tinea Tonsurans.—Note the more-or-less circular outline of the patches, the fact that they are not depressed because the infected hairs break off, but do not fall, the irregular "lie" of the latter, and the presence of scales.

[*To face p* 1015.

hair gets successively grey and white. This is a senile change which may occur prematurely. But occasionally sudden whitening of the hair occurs after fright, intense emotion, or in consequence of neuralgia. It is probably due to the development of air bubbles, which conceal the pigment. The colour sometimes returns spontaneously, but nothing can be done for it.

Of the above ailments *alopecia* will be more fully described.

### ALOPECIA

Baldness is most familiar to us as it appears in old age (*senile alopecia*), though it occurs also in quite young people (*premature alopecia*). Different degrees of atrophy of the hair follicles and structures of the skin have been found in old cases of baldness, but its immediate antecedent is doubtful. Premature baldness is not explained by mental exertion, wearing tight hats, insufficient lubrication of the scalp, etc., but it is certainly sometimes hereditary, and it is constantly associated with the presence of pityriasis capitis or seborrhoic eczema, so that micro-organisms are now often regarded as the primary cause. The hair thins first at the back of the vertex, and at the front part of the temples. Ultimately there is only a fringe left round the temples and occiput. A temporary alopecia is caused by transient interference with the nutrition of the hair bulbs; thus the hair falls not infrequently after fevers, after erysipelas, in the second stage of syphilis, and in the parasitic diseases which will presently be described. A localised alopecia is a necessary part of any scar following ulceration deep enough to destroy the hair follicles.

Premature loss of hair is practically incurable, though it may be checked or prevented by adequate treatment of the pityriasis or other disorder of the scalp which precedes it. Restoration of the hair after fevers, etc., may be hastened by the use of tonics internally, faradising by means of the wire brush, and by the use of stimulants, such as oil of mace, cantharides, and ammonia, which will be again mentioned under the next complaint.

### ALOPECIA AREATA. (*See Plate XLVIII.*)

(*Area Celsi*)

This form of alopecia usually begins by the sudden appearance of a bald patch on the scalp, which gradually increases in size. Frequently other patches are discovered soon afterwards either near to or at a distance from the original patch, and by coalescence large areas of baldness may result, or less commonly the hair of the whole scalp is lost. Other hairy parts may be affected, particularly the eyebrows, and the beard region in males, but the disease usually, though not always, attacks the scalp first. Recent patches of alopecia areata are pinker than the surrounding scalp, and on them the orifices of the empty follicles, often filled with sebaceous matter, are clearly visible. Older patches become pale, absolutely smooth, and depressed below the level of the unaffected parts of the scalp owing to the atrophy of the hair. At the edges of a patch, and sometimes all over it, are seen the characteristic "exclamation mark" hairs. These are from $\frac{1}{16}$ to $\frac{1}{2}$ inch in length, and their distal end is frayed out and appears thickened, while the proximal part is very thin and devoid of pigment. On extracting such a hair it will be found to come out entire and without a bulb; *i.e.* it has left its papilla and is in the process of being shed. These "exclamation mark" hairs may sometimes be seen after X-ray treatment has been given for ringworm of the scalp, but otherwise they are characteristic of alopecia areata; when, however, the fall of hair has ceased, they may, of course, be entirely absent, and their presence at the edges of the patches is evidence that

the disease is still progressing. After a while new hair begins to make its appearance, as a rule in the central older parts of the patches; at first it is fair and downy, but in time it acquires the normal colouring, although in some cases it may return quite white and remain so indefinitely. As a rule, complete regrowth of hair takes place eventually, but there may be a partial return, associated with persistent baldness in certain areas, lasting for years; recurrent attacks of alopecia areata are far from uncommon, and occasionally the hair is permanently lost over part or the whole of the scalp and other hairy regions. The disease is apt to be most persistent in those cases in which a complete band of baldness is formed around the lower and posterior part of the scalp (ophiasis of Celsus).

Alopecia areata may be associated with dystrophy and atrophy of the nails, and also with leucomelanoderma (vitiligo). It also occurs in Graves' disease more frequently than can be explained by mere coincidence. Acute infections such as influenza, erysipelas, scarlet fever, and follicular tonsillitis may not only be followed by a general fall of hair, but also by typical alopecia areata.

**Ætiology.**—Various hypotheses have from time to time been suggested as to the cause of the disease. The view that it is due to a parasitic infection of the hair follicles has now been abandoned by most dermatologists, although a very rare epidemic variety may perhaps be of parasitic origin. Many still regard it as a trophoneurosis, and in adults whose occupation involves eye strain Whitfield has shown that alopecia areata may apparently be due to an error of refraction, since it disappears when this is corrected. Jacquet, whose careful work did much to disprove the parasitic theory, attributed most cases of the disease to reflex irritation from the teeth or nasopharynx. Recently Barber and Leslie Roberts have shown that, even in cases of long standing, regrowth of hair may rapidly take place after removal of chronic foci of infection, such as occur in the teeth, tonsils, and nasal sinuses, with or without subsequent vaccine treatment, and since true alopecia areata not infrequently appears after acute infections, particularly erysipelas, influenza, scarlet fever, and tonsillitis, it is reasonable to suppose that a chronic infection may also be responsible for the disease. As in vitiligo and scleroderma the condition depends on involvement of the sympathetic nerve-fibres.

The sexes are about equally affected, and, although the condition may be met with at almost any age, it is commoner in children and adults below the age of forty. A familial incidence is extremely common. (*Vide* Ætiology of Vitiligo.)

**Diagnosis.**—This, as a rule, is not difficult. In ringworm the patches are usually scaly, and not smooth or depressed, and the fungus is easily demonstrable in the scales and in the characteristic broken stumps. In lupus erythematosus of the scalp there are scaly erythematous patches, which leave permanent scars and lead to complete destruction of the hair follicles; moreover, the scalp is rarely affected alone in this disease. In folliculitis decalvans and pseudo-pelade there is also atrophic scarring, with destruction of the follicles.

**Treatment.**—Many cases recover spontaneously without treatment, but the appearance of alopecia areata should lead to a thorough examination of the patient. Chronic infection in the mouth, nasopharynx, tonsils, nasal sinuses, and elsewhere, should be sought for and dealt with, and in persistent or recurrent cases removal of septic foci should be followed by vaccination with the predominant organism (usually a *Streptococcus longus*) isolated from them, since secondary foci in lymphatic glands may exist. In adults the possibility of eye strain being responsible should be remembered. Tonics, particularly iron, arsenic, and strychnine, are certainly often of value, and if the patient is suffering from worry and nervous exhaustion, complete rest and small doses of bromide should be prescribed. In some cases thyroid gland preparations are indicated. Locally the scalp should be rendered as healthy as possible by treating seborrhœa or pityriasis, if present, and the bald patches should be stimulated by painting them from time to time with pure carbolic acid, pure lysol, or a mixture of one part

carbolic and four parts lactic acid; the irritation produced by such applications certainly hastens the regrowth of hair, and seems actually to prevent the spread of the patches. In chronic cases the bald areas may be treated with high frequency currents or the ultra-violet light with gratifying results.

### UNIVERSAL ALOPECIA AND CONGENITAL ALOPECIA

In the former the hair falls from every part of the body, scalp, eyebrows, axillæ and pubes, so that not a hair is left. As a rule, no cause can be assigned, and the cases are incurable.

In the latter the nails are wanting as well as the hair. These cases may be hereditary.

## NEW GROWTHS IN THE SKIN

Only a small number of these will be here described. For *nævi*, or vascular tumours, *carcinoma*, *epithelioma*, *rodent ulcer*, and *sarcoma* the reader is referred to works on surgery.

### FIBROMA MOLLUSCUM

This is a soft, flaccid, wrinkled, often pendulous tumour, consisting of a covering of scarcely altered cutis and epidermis, containing a fibrous meshwork with a variable proportion of round cells and albuminous fluid. The tumours may be very few, or exceedingly numerous; they vary in size from a pin's head to the head of a man, and they occur especially on the trunk. They may apparently be congenital, but are generally first seen in early childhood. According to von Recklinghausen, they are really neuro-fibromata, starting from the fibrous sheaths of the smaller cutaneous nerves, and thence invading the fibrous structures of the vessels, the sweat glands, and the hair follicles. In an allied condition there are no separate tumours, but the skin is thickened and overgrown, or lies in large, loose, overlapping folds (*dermatolysis*). Cases of *multiple fibroma* are rare; they are known as *Recklinghausen's disease*.

**Treatment.** Removal by the knife is the only possible treatment; but if the tumours are very numerous, only such growths can be removed as are in specially inconvenient positions.

### CHELOID

Cheloid is a growth of the skin and subcutaneous tissue, consisting chiefly of dense bands of fibrous tissue, containing in its earlier stages numerous spindle cells. Its more common seats are on the chest, over the sternum, on the mammæ, on the neck, back, lobules of the ears, and on the limbs. It is usually single. It begins as a flat, smooth, pink nodule, which extends laterally to a considerable size and becomes paler in the centre, while the skin around is more or less reddened. After a time bands and ridges, separated by furrows, develop, running in various directions across the tumour and into the surrounding skin. By the slow contraction of these bands much deformity may be caused, and movements of adjacent joints may be seriously restricted. The growth of the tumour is often accompanied by considerable pain and tenderness.

Similar growths not infrequently develop on former scars, such as those of cuts, burns, acne, varicella, vaccination, or small-pox. These have been called false cheloid, but it does not seem that they are essentially different. Cheloid grows slowly, and rarely disappears spontaneously. If removed by a knife or caustic, it almost inevitably returns; but it always remains a strictly local disease, invading neither lymph glands nor viscera.

**Treatment.**—Cure has been obtained by the use of Röntgen rays, and by radium. Small cheloids may be successfully removed by repeated freezing with carbon dioxide snow.

### MYOMA, NEUROMA, LYMPHANGEIOMA

These occasionally occur as cutaneous affections.

Myoma occurs in rare cases as multiple small, hard nodules, from the size of a pin's head to that of a pea or bean, on the face, trunk, or limbs. Each is a small tumour in the corium, consisting of smooth muscular fibres (*leiomyoma*), related, apparently, in some cases to the arrectores pilorum.

*Neuroma* forms multiple, painful small growths in the course of the nerve fibres of both trunk and limbs (*see* p. 743).

*Lymphangeioma* is a rare growth, due to dilatation of lymphatic vessels into visible cysts and overgrowth of the intervening connective tissue. It has been seen in association with ordinary vascular nævi.

### XANTHOMA

(*Xanthelasma, Vitiligoidea*)

In the common variety (*X. planum*), one finds generally on each upper eyelid, near the inner canthus, a small sharply defined patch of whitish-yellow, soft, smooth skin, level with, or scarcely raised above, the general surface. Such patches may remain stationary for years, or may slowly increase, spreading outwards along each upper lid, or other patches appear on the lower lids, and the orbit is completely surrounded by a broad patch of the altered skin. In many cases no other part of the body is affected; but in others spots and streaks of a similar kind appear on the trunk, on the backs and palms of the hands and the soles of the feet, or on the scrotum. They have also been seen in the mucous membrane of the gums, palate, side of the tongue, larynx and trachea, and in the mucous lining of the bile ducts.

Another form (*X. tuberosum*) consists of firm, rounded nodules, from the size of a pea to that of a nut, occurring on the skin over the elbow, on the knuckles, on the lobules of the ears, and on the buttocks. These growths occur, as a rule, in persons of middle age or older, and are more frequent in women than in men; they may, however, be met with in young children.

The most extensive lesions in the above forms are sometimes associated with long-standing jaundice, but small patches affecting the eyelids alone are often seen without jaundice, and were thought by Hutchinson to be related to attacks of sick headache.

*Xanthoma diabeticorum* is a rare form which occurs usually in those who have glycosuria. It appears as yellow conical spots surrounded by a red raised area; these are seen first on the extensor surfaces of the arms, and at the lower part of the back and abdomen, and subsequently in other parts. They often subside rapidly.

**Pathology.**—Recent research has shown conclusively that xanthoma is associated with cholesterinæmia, *i.e.* a high blood content of cholesterol, and the skin lesions are due to the deposition of cholesterol in the tissues, where it induces a secondary connective tissue hyperplasia. Such deposition is particularly liable to occur at sites of friction or pressure. Xanthoma diabeticorum is not always accompanied by glycosuria, and the association, when it occurs, is due to the cholesterinæmia which is associated with the lipæmia (*see* p. 568) in diabetes.

**Anatomy.**—In the flat patches the corium shows a newly formed connective tissue, with large round or fusiform cells, often multinucleated, filled with fat granules and closely aggregated fat drops (*xanthoma cells*). The nodules have a similar structure, but the fibrous tissue is more abundant. In a very rare form (*xanthoma of Balzer*) the chief lesion is thickening and deformity of the elastic fibres.

**Treatment.**—Small patches may be excised, but the removal of larger

patches from the eyelids would risk serious deformities. If their removal is called for, caustics are to be preferred. In other cases the galvano-cautery at a dull red heat, or the Röntgen rays, may be employed.

### MYCOSIS FUNGOIDES

In this rare disease, the skin is affected by a number of tumours, the appearance of which is often preceded, sometimes for years, by erythematous, eczematous, or urticarial patches. The tumours vary from the size of a bean to that of an orange, or larger. They are round, oval, or lobulated; the skin is stretched over them, tense and shining; the skin around them is often infiltrated. After a long duration some of them may shrink and disappear, others ulcerate on the surface and form fungating masses, discharging a clear watery serum. In this stage they are generally painless and free from itching or smarting. The disease lasts months or years, but ultimately the health fails, and the results have often been fatal. The view has been held that the disease is an infective granuloma rather than a sarcoma or a lympho-sarcoma; but no organisms have been found, and the tumours consist of lymphoid cells in a fine stroma of connective tissue. (*Vide also* Premycosic Erythrodermia.)

**Treatment.**—Great improvement, though sometimes only temporary, has been recorded from the use of Röntgen rays.

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# APPENDIX

## THE ÆTIOLOGY OF MALIGNANT NEOPLASMS

In 1888, Hanau transplanted a tumour from one rat into another; but the expérimental as distinguished from the purely histological study of neoplasms only began after Jensen (1902) effected the transplantation of a carcinoma among mice.

Subsequent work carried out under the auspices of the Cancer Research Fund showed that this was a true transplantation, the cells of the transplanted portion multiplying and themselves forming a new tumour. More recent observations of Fibiger, that the presence of neumatodes in the stomachs of rats caused carcinoma, and of Bullock and Curtis, that sarcoma developed in the walls of cysticercus cysts in the livers of rats, all tended to make people believe that cancer was due not to a germ, but to an abnormal proliferation of the cells of the organism in response to irritation, and this seemed to be confirmed when numerous workers succeeded in producing carcinoma in the skins of mice and rabbits by painting them repeatedly with tar.

However, Peyton Rous (1911) had produced sarcoma in chicks, not merely by transplanting the cells from a fowl, but by introducing an extract which had been rendered cell-free by filtration. This agreed with the earlier observations of Bellingham Smith and Washbourn (1897) that in the dog a sarcoma could be transmitted from vagina to penis during coitus, and pointed to an infective origin of cancer.

Only this last summer, W. E. Gye (*Lancet*, II, p. 109, 1925) has proved the infective origin of cancer by showing that both carcinoma and sarcoma of fowls, mice, rats, dogs and man, contain an ultra-microscopic virus, probably intracellular. However, the virus alone does not produce a tumour when injected, but only when there is added to it a virus-free extract of a tumour. The latter is called the "specific factor," since it is only obtained from the cells of the tumour which it is desired to reproduce, and which must be growing in the same species in which it is desired to reproduce it. The virus itself is not specific, and the virus from a carcinoma will, in fact, cause sarcoma if the specific from a sarcoma is present. Thus, in one experiment, the virus from a human carcinoma added to a fowl sarcoma specific produced no effect when introduced into a mouse, but produced sarcoma when introduced into a fowl.

Further, J. E. Barnard (*loc. cit.*, p. 117) has described the virus from microscopical examination, viewing it by illumination against a dark ground, and photographing it with ultra-violet light. It appears as a minute spheroid, and develops by particles, which appear on the edge of the spheroid and themselves turn into spheroids.

# INDEX


**Abdomen,** auscultation of, 391
examination of, 389
inspection of, 389
palpation of, 390
percussion of, 391
X-ray examination of, 391
**Abdominal** discomfort in gastroptosis, 416
type of influenza, 63
**Abductors** of vocal cords, paralysis of, 208
differentiation from syphilitic laryngitis, 205
**Abscess** or **Abscesses.**
alveolar, differentiation from facial erysipelas, 54
amœbic in dysentery, 167
at apex of roots of teeth, 392
causing obstruction of bronchi, 230
causing pulmonary embolism, 269
complicating dysentery, 165
cutaneous, in melioidosis, 137
differentiation from gonococcal infections, 74
gout, 666
gangrene of lung following, 247
hepatic, causing subphrenic abscess, 512
in appendicitis, 452
lumbar, 512
malignant endocarditis in, 330
mediastinal, 384
metastatic, in chronic pneumonia, 246
of brain, 836
*See also* Cerebral abscess.
of liver, 479
*See also* Liver, abscess of
of lung, 245
*See also* Lung, abscess of
pelvic, in appendicitis, 452
perigastric, 422
perinephric, 639
differentiation from cancer of kidney, 653
hydronephrosis, 645
perisplenic, 512
peritonsillar, 396
pleurisy complicating, 271
pulmonary, following mustard gas poisoning, 266
in melioidosis, 137
pyæmic, pleurisy in, 272
retropharyngeal, 400
rheumatoid arthritis and, 668
subcutaneous and intramuscular, in filariasis, 555
subphrenic, 422
in appendicitis, 452
in peritoneum, 512
simulating pleurisy, 276
tonsillar, 396
**Acanthosis,** 985
**Acapnia,** 585
**Accessory** food factors, 5, 685
**Accommodation** of the eye, defects of, in diabetes, 570
**Aceto-acetic** acid, 568
as a poison, 548
in blood, 547
in urine in diabetes, 569
secretions in cyclical vomiting, 418
**Acetone,** 568
in cyclical vomiting, 418
in urine in diabetes, 569
**Achalasia,** of anus, Hirschsprung's disease secondary to, 466
of cardia, 403
differentiation from carcinoma of œsophagus, 402
**Achlorhydria,** 407, 419
**Acholuric** jaundice, 474, 531
resemblance to pernicious anæmia, 529
**Achondroplasia,** 674
*Achorion gypseum,* 956
*Quinckeanum,* 956
*Schoenleini,* 956
**Achylia** gastrica, 418
achlorhydria in, 434
due to neutralisation of HCl, 408
eczema associated with, 987
in pernicious anæmia, 527
*See also* Achlorhydria *and* Hypochlorhydria.
**Acid** dyspepsia, 412
hæmatin spectrum in urine, 647
**Acidæmia,** 547
due to accumulation of $CO_2$, 548
in chronic interstitial nephritis, 629
in suppurative nephritis, 638
**Acidosis,** 547, 568
**Acne** chloid, 942
frontalis, 944
necrotica, 944
rosacea, achlorhydria in, 434
achylia associated with, 419.
varioloformis, 944
vulgaris, 937
differentiation from follicular syphilide, 111
rosacea, 975
**Acromegaly,** 593, 594
differentiation from osteitis deformans, 676
**Acromicria,** 595
**Acroparæsthesia,** 883
**Acrosclerodermia,** 1007
*Actinomyces bovis* 140–141
**Actinomycosis,** 140
in appendicitis, 452
of tonsil, 398
*Actomyx bovis,* 183
**Acu-puncture** in chronic parenchymatous nephritis, 627
**Acute** bulbar paralysis, 810
yellow atrophy of liver, 481 *See also* under Liver
**Adams-Stokes** syndrome, 306
**Addison's anæmia,** 523, 526
*See also* Anæmia, pernicious.
**Addison's disease,** 589
differentiation from jaundice, 474
neurasthenia, 888
growths in capsules, 590
low arterial tension in, 371
thrombosis of vessels in, 590
*See also* Suprarenal glands
**Adductors** of vocal cords, paralysis of, 209
spasm of, differentiation from paralysis of abductors of vocal cords, 209
**Adenitis,** tuberculous, differentiation from glandular fever, 50
**Adenoid** facies, 399
**Adenoids,** 398, 399
impetigo contagiosa associated with, 927
**Adenoma,** encapsuled, of thyroid gland, 577
of pituitary gland, 594
of stomach, 434
sebaceum, 1014
toxic, of thyroid gland, 577, 578
operative treatment, 581
**Adeno-papilloma** in bilharziasis, 650
**Adherent** pericardium, 359
**Adhesions** due to gastric ulcer, 422
intestinal obstruction due to, 459
in treatment by artificial pneumothorax, 263
of peritoneum, 516
perigastric, 423
pleural, 272
**Adhesive** pylephlebitis, 502
**Adiposis** dolorosa, 595, 599

**Adiposity**, under-activity of pituitary gland causing 593
**Adrenalin** in arseno-benzol poisoning, 123
in heart block, 307
**Adrenin**, 589
hydrochloride in treatment of asthma, 229
normal functions of, 589
**Adventitious** sounds, 194
*Ædes argenteus*, 176, 178
*variegatus*, 554
**Ægophony**, 195
in pleurisy, 274
**Aerophagy**, 415
associated with spasmodic stricture of œsophagus, 402
cause of dyspeptic pain, 410
dyspeptic, 415
spasmodic stricture of œsophagus in, 402
**Aeroplane** dope, acute yellow atrophy of liver caused by, 481
**Æstivo-autumnal** infection, 153
**Age**, relation to basal metabolism, 559, 560
**Agglutination**, 16
tests in cholera, 181
diagnosis of paratyphoid fevers, 93
differential diagnosis of Mediterranean fever, 173
**Agglutinins**, 16
**Agraphia**, 719, 723
**Ague**, 149. *See also* Malaria.
**Air** hunger in diabetic coma, 570
**Alastrim**, 38
**Albinism**, 1011
**Albuminuria**, athletic, 661
causes of, 615
conditions in which found, 615
detection of, 613
dietetic, 661
functional, 660
globulin ratio in, 615
hyperpiesia and, 370
in acute bulbar paralysis, 811
in acute nephritis, 620
in acute yellow atrophy of liver, 482
in bubonic plague, 184
in cholera, 180
in chronic bronchitis, 219
in chronic interstitial nephritis, 629
in chronic parenchymatous nephritis, 625
in cystic disease of kidney, 653
in delirium tremens, 678
in diabetes, 569
in diphtheria, 68
in embolism of kidney, 380
in emphysema, 232
in endocarditis lenta, 333
in enteric fever, 83
in erysipelas, 54
in glanders, 137
in Henoch's purpura, 543
in lead poisoning, 681
in lobar pneumonia, 240
in mediastinitis, 384
in Mediterranean fever, 173
in myocardial degeneration, 325
in primary contracted kidney, 632
in pyelitis, 636
in renal calculus, 657
in scarlet fever, 30
in small-pox, 39
in spirochætosis, 175
in syphilis, 112
in tertian fever, 153
in trypanosomiasis, 160
in tubercle of kidney, 641
in yellow fever, 176, 177
lordotic, 661
of pregnancy, 661
orthostatic, 661
postural, 661
quantitative tests in, 614
**Albuminuric** retinitis, 629
**Albumoses** in urine, 616
production of, by bacterial action, 11
**Alcohol** as cause of primary contracted kidney, 631
causing multiple neuritis, 740, 741
**Alcoholic** intoxication as cause of acute encephalitis, 829
**Alcoholism**, 677
chronic, 678
**Alcoholism**, chronic, fatty degeneration of heart in, 323
differentiation from general paralysis, 863
fibroid degeneration of heart in, 324
**Aleukia** hæmorrhagica, 529
**Alexine**, 17
**Alimentary** system in psychopathology, 891
toxæmia, peritoneal adhesions and, 516
**Alkalæmia**, 547, 585
associated with ketosis, 549
high altitudes causing, 547
**Alkali** reserve of blood, 547
raised in tetany, 585
**Alkaline** treatment of rheumatism, 60
**Alkalosis**, 547
**Alkaptonuria**, ochronosis associated with, 1011
urine in, 608
**Allergic** state and **Allergy**, 224
dermatitis herpetiformis and, 1005
dyspepsia in, 412
nature of, 226
resemblance to anaphylaxis, 17
**Alopecia**, 1014
areata, 1015
differentiation from syphilitic alopecia, 111
following acute infections, 1016
regrowth of hair following removal of chronic foci of infection, 1016
congenital, 1017
universal, 1017
**Alternation** of pulse, 313
**Altitudes**, high, causing alkæmia, 547
residence at, clubbing of fingers in, 673
**Alveolar** carbon dioxide, measurement of, in acidæmia, 548
ectasis, 231
test in ketosis, 569
*Amaurosis fugax* in arteriosclerotic retinitis, 368
**Amaurosis**, uræmic, 621
**Amblyopia** in diabetes, 570
in lead poisoning, 681
**Ambulatory** typhoid, 87
**Amenorrhœa** in chlorosis, 525
in lead poisoning, 682
**Ammonia** nitrogen ratio in acidæmia, 548
test in ketosis, 568
**Amnesia**, hysterical, 904
**Amœba** coli in peritonitis, 510
**Amœbic** abscesses in dysentery, 167
**Amphoric** breathing, 192, 194
in phthisis, 253
**Amphoteric** reaction of urine, 609
**Amyl** nitrite in angina pectoris, 364
**Amyloid**. *See* Lardaceous.
**Amyotonia** congenita, 918
**Amyotrophic** lateral sclerosis, 802
**Amyotrophy**, 914
**Anacrotic** pulse in aortic stenosis, 340
**Anæmia**, 522, 523
aplastic, 529
purpura in, 542
cerebral, in Adams-Stokes syndrome, 306
chlorosis, 525
differentiation from cerebral tumour, 856
valvular heart disease, 344
due to Dibothriocephalus latus resembling pernicious anæmia, 445
dyspepsia in, 412
glossitic, 526. *See also* Anæmia, pernicious.
idiopathic, 523, 526
in acholuric jaundice, 531
in acute nephritis, 621
in aortic regurgitation, 341
in bilharziasis, 650
in carcinoma of kidney, 652
in carcinoma of stomach, 432
in chronic mercurial poisoning, 683
in hæmophilia, 544
in Hodgkin's disease, 552
in kala-azar, 161
in lead poisoning, 681, 683
in malignant endocarditis, 332, 333
in Mediterranean fever, 172
in mixed infections, 154
in myelocytic leukæmia, 534
in phthisis, 255
in scurvy, 639
in tertian fever, 153
intestinal worms causing, 471
megaloblastic, 527

**Anæmia,** megalocytic, 527
of larynx in phthisis, 203
pernicious, 526
achlorhydria in, 419, 434
associated with subacute combined degeneration, 527, 794
differentiation from carcinoma of stomach, 433
jaundice, 474, 476
neurasthenia, 888
secondary anæmia by latent jaundice, 476
splenic anæmia, 530
fatty degeneration of heart in 323
hæmolytic jaundice in, 476
resemblance to sprue, 448
pseudo-leukæmica infantum, 530
secondary, 523, 531
causing tinnitus, 750
differentiation from pernicious anæmia by latent jaundice, 476
septic, 529
severe allied forms of, 529
splenic, 502, 529, 550
differentiation from malignant endocarditis, 334
**Anærobic** bacilli in malignant endocarditis, 330
**Anæsthesia,** cutaneous, in tabes, 789
hysterical, 905
in subacute combined degeneration, 795
of larynx, 212
**Anæsthetics,** general, in auricular fibrillation, 316
in simple paroxysmal tachycardia 312
ventricular fibrillation in, 318
**Analgesia** in subacute combined degeneration, 795
**Anaphylactic** shock, 18
**Anaphylatoxin,** 17
**Anaphylaxis,** 17, 225
in blackwater fever 157
in hæmophilia, 545
in urticaria, 976
**Anasarca** in chronic parenchymatous nephritis, 625
in failure of heart, 337
in mitral disease, 342 *See also* Œdema.
*Anchylostoma duodenale,* 471
**Aneurysm,** abdominal, 377
aortic, compression of trachea by, 214
differentiation from mediastinal neoplasms, 387
arterial, murmurs in, 289
arterio venous, murmurs in, 289
auscultation in, 301
causing bronchiectasis, 222
causing obstruction of bronchi, 229
clubbing of fingers in, 673
differentiation from
syphilitic laryngitis, 205
valvular heart disease, 345
dissecting, 371
fusiform, 371
local, causing œdema of lungs, 236
murmurs in, 373
of coronary arteries, 326
of heart, acute and chronic, 326
pulmonary artery in phthisis, 255
thoracic aorta, 373
valves in malignant endocarditis, 331
of physical signs, 373
of symptoms, 374
pressure from, 372
causing gangrene of lung, 247
ruptured, simulating peritonitis, 511
sacculated, 371
symptoms of, 372
thoracic, differentiation from asthma, 228
paralysis of abductors of vocal cords in, 209
treatment of, 376
varicose, 373
**Angeio-neurotic** œdema, 383
in toxic idiopathies, 225
of laryngeal tissues, 200
**Angina** minor, 363
pectoris, 361, 373
differentiation from acute pulmonary œdema, 237
hyperpiesia and, 370
in aortic regurgitation, 341
tobacco, 363
**Anginal** feeling in simple paroxysmal tachycardia, 311
pain in estimating heart's capacity for effort, 294
**Anidrosis,** 1012
**Animal** parasites, diseases of skin due to infection with, 964
**Anisocoria** in aneurysm, 375
**Anisocytosis,** 520
**Ankle** clonus, 735
jerk, 734
jerks disappearance in multiple neuritis, 741
*Ankylostoma duodenale.* See *Anchylostoma.*
cause of anæmia, 523
*Anopheles maculipennis,* 151
*Anopheles* in malaria, 149
**Anorexia** nervosa, 412
**Anosmia,** 743
in cerebral tumours, 855
**Anthelmintic** treatment, 468
**Anthracosis,** 246
**Anthrax,** 137
cutaneous, 139
**Antibacterial** serum, 15
**Anti-beri-beri** factor, 686
**Anti-bodies,** 11, 16
**Antigens,** 16
**Antimicrobic** serum, 15
**Antimony** in bilharziasis, 651
in kala-azar, 162
in trypanosomiasis, 160
**Antipyretic** drugs, 25
treatment, 25
of typhoid fever, 90
**Antipyrin** causing skin eruptions, 979
**Antirachitic X** vitamin, 685
**Antiscorbutic** factor, 686
**Antiseptic** treatment of typhoid fever, 91
**Antiseptics** in diphtheria, 71
**Antispasmodics** in chronic bronchitis, 220
in whooping-cough, 49
**Antitoxic** serum, 16
in scarlet fever, 32
*See also* Serum.
**Antitoxins,** 15, 16
**Anuria,** 618
**Anxiety** neurosis, 894
neurosis, differentiation from neurasthenia, 888
**Aorta** and pulmonary artery, transposition of, 353
aneurysm of, angina pectoris in, 361
congenital coaretation of, 377
fardaceous disease of, 489
tension of, as cause of angina, 362
thoracic, aneurysm of, 373
differentiation from mediastinal neoplasm, 387
**Aortic** area, systolic murmur in, in aortic regurgitation, 340
diastolic murmurs, in aortic regurgitation, 340
disease, measurement of hypertrophy in, 319
regurgitation, 339
anæmia in, 523
murmurs in, 290, 291
stenosis, 339
murmurs in, 289, 291
**Aortitis,** syphilitic, angina pectoris in, 362
*See also* Syphilis.
**Apathy** in encephalitis lethargica, 834
**Aphasia,** 723, 820
in cerebral embolism, 837
in hemiplegia, 818
in migraine, 878
motor, 719, 723
in cerebral tumours, 853
sensory, 719, 724
**Aphonia,** differentiation from aphasia, 820
functional, 209
hysterical, 209, 905
**Apicolysis,** 263
**Apnœa,** 189
**Apoplexy,** 826
**Appendicitis,** 451
as cause of acute peritonitis, 508
suppurative pylephlebitis, 502
causing reflex diarrhœa, 440
causing subphrenic abscess, 512
chronic, 452, 454
differentiation from chronic gastric ulcer, 425
dyspepsia in, 411
differentiation from acute gastritis, 421

**Appendicitis,** differentiation from biliary colic, 500
distended gall bladder, 500
enteric fever, 88
intestinal obstruction, 462
perinephric abscess, 640
pneumonia, 241
gastroptosis in, 416
**Appendicostomy** in muco-membranous colitis, 450
in ulcerative colitis, 451
**Appendix,** controlling, 435
dyspepsia, 454
**Appetite,** causing hypertonic stomach, 405
**Apraxia,** motor, 719
in cerebral tumours, 855
**Area** cœli, 1015
**Argyll-Robertson** pupils, 726
in encephalitis lethargica, 726, 832
in general paralysis of insane, 863
in tabes, 789
**Arrhythmia,** cardiac, 304
**Arsenic** causing multiple neuritis, 740
causing skin eruptions, 979
in treatment of Hodgkin's disease, 553
leukæmia, 535
pernicious anæmia, 528
**Arsenical** neuritis, 741
pigmentation, differentiation from pigmentary syphilide, 111
poisoning, 683
differentiation from intestinal obstruction, 463
herpes zoster in, 999
**Arsenobenzol,** 116, 117
in treatment of actinomycosis, 143
anthrax, 139
framboesia, 125
malaria, 156
rat-bite fever, 144
relapsing fever, 148
syphilis, 116
tabes, 792
poisoning, 123, 473, 777
causing acute yellow atrophy of liver, 481
jaundice in, 476
provocative dose of, when Wassermann reaction is negative, 115, 119
treatment with, latent jaundice in, 476
**Arsenobillon,** 116, 118
**Arsphenamine,** 116, 118
**Arterial** pressure, high, 369
low, 371
**Arteries,** auscultation of, 301
coronary, aneurysms of, 326
diseases of, atheroma, 365
diffuse hyperplastic sclerosis, 366
in primary contracted kidney, 631
senile degeneration, 366
blood pressure in, 366
pulmonary, embolism and thrombosis of, 268
retinal, thrombosis of, in arterio-sclerosis, 368
silver wire, in arterio-sclerosis, 367
walls of, variation in thickness on palpation, 300
*See also* Blood pressure
**Arterio-capillary** fibrosis, 366
**Arterio-sclerosis,** ætiology of, 367
cause of chronic pancreatitis, 506
cerebral, 368
differentiation from dementia paralytica, 863
blood pressure in, 367
diabetes mellitus and, 562
hypothyroidism causing, 582
in gout, 664
in primary contracted kidney, 631
ophthalmoscopic examination in, 367
retinitis in, 368
**Arteritis,** 365
local, acute and subacute, 365
obliterans, 113
obliterative, in intermittent claudication, 382
syphilitic, 113, 365
*See also* Syphilis.
**Arthritis deformans,** 668
gonococcal, 73
infective, causes of, 667
in dysentery, 165
in Mediterranean fever, 172
in rheumatic fever, 56
muscular atrophy in, 914
rheumatoid, 668
**Arthropathy** in tabes, 791
**Artificial** pneumothorax in treatment of phthisis, 262
**Arytenoids,** ankylosis of, differentiation from paralysis of abductors, 209
*Ascaris lumbricoides,* 469, 875
**Aschoff's** nodes, 322
**Ascites,** 477
chylous and chyliform, 478
in carcinoma of liver, 492
in chronic parenchymatous nephritis, 625
in chronic peritonitis, 513
in heart disease, 338, 342
in perihepatitis, 501
in portal cirrhosis, 485
in splenic anæmia, 529
in tuberculous peritonitis, 514
pseudo-chylous, 478
sign of hepatic inefficiency, 473
**Asiatic cholera,** 179
**Aspergillosis,** 144
*Aspergillus fumigatus,* 144
**Asphyxia,** local, in Raynaud's disease, 381
**Aspiration** in treatment of hydronephrosis, 645
of amœbic abscess of liver, 171
**Association** fibres in spinal cord, 703
**Astasia-abasia,** 906
**Astereognosis** in cerebral tumours, 854
in disseminated sclerosis, 781
in hemiplegia, 819
**Asthma,** 224
cardiac, 227, 325
differentiation from acute bronchitis, 218
following mustard gas poisoning, 266
infection and, 227
prevention of, 228
renal, 227
secondary to bronchitis, 227
thymic, 587
uræmic, 227
*See also* Bronchial spasm
**Ataxy** in chronic mercurial poisoning, 683
in subacute combined degeneration, 796
**Atelectasis** pulmonum, 234
**Ateleiosis,** 600
**Atheroma,** 365
angina pectoris in, 361
causing lesions of valves of heart, 336
gout predisposing to, 667
in intermittent claudication, 382
in syphilis, 113
**Atheromatous ulcer,** 366
**Athetosis,** 730
in encephalitis lethargica, 834
in hemiplegia, 819
**Atlas** and axis, tuberculosis of, 754
**Atophan** in gout, 664, 667
in neuritis, 740, 758, 762
**Atoxyl** in treatment of trypanosomiasis, 160
**Atrophodermia** neuritica, 1009
striata et maculata, 1009
**Atrophy,** muscular, 731, 914
in neuritis, 739
peroneal, 798
optic, due to pressure from tumours, 594
in arteriosclerotic retinitis, 368
in diabetes, 570
in infantile cerebral diplegia, 839
in lead poisoning, 681
in tabes, 791
primary and secondary, 745
**Atropine,** injection of, in angina, 364
in cholera 182
test in enteric fever, 88
**Auricles,** dilatation of, 321
hypertrophy of, 320
**Auricular** extra systoles, 308, 309
fibrillation, 302, 313
causing pulmonary infarcts, 269
in mitral regurgitation, 342
stenosis, 343
in myocardial degeneration, 325
in toxic adenoma of thyroid, 580
flutter, 312
**Auriculo-systolic** murmurs, 289
not heard in mitral stenosis, 315
*See also under* Murmurs
**Auriculo-ventricular** heart block, 305
bradycardia in, 318
bundle, 285
node, 285

**Auscultation,** 192
in measurement of blood pressure, 300
of abdomen, 391
of arteries, 301
of heart, 287
of the cough, 196
of veins, 303
of voice, 195
**Autographism,** 977
**Axilla,** temperature of, normal, 18
**Axis** and atlas, tuberculosis of, 754
**Ayerza's** disease, chronic bronchitis with, 219

**Bacilli,** infection by, 9
**Bacilluria,** 634
*Bacillus abortus* in Malta fever, 173
*acnes,* 936
*aerogenes capsulatus,* 108
*anthracis,* 138
bottle, 925
in pityriasis simplex, 935
*botulinus* in food poisoning, 447
*coli communis,* 142
as cause of gall stones, 500
in amœbic abscess, 170
in appendicitis, 452
in cholecystitis, 496
in gangrene of lung, 247
in meningitis, 843
in methæmoglobinæmia, 541
in peritonitis, 510
in pleurisy, 271
in pyelitis, 635
in rheumatoid arthritis, 668
in suppurative cholangitis, 495
in suppurative pancreatitis, 505
in ulcerative colitis, 450
in urine, 634
*diphtheriæ,* 66, 69
in broncho-pneumonia, 243
in infective endocarditis, 331
in pleurisy, 271
*dysenteriæ,* 163
in ulcerative colitis, 450
*enteritidis* in acute gastritis, 420
(*ertrycke*) in food poisoning, 447
*of Gaertner* in food poisoning, 447
*fluorescens putridus* in gangrene of lung, 247
Friedlander's, causing pneumonia, 238
in broncho-pneumonia, 243
in gangrene of lung, 247
in infective endocarditis, 330
in lobar pneumonia, 238
in pleurisy, 271
*fusiformis,* in ulcerative stomatitis, 393
in Vincent's angina, 397
*influenzæ,* 61
in acute encephalitis, 829
in broncho-pneumonia, 243
in lobar pneumonia, 238
in meningitis, 843
in pleurisy, 271
Klebs-Lœffler, 66, 69
*lactis aerogenes,* 442
in peritonitis, 510
*lepræ,* 133
*mallei,* 136
*œdematiens,* 108
*paratyphosus A,* 79, 92
*paratyphosus B,* 79, 92
*perfringens,* 108
*pertussis,* 47
*pestis,* 183
*proteus,* in gangrene of lung, 247
in typhus, 78
*pyocyaneus,* in appendicitis, 452
in peritonitis, 510
*suipestifer* in food poisoning, 447
*tetani,* 104
*tuberculosis,* 125
and myelitis, 772
enlargement of tonsils due to, 398
in appendicitis, 452
in gangrene of lung, 247
in infective endocarditis, 331
in meningitis, 843
in pericarditis, 356
in pleurisy, 271
in urine, in tubercle of kidney, 641
*Bacillus tularense,* 185
*typhosus,* 79
as cause of gall stones, 498
in appendicitis, 452
in cholecystitis, 496
in infective endocarditis, 331
in meningitis, 843
in peritonitis, 510
in pleurisy, 271
in suppurative cholangitis, 495
**Bacterial** endocarditis, 330
infections of kidney, 634
**Bacteriology,** definition of, 8
**Bacteriolysis,** 16
*Bacterium pneumosintes,* 61
*whitmori,* 137
**Balanitis** complicating diabetes, 569
**Baldness,** premature, in myotonia atrophica, 918
**Banting, F. G.,** work on insulin, 564
**Banting's** treatment in obesity, 600
**Banti's** disease, 484, 502, 529
*See also* Splenic Anæmia.
**Barbados** leg, 556
**Barlow's** disease, 690
**Barnard, J. E.,** recent work on carcinoma, 1020
**Basal** metabolic rate, 558
**Basedow's** disease, 578
**Basophilia** in differentiation from hydatid disease, 268
**Bastedo's** sign, 455
**Bath,** permanganate, in small-pox, 42
**Baths** in treatment, 7
of fibrositis, 911
subthermal, in obesity, 600
**Bayer "205"** in treatment of trypanosomiasis, 160
**Bazin's** disease, 953
**Beard,** ringworm of, 958
**Bedsores** in disseminated sclerosis, 783
treatment of, 771
**Beggiatoa,** 10
**Behaviour,** cause of, 717
**Bell-sound,** 196
in pneumothorax, 280
**Bell's** palsy, 100, 748
**Benedict's** qualitative test for sugar, 566
**Benign** tertian malaria, 153
**Benzoin** test of cerebro-spinal fluid, 738
**Benzol,** handling of, inhalation of, purpura from, 542
in treatment of leukæmia, 535
**Benzyl** benzoate in treatment of asthma, 229
**Bergonié** treatment in obesity, 600
**Beri-beri,** 686
causing multiple neuritis, 740
**Bial's** orcin test for pentose in urine, 567
**Bile,** disinfection of, 497
inspissated, causing jaundice, 476
pigment in blood in acholuric jaundice, 531
tests for, 475
regurgitation of, after gastro-jejunostomy, 427
salts in urine, Hay's test for, 475
Van den Bergh's test in serum, 475
**Bile Ducts,** carcinoma of, due to gall stones, 500
catarrh of, 480
congenital obstruction of, in new-born, 477
obstruction of, causing jaundice, 476
stricture or obliteration of, causing jaundice, 476
*Bilharzia hæmatobia,* renal calculus and, 657
*hæmatobium,* 649
*japonica,* 650
**Bilharziasis,** 172, 649
portal cirrhosis in, 484
**Biliary** colic in gall stones, 498
**Bilious** attacks, relation to migraine, 876
vomiting in acute dilatation of duodenum, 420
**Bilirubin,** formation by reticulo-endothelial system, 475
in blood serum in jaundice, tests for, 475
**Biot's** respiration, 130, 189
**Birth** injury, 838
**Bisferiens** pulse in aortic stenosis, 340
**Blackhead** (comedo), 935
**Blackwater** fever, 157
**Bladder,** carcinoma of, complicating bilharziasis, 650
dilatation and hypertrophy of, in diabetes insipidus, 596
distended, simulating ascites, 478
distension of, in pyrexia, 21
disturbances of reflexes of, 735
examination of, 603

**Bladder**, innervation of, 721
paralysis of, treatment of, 771
tuberculosis of, 641
**Blastomycosis**, 961
**"Bleeders,"** 544
**"Bleeding time"** in purpura, 542
**Blindness**, hysterical, 904
in arteriosclerotic retinitis, 368
**Blood**, arterial, sugar in, insulin and, 563
bicarbonate in *See* Alkali reserve of blood
bile pigment in, in acholuric jaundice, 531
cells in urine *See* Hæmaturia
cholesterol in, xanthoma associated with, 1018
coagulation of, in thrombosis, 379
coagulation time of, determination of, 549
colour index of, 520
compatibility of, in transfusion, 546
concentration of, in cholera, 180
corpuscles in, microscopic examination of, 520
-count, method of, 549
cultures in enteric fever, 87, 88
in malignant endocarditis, 334
in paratyphoid fevers, 93
defibrinated, in blood transfusion, 546
prophylactic inoculation in typhus fever, 79
delayed coagulation of, in hæmophilia, 544
dextrines in, in pancreatic disease, 505
dextrose concentration in, in diabetes, 564
diseases of, 519
examination of, 519
excess of uric acid in, 664
fragility of corpuscles, 519
in acholuric jaundice, 532
hæmoglobin in, estimation of, 520
in cerebro-spinal fluid, 736
in fæces. *See under* Fæces
in urine *See* Hæmaturia
isolation of micro-organism of rheumatic fever in, 55
platelet-counting, 520
platelets *See also* under Platelets.
pressure, diastolic, 300
in aortic regurgitation, 340
in acute nephritis, 620, 621
in primary contracted kidney, 674
in senile degeneration of arteries, 366
low, 301, 371
lowered, in Addison's disease, 590
measurement of, 300
raised, 301, 369
epistaxis in, 198
in chronic alcoholism, 679
in chronic interstitial nephritis, 629
in cystic disease of kidney, 654
in hyperpnœa, 369
Price-Jones cell distribution curve, 521
reaction of, 547
acapnia in, 585
showing lipæmia in diabetes mellitus, 568
sugar tolerance curves, 561, 565
with insulin treatment in diabetes, 572
transfusion of, with indications, 545
in diphtheria, 71
in hæmophilia, 545
in malignant endocarditis, 334
in purpura, 544
in septicæmia, 50
in splenic anæmia, 530
urea in, estimation of, 610
*See also under* Urea in blood
venous, sugar in, insulin and, 563
vessels, diseases of, 365
examination of, 297
viscosity of, increased in hyperpnœa, 369
visible, in carcinoma of stomach, 434
*See also* Plasma
**Blue** Cross shells, poisoning by, 265
line in lead poisoning, 681
**Bockhardt,** impetigo of, 927, 938
**Body** length, relation to basal metabolism, 560
weight, relation to basal metabolism, 559, 560
**Boils,** 943
complicating diabetes, 569
**Bone-marrow**, infection of, causing aplastic anæmia 529
in leukæmia, 532
**Bones,** diseases of,
achondroplasia, 674
osteitis deformans 675
osteogenesis imperfecta, 675
**Bones,** diseases of,
osteomalacia, 673
fracture of, in sarcoma, carcinoma and myeloma, 674
marrow in, excessive in pernicious anæmia, 527
tuberculosis of, with phthisis, 256
**Borax** and boric acid causing skin eruptions, 979
**Borborygmi,** 391, 416
in carcinoma of intestine, 456
**Bordet-Durham** reaction, 16
**Bordet-Gengou** fixation of complement, 114
reaction in diagnosis of tuberculosis, 258
*Bothriocephalus latus* *See Dibothriocephalus latus.*
cause of anæmia, 523
differentiation from pernicious anæmia, 527
*Bothriomyosis hominis*, 946
**Bottle-bacillus,** 925
**Botulism,** 148
**Bovine** and human tubercle, distinction between, 127
**β-oxybutyric** acid, 568
in blood, 547
in urine in diabetes, 569
**Brachial** plexus, lesions of, 757
**Bradycardia,** 317
in convalescence after acute infectious disease, 318
in jaundice, 474
in Malta fever, 173
vagal stimulation in, 317
**Brain,** abscess of, 836
*See also* Cerebral abscess.
actinomycosis of, 142
amœbic abscesses in, 167
arteries of, 816
compression of, in meningeal hæmorrhage, 828
cyst of, 825
diseases of, 812
in leukæmia, 534
localisation of functions and effects of lesions, 813
softening and abscess of, in malignant endocarditis, 334
softening of, 826
red, 825
white, 822
yellow, 822
tumours of, 849
*See also* Cerebral tumour.
**Branch-bundle** block, 304
**Breathlessness** following gassing, 265
paroxysmal, in myocardial disease, 349
**Breath** sounds, diminution of, in subphrenic abscess, 512
mode of production of, 289
**Bright's** disease, anæmia in, 523
ascites in, 477
bronchitis in, 215
differentiation from convulsions in epilepsy, 873
diabetes insipidus, 597
neurasthenia, 888
dyspepsia in, 412
epistaxis in, 198
hydrothorax in, 278
hyperpnœa in, 370
meaning of term, 611
pathology of, provisional theory of, 612
pleurisy in, 271
predisposing to peritonitis, 509
purpura in, 542
**Broadbent's** sign in adherent pericardium, 359
**Broca's** area, 719
**Bromides** causing skin eruptions, 979
**Bromidrosis,** 1013
**Bronchi,** obstruction of, 229
in mediastinal neoplasms, 386, 387
**Bronchial** breathing, 193
glands, enlarged, compression of trachea by, 214
tuberculosis of, 385
tuberculous infiltration of, 249
muscles, spasm of, in acute bronchitis, 217
spasm in cardiac asthma, 325
in congenital heart disease, 354
*See also* Asthma
**Bronchiectasis,** 216, 249, 222, 230
cerebral abscess in, 837
chronic, pneumonia following, 245
chronic, pyæmia from, 51
differentiation from phthisis, 259
in pneumonia, 241
in syphilis of lung, 264
in whooping-cough, 49
neoplasm of lung causing, 266

**Bronchiolectasis,** 223
**Bronchiolitis,** 216
**Bronchitis,** 197, 215
acute, 217
asthma secondary to, 227
capillary, 216, 217
differentiation from broncho-pneumonia, 244
causing bronchiectasis, 222
chronic, 218
chronic pneumonia following, 245
complicating emphysema, 233
whooping-cough, 48
differentiation from acute tuberculosis, 132
phthisis, 259
fibrinous, 221
fœtid, 219, 221
following mustard gas poisoning, 266
in chronic parenchymatous nephritis, 626
in enteric fever, 85
in rickets, 694
plastic, 221
purulent, 216
putrid or fœtid, 219
resulting from foreign body in bronchus, 230
**Bronchocele,** 576
**Bronchophony,** 195
in pleurisy, 274
**Broncho-pneumonia,** 216, 243
caseous, 249
causing bronchiectasis, 222
chronic pneumonia following, 245
complicating enteric fever, 86
measles, 35
whooping-cough, 48
confluent, 243
differentiation from acute tuberculosis, 132
in acute poliomyelitis, 100
in infantile enteritis, 445
in pulmonary embolism, 269
in rheumatic fever, 57
interstitial, 244
primary, 243
secondary, 243
tuberculous, 249
**Bronchorrhœa,** 219
**Broncho-vesicular** breathing, 193
**Bronchus,** carcinoma of, 266
**Brown** atrophy of heart, 523
**Brown-Séquard** syndrome, 712
*Bruit d'airain,* 192, 196
in diaphragmatic hernia, 282
in pneumothorax, 280
*Bruit de diable,* 303
in anæmia, 524
*Bruit de pot fêlé* in phthisis, 253
**Bruits.** *See* Murmurs.
**Bubo,** "climatic," differentiation from plague, 185
**Bubonic** plague, 183, 184
**"Buffers,"** function of phosphates in urine, 605
**Bulbar** paralysis, 748
acute, 810
anæsthesia of larynx in, 212
causing complete paralysis of vocal cords, 208
differentiation from myasthenia gravis, 920
ophthalmoplegia in, 746
progressive, 809
*Bullimus africanus,* 649
*contortus,* 649
*dybowski,* 649
**Bullous** eruptions of skin, 1002
**Bundle,** auriculo-ventricular, 285
**Bunion,** 1006
**Bursitis** in gonorrhœal rheumatism, 73
**Buttonhole** orifice of mitral valve, 335

**Cadaveric** position of vocal cords, 208
**Cæcostomy** in ulcerative colitis, 451
**Caisson** disease, 778
**Calabar** swellings, 557
**Calcifying** vitamin, 685
**Calcium** chloride in treatment of hæmoptysis, 264
of tetany, 586
of purpura, 544
in serum, low, parathyroid in treatment of, 584
lack of, in rickets, 691
oxalate in urine, 606
**Calculus** complicating bilharziasis, 650
in ureter, impacted, cause of hydronephrosis, 644
**Calculus,** renal, 665
differentiation from tuberculous kidney, 642
dyspepsia in, 411
formation of, 657
**$CO_2$,** fixed, in blood. *See* Alkali reserve of blood
in stomach in dyspepsia, 412
**Calf** lymph, glycerinated, for vaccination, 43
**Callosities,** 1005
**Calmette's** ophthalmic reaction, 257
**Cancer.** *See* Carcinoma.
**Cancrum** oris, 394
**Canities,** 1014
**Canter-rhythm,** 288, 305
**Capillary** pulsation in aortic regurgitation, 341
**Carbohydrate** metabolism, physiology of, 562
**Carboluria,** ochronosis associated with, 1011
**Carbon** dioxide pressure in blood in chronic bronchitis, 219
in neoplasm of lung, 267
tetrachloride in *ancylostomes* infection, 472
in *tænia saginata* infection, 468
**Carbuncle,** 943
complicating diabetes, 569
**Carcinoma,** abscess of lung following, 245
causing bronchiectasis, 249
causing jaundice, 476
causing suppurative cholangitis, 495
causing thrombosis of inferior vena cava, 380
chyluria due to, 556
fracture of bones in, 674
gangrene of lung in, 247
Gye and Barnard's recent work on, 1020
intestinal, intussusception and, 465
intestinal obstruction caused by, 458
intrathoracic, differentiation from chronic pneumonia, 246
in vertebræ causing compression of cord, 767
mediastinal, 386
of bladder complicating bilharziasis, 650
of brain, 850
of bronchus, 266
of colon, differentiation from cholecystitis, 497
of gall bladder, differentiation from aneurysm, 377
due to gall stones, 500
production by foreign bodies, 500
of kidney, epithelial, 651
differentiation from renal calculus, 659
of larynx, differentiation from chronic catarrhal laryngitis, 202
laryngeal tuberculosis, 203
syphilitic laryngitis, 205
extrinsic, 206
intrinsic, 206
of liver, 491
causing ascites, 477, 486
jaundice from, differentiation from catarrhal jaundice, 481
secondary growths in, in carcinoma of stomach, 433
of œsophagus, 401
of pancreas, 507
of peritoneum causing ascites, 477
colloid, 517
secondary, 517
of stomach, 430
achlorhydria or diminished HCl in, 434
differentiation from aneurysm, 377
chronic gastric ulcer, 425
pernicious anæmia, 528
metastases and, 431
ulcer and, 431
of suprarenal capsules, symptoms due to activity of cells, 592
of thyroid gland, 577
of tonsils, 318
producing hour-glass contraction of stomach, 429
**Cardia,** achalasia of, 403
mechanism of opening, 403
**Cardiac** action, abnormalities of, 303
asthma, 227, 325
disease, ascites in, 477
failure in endocarditis lenta, 333
primary contracted kidney, 631
irregularities in chronic interstitial nephritis, 629
pain, differentiation from chronic dyspepsia, 409
syncope, differentiation from minor epilepsy, 872
*See also under* Heart.
**Cardial** sphincter. *See* Cardia.

**Cardialgia**, 412
**Cardiolysis** in mediastinitis, 385
**Cardio-pulmonary** murmurs, 293
in hypertrophy, 320
**Cardio-spasm**, 403
**Carrel's** continuous drainage for empyema, 278
**Carriers**, acute poliomyelitis, 98
cerebro-spinal fever, 94, 97
cholera, 179
diphtheria, 66
of infection, 12
typhoid, 81, 89
**Carrotin**, yellow colour due to, differentiation from jaundice, 474
**Caseous** broncho-pneumonia, 249
lobar pneumonia, 249
**Casoni's** intradermal test in hydatid disease of liver, 495
**Casts**, fatty, doubly refracting, in myelin kidney, 624
in urine in acute nephritis, 620, 621
in chronic interstitial nephritis, 629
in chronic parenchymatous nephritis, 625
various types of, 617, 618
**Cataphoresis** in treatment, 5
**Cataract** in diabetes, 570
**Cataracts**, perinuclear, in latent tetany, 586
**Catarrhal** jaundice, 480
sore throat, 395
**Catheterisation** of ureters in diagnosis of pyelitis, 637
of tuberculous kidney, 642
**Cauda** equina, lesions of, 766
differentiation from polyneuritis, 742
**Cavernous** breathing, 194
**Cellulitis** of neck, œdematous laryngitis in, 200
submaxillary, 395
**Centrifugal** fibres, differentiation of function among, 707
**Centripetal** fibres, differentiation of function among, 704
**Cereals** in diet, excess of, effect on production of rickets, 691
**Cerebellar** ataxy, hereditary, 839
differentiation from Friedreich's ataxia, 798
in infantile cerebral diplegia, 838
disease, differentiation from tabes, 792
hæmorrhage, 829
tumour, associated with vertigo, 751
differentiation from disseminated sclerosis, 783
Friedreich's ataxia, 798
**Cerebellum**, function of, 715
lesions of, symptoms of, 816
tumours of, symptoms of, 855
**Cerebral** abscess, 836
complicating bronchiectasis, 224
differentiation from cerebral tumour, 856
encephalitis lethargica, 834
suppurative meningitis, 844
tuberculous meningitis, 132
in chronic pneumonia, 246
arteries, embolism of, 825
thrombosis of, 825
arteriosclerosis, paroxysmal headache due to, differentiation from migraine, 879
compression, bradycardia in, 317
in meningeal hæmorrhage, 828
diplegia, infantile, 838
disease, fits due to, 876
embolism, 837
hæmorrhage, 821
differentiation from pachymeningitis interna hæmorrhagica, 848
due to birth injury, 838
gout predisposing to, 667
hyperpiesia and, 370
in cystic disease of kidney, 654
in primary contracted kidney, 632
or thrombosis, differentiation from encephalitis lethargica, 834
pachymeningitis, 847
sinuses, thrombosis of, 848
tumour, adiposity in, 599
causing lesions of optic radiations, 745
causing internal hydrocephalus, 861
differentiation from encephalitis lethargica, 834
general paralysis, 863
syphilitic meningitis, 846
tuberculous meningitis, 132
**Cerebral** tumour, in Bell's palsy, 748
localising signs in, 853
**Cerebro-spinal** fever, 93
causing internal hydrocephalus, 861
differentiation from tuberculous meningitis, 132
purpura in, 542
*See also* Meningitis.
**Cerebro-spinal** fluid, albumin content in, 737
bacteriology of, 737
benzoin test, 738
blood in, 736
cell content of, 737
cell-count of, in poliomyelitis, 99
chlorides in, 737
colloidal reactions, 738
examination of, 736
in acute myelitis, 774
poliomyelitis, 100
in cerebral abscess, 838
in compression of spinal cord, 769
in encephalitis lethargica, 835
in pneumococcal meningitis, 845
in spinal meningitis, 806
in suppurative meningitis, 844
in syphilitic meningitis, 846
in tabes dorsalis, 787, 792
in trypanosomiasis, 160
in tuberculous meningitis, 131, 132
pressure of, 736
Wassermann reaction in, 738
in general paralysis, 861
*See also* Lumbar puncture.
meningitis, 93
**Cerebrum**, examination of function of, 723
reflex function in, 716
**Cervical** ribs, 758
differentiation from progressive muscular atrophy, 801
**Cestoda**, 466
**Chancre**, Hunterian or hard, 110
soft, 954
**Chancres**, hard and soft, difference between, 955
**Chancroid**, 954
**Charcot's** disease in tabes, 791
joints, rheumatoid arthritis and, 669
**Charcot-Leyden** crystals, 222, 228
in myelocytic leukæmia, 533
**Charcot-Marie-Tooth** disease, 798
**Chaulmoogra** oil in treatment of leprosy, 135, 136
**Cheiropompholyx**, 1001
**Cheloid**, 1017
false, 1017
**Chemiotaxis**, 16
**Chest**, actinomycosis of, 141
ectoscopy in examination of, 188
examination of, 187
exploration of, in diagnosis of neoplasms of lung, 267
in pleurisy, 276
inspection of, 187
palpation of, 190
paracentesis of, 277, 282
percussion of, 190
tightness in, following gassing, 265
**Cheyne-Stokes** respiration, 189
in myocardial degeneration, 325
in pulmonary embolism, 270
**Chicken-pox**, 44
and herpes zoster, relationship between, 999
bullosa, 45
differentiation from small-pox, 42, 45
urticaria papulosa, 978
gangrenosa, 45
quarantine period in, 13
**Chilblain**, 970
**Child-crowing**, 211
**Children**, rheumatism in, 58
**Chloasma uterinum**, 1011
differentiation from Addison's disease, 591
**Chloral** causing skin eruptions, 980
**Chlorides** in cerebro-spinal fluid, 737
in urine, 605
**Chloroform** poisoning, jaundice in, 476
**Chloroma**, 537
in leukæmia, 533
**Chloropicrin**, poisoning by, 265
**Chlorosis**, 523, 525
**Cholæmia**, family, resemblance to pernicious anæmia, 529

**Cholangitis,** 495
catarrhal, 480
in cholecystitis, 496
in gall bladder, 498
suppurative, as cause of abscess of liver, 479
**Cholecystectomy,** 497, 501
**Cholecyst-enterostomy** for pancreatic cysts, 508
**Cholecystitis,** 496
acute pancreatitis in, 505
catarrhal or suppurative, 496
causing reflex diarrhœa, 440
differentiation from appendicitis, 455
from pneumonia, 241
from gall stones, 498
gangrenous, 496
membranous, 496
phlegmonous, 496
**Cholecystotomy,** 497, 501
for pancreatic tumours, 508
**Cholelithiasis,** 497. *See also* Gallstones.
**Cholera,** 179
differentiation from intestinal obstruction, 463
*Choleræ vibrio,* 179
**Cholerine,** 181
**Cholesterin** crystals in gall-bladder disease, 500
**Cholesterinæmia,** xanthoma associated with, 1018
**Chorea,** 864
acute endocarditis in, 328
and rheumatic fever in children, 58
differentiation from habit spasm, 881
writer's cramp, 884
Huntington's, 867
in rheumatic fever, 55
paralytic, 866
spasm of glottis in, 211
**Choreiform** movements, 730
in encephalitis lethargica, 834
in Friedreich's ataxia, 797
**Choroid,** tubercles in, 130
plexus, papilloma of, 850
tubercle in, 132
**Choroiditis** in syphilis, 112, 122
**Chromaffine** body, 589
**Chromatolysis** in encephalitis lethargica, 832
**Chromidrosis,** 1013
**Chromogens** in urine, 608
**Chronaxie** of muscles, 733
**Chrysops,** spread of tularæmia by, 185
**Chvostek's** sign in latent tetany, 586
**Chyliform** effusion, 279
**Chylo-hæmothorax,** 279
**Chylothorax,** 279, 555
**Chylous** ascites, 555
hydrocele, 555
pleurisy, 555
**Chyluria,** 555
**Circulation,** conditions of, in pyrexia, 20
diseases of, 284
in psychopathology, 891
**Circum-oral** pallor in scarlet fever, 29
**Cirrhosis of liver,** 483
atrophic, 485
biliary, 487
obstructive form of, 487
carcinomatosa, 485, 491
causing adhesive pylephlebitis, 502
jaundice, 476, 679
clubbing of fingers in, 673
differentiation from carcinoma, 492
enlargement of spleen in, 550
epistaxis in, 198
hydrothorax in, 278
hypertrophic, 485
biliary, 487
in *Bilharzia japonicum* infection, 650
in chronic alcoholism, 679
in Kala-azar, 161
in mixed infections, 154
in progressive lenticular degeneration, 840
in splenic anæmia, 529
jaundice from, differentiation from catarrhal jaundice, 481
multilobular, alcoholic, 483, 484
in syphilis, 490
pericellular, in congenital syphilis, 490
portal, 483
purpura in, 542
resembling chronic peritonitis, 513
splenomegalic, 487
unilobular, 487
**Cladothrix,** 10
**Claude and Gougerot,** pluri-glandular insufficiency of, 601
**Claudication,** intermittent, 382
erythromelalgia in, 383
in arterio-sclerosis, 368
in thrombo-angiitis obliterans, 365
**Climatic** treatment in phthisis, 260
**Clonus,** ankle, in hemiplegia, 818
types of, 735
**Clothing,** disinfection of, 13
**Clubbing** of fingers, causes of, 672
in chronic bronchitis, 219
in chronic pneumonia, 246
in congenital heart disease, 355
in empyema, 276
in endocarditis lenta, 333
in phthisis, 254
in whooping-cough, 49
**Coagulation** of blood in aneurysm, 372
in thrombosis, 379
**Coarctation** of aorta, congenital, 377
**Cocci,** infection by, 9
**Cochlea,** disease of, causing tinnitus, 750
**Cod-liver** oil in hunger osteomalacia, 695
in rickets, 695
**Cœliac** disease, 448
infantilism, 448
steatorrhœa in, 504
**Coffee** grounds vomit, 406
in carcinoma of stomach, 432
in gastric ulcer, 424
**Cog-wheel** breathing, 193
**Cold** bath, 25
**Colic,** biliary, in gall stones, 499
intestinal, 441
differentiation from biliary colic, 500
lead, 441, 680
treatment of, 682
mucous, 449
renal, differentiation from biliary colic, 500
intestinal obstruction, 463
in hydatid disease, 649
renal calculus, 657
treatment of, 660
simulating peritonitis, 511
**Colitis,** bacilluria associated with, 634
catarrhal, 449
following amœbic dysentery, 168
muco-membranous, 449
mucous, 449
ulcerative, 450
diarrhœa in, 440
in chronic parenchymatous nephritis, 626
**Collapse** in cholera, 180
in melioidosis, 137
**Colles'** law, 120
**Collodion,** non-flexible, in treatment of erysipelas, 54
**Colloidal** benzoin test in encephalitis lethargica, 833
reactions in cerebro-spinal fluid, 738
silicic acid, tubercle and, 126
**Colon,** adeno-papilloma of, 456
*bacillus. See Bacillus Coli.*
carcinoma of, differentiation from cancer of kidney, 653
differentiation from cholecystitis, 497
dilatation and hypertrophy of, in Hirschsprung's disease, 466
"greedy," 435
growth in, differential diagnosis of gall stones from, 500
injections in dysentery, 166
pelvic, carcinoma of, 456
polypoid growths of, 457
ptosis of, 416
tumour of, causing jaundice, 476
ulceration of, amœbic, 166
**Colony,** tuberculosis, 260
**Coloptosis,** 416
**Colostomy** for stricture of colon, 463
**Colour** index of blood, 520
in congenital acholuric jaundice, 531
in erythræmia, 538
in lead poisoning, 681
in myelocytic leukæmia, 533
in pernicious anæmia, 528
in scurvy, 689
in splenic anæmia, 529

**Coma,** diabetic, 570, 827
blood transfusion in, 546
differentiation from intestinal obstruction, 463
followed by hypoglycæmic coma, 575
perforative peritonitis simulating, 511
treatment of, 575
from alcoholic poisoning, 826
from asthenic uræmia, 827
from high concentration of aceto-acetic acid in blood, 568
from injury to brain, 826
from opium poisoning, 826
hypoglycæmic, rapidly following diabetic coma, 575
symptoms of, 575
in acute inadequacy of suprarenals, 589
in acute yellow atrophy of liver, 482
in anthrax, 139
in arseno-benzol poisoning, 123
in cerebral abscess, 837
in cerebral embolism, 826
in cerebral hæmorrhage, 823
in confluent small-pox, 41
in cyclical vomiting, 418
in enteric fever, 84
in glanders, 137
in heat stroke, 27
in hydrophobia, 103
in lead poisoning, 681
in malignant endocarditis, 332
in malignant tertian infection, 153
in pachymeningitis interna hæmorrhagica, 848
in pyrexia, 20
in scarlet fever, 30
in suppurative meningitis, 844
in suppurative nephritis, 638
in tuberculous meningitis, 130
in yellow fever, 177
uræmic, in acute nephritis, 624
**Comedo,** 935
*Comma bacillus*, 179
**Compensation** of heart, 336
effects of failure upon other organs, 337
**Complement,** 17
deviation in hydatid disease of liver, 495
fixation test in diagnosis of tuberculosis, 258
*See also* Wassermann reaction.
**Compression** of spinal cord *See under* Spinal cord
**Conditioned** reflex, 892
reflexes in anxiety neurosis, 894
**Condyloma,** vel verruca acuminata, 962
**Confluent** small-pox, 41
**Congenital** heart disease, cyanosis in, 354
differentiation from bronchiectasis, 224
hydronephrosis, 643
laryngeal stridor, 212
malformations of heart, 353
obstruction of bile duct in new-born, 477
pulmonary stenosis and deficiency of interventricular septum, 353
**Conjugate** deviation, 747
in hemiplegia, 819
**Conjunctivitis** in gonorrhœal rheumatism, 73
in hay fever, 227
latent, in latent tetany, 586
**Constipation,** 434
after gastro-jejunostomy, 428
chronic, 438
in anxiety neurosis, 896
in appendicitis, 453
in carcinoma of intestine, 456
in chlorosis, 525
in diabetes insipidus, 596
in gastroptosis, 416
in intestinal obstruction, 460, 461
in jaundice, 474
peritoneal adhesions and, 516
**Consumption,** 248
*See also* Phthisis *and* Tuberculosis.
**Contacts** of cerebro-spinal fever cases, treatment of, 97
**Convulsions,** epileptiform, in arseno-benzol poisoning 123
in malaria, 153
hysterical, 904
diagnosis of, 907
infantile, cause of, 875
in acute inadequacy of suprarenals, 589
in cerebral tumours, 853
in children in lobar pneumonia, 239
**Convulsions** in congenital heart disease, 355
in cyclical vomiting, 418
in lead poisoning, 681
in measles, 34
in myocardial degeneration, 324
in rickets, 693
in suppurative meningitis, 844
tetany associated with, 586
uræmic, in acute nephritis 621
treatment of, 623
**Copaiba** causing skin eruptions, 980
**Cor bovinum,** 319
**Cornea,** inoculation on, in vaccinia, 44
ulceration of, in frontal herpes, 999
**Corneal** reflex, diminution or loss of, in fifth nerve lesions, 748
**Corns,** 1005
**Cornu** cutaneum, 1006
**Coronary** arteries, obstruction of, fatty degeneration of heart in, 323
fibrosis of heart due to, 324
sclerosis of, in myocardial degeneration, 325
dilatation of heart in, 320
**Corpora** quadrigemina, localisation of function in, 815
**Corpus** striatum affected in chronic manganese poisoning, 684
lesions of, 813
**Corpuscles,** enumeration of, 519
fragility of, 519
microscopic examination of, 520
red, 520
white, 521
**Coryza,** 196
**Cough,** auscultation of, 196
in empyema, 276
in tuberculosis of bronchial glands, 385
persistent, following gassing, 265
**Counter-irritants** in treatment of chronic bronchitis, 220
**Cow-pox,** inoculation of, 42
**Crab** louse infection, 965
**Cracked** pot sound in chest percussion, 192
in phthisis, 253
**Cramp,** 924
in alcoholic neuritis, 741
in neuritis, 739
writer's, 883
**Cranial** nerves, examination of, 726
disorders of, in hysteria, 905
lesions of, 743
**Craniotabes** in rickets, 693
**Creatorrhœa** in pancreatic disease, 504
**Crepitation,** 195
in mitral disease, 342
redux, in pneumonia, 238, 240
**Cretinism,** 583
associated with goitre, 576
endemic, 583
infantilism associated with, 600
sporadic, 583
**"Crick"** of the neck, 754
**Crises,** gastric, in tabes, 789, 790
*See also* Tabes.
**Crisis,** hæmoclasic, 17
in lobar pneumonia, 240
in pyrexia, 22
laryngeal, spasm of glottis in, 211
**Crus** cerebri, symptoms of lesions of, 815
**Cryptorchidism,** 598
**Crystals,** cholesterin, in gall-bladder disease, 500
*Cubex fatigans*, 554
**Curschmann's** spirals, 222, 227
**Cutaneous** reaction, Von Pirquet's, 257
**Cyanosis,** enterogenous, 544
heliotrope, in pneumonic influenza, 62
in congenital heart disease, 354
in erythræmia, 538
in heat stroke, 27
in lobar pneumonia, 240
in œdema of lungs, 236
in pulmonary embolism, 270
venesection for, 348
**Cyclical** vomiting, 418
relation to migraine, 876
**Cylindroids** in urine, 618
**Cyst** *or* **Cysts,**
in brain, 850
in chronic interstitial nephritis, 628
in hydronephrosis, 644
in kidney, 653, 654

**Cyst** *or* **Cysts** in pancreas, retention, 508
in primary contracted kidney, 631
of stomach, 434
sebaceous, 1014
**Cystic** disease of kidney, 492, 653
*Cysticercus telæ cellulosæ*, 467
**Cysticercus** of heart, 328
**Cystitis,** bacilluria in, 635
complicating bilharziasis, 650
differentiation from renal calculus, 659
in acute myelitis, 774
pyelitis associated with, 635
**Cystoscope,** in diagnosis of pyelitis, 637
tuberculous kidney, 642
in examination of kidneys, 603
**Cytase,** 17
**Cytolysis,** 16

**Dandruff,** 934
**"Dangles,"** the, in lead paralysis, 681
**Darier's** disease, 1006
**Deafness,** hysterical, 905
quinine treatment causing, 156
**Death** in fevers, 22
**Decompression,** operation of, in cerebral tumours, 858
in hydrocephalus, 861
**Decortication** of lung for empyema, 278
**Defervescence** in pyrexia, 22
**Defibrinated** blood, prophylactic inoculation of, in typhus fever, 79
**Degeneration** of heart, fatty, 323
fibroid, 324
chronic aneurysm in, 326
myocardial, 323
pigmentary, 323
simple paroxysmal tachycardia in, 311
of posterior nerve roots in diabetes, 569
of spinal cord, subacute, combined with pernicious anæmia, 527
subacute, differentiation from compression, 770
from multiple neuritis, 742
reaction of, 733
*See also under* Reaction of degeneration.
**Delirium** in lobar pneumonia, 240, 242
tremens, 677
**Deltoid** muscle, paralysis of, 756
**Dementia** following epilepsy, 877
in chronic alcoholism, 679
in pellagra, 696
paralytica, 861
præcox, differentiation from anxiety neurosis, 897
histological changes in suprarenal capsules, 589
**Dengue,** 178
differentiation from sand-fly fever, 148
**Dental** caries, vitamins in diet in relation to, 686
**Depression** in gastroptosis, 447
in masturbation, 900
**Dercum's** disease, 595, 599
**Dermatitis,** exfoliative, 992
exfoliativa neonatorum, 927
exfoliative, differentiation from small-pox, 42
gangrenosa infantum, 929
herpetiformis, 1005
differentiation from pemphigus vulgaris, 1003
in arseno-benzol poisoning, 123
infectiosa eczematoides, 944, 983, 987
papillaris capillitii, 942
seborrhœic, 939
streptococcal, 983, 987
traumatic and solar, 981
uræmic, 979
venenata, 979
**Dermatolysis,** 1017
**Dermatoses,** 923
**Dermographism,** 977
**Dermoid** cysts, mediastinal, 386
**Desensitisation,** 18
**Desquamation** in dengue, 178
in erysipelas, 54
in measles, 34
in rubella, 37
in scarlet fever, 29
**Deviation** of complement in hydatid disease of liver, 495
**Dhobie** itch, 957

**Diabète bronzé,** cirrhosis of liver in, 484
**Diabetes,** coma of, 570, 827
experimental, 564
innocens, 570
insipidus, 596
disease of pituitary gland as cause of, 593, 596
infantilism associated with, 600
mellitus, 562
and Graves' disease, association of, 580
arterio-sclerosis associated with, 367
boils in, 943
causing multiple neuritis, 740
chronic pancreatitis causing, 507
differentiation from diabetes insipidus, 597
pathology of, 564
pruritus vulvæ in, 997
"puncture," 562
supervening in diabetes insipidus, 597
transient, complicating mumps, 47
xanthoma in, 1018
*See also under* Glycosuria.
**Diabetic** coma, 570, 827
blood transfusion in, 546
differentiation from intestinal obstruction, 463
followed by hypoglycæmic coma, 575
neuritis, 742
perforative peritonitis simulating, 544
**Diacetic** acid, 568
**Diadochokinesis** test in tumours of cerebellum, 855
**Diaphoresis** in treatment of chronic parenchymatous nephritis, 626
**Diaphragm,** paralysis of, 235, 755
**Diaphragmatic** hernia, 282
differentiation from pneumothorax, 281
**Diarrhœa,** 139
after gastro-jejunostomy, 428
bilious, 140
choleraic, 180, 440
colliquative, 140
complicating relapsing fever, 147
critical, 140
differentiation from cholera, 181
dysenteric, 140
gastrogenous, 139
in anxiety neurosis, 896
in bilharziasis, 650
in cholera, 180
in congenital pyloric stenosis, 430
in exophthalmic goitre, 580
in hunger œdema, 697
in hypoglycæmia from insulin, 572
in malignant endocarditis, 342
in melioidosis, 137
in phthisis, 255
treatment of, 264
in sprue, 448
in syphilis, 1044
in trichinosis, 471
in typhoid, epidemic, 441
intestinal, 140
predisposing to tetany, 584
reflex, 140
spurious, 440
in carcinoma of intestine, 456
summer, 444
**Diarsenol,** 116, 118
**Diastase** in urine, estimation of, 610
in acute nephritis, 620
in chronic interstitial nephritis, 628
in pancreatic disease, 504
**Diastolic** blood pressure, 300
murmurs, 289
*See also under* Murmurs.
shock in aortic aneurysm, 374
**Diathermy** in treatment, 6
of fibrositis, 911
of lupus vulgaris, 950
of neuritis, 740
**Diazo** reaction in Van den Bergh's test in jaundice, 475
*Dibothriocephalus latus*, 468
**Dichlorethyl** sulphide, poisoning by, 265
**Dick** test, 32
**Dicrotic** notch, 299
pulse in enteric fever, 82
wave, 299
*See also* Pulse, dicrotic.
**Diet,** carbohydrate, in syphilitic disease of liver, 490
**Diet** containing purins, 664
deficiencies in, diseases due to, 685
effect of excess of cereals in producing rickets, 691

**Diet** in chronic dyspepsia, 413
in constipation, 437
in diabetes, 573
in eczema, 986
in prevention of beri-beri, 688
of scurvy, 690
in pyrexia, 24
in treatment, 5
of acne vulgaris, 937
of infants in enteritis, 445
protein, high, causing albuminuria, 661
vegetable, in psoriasis, 992
vitamins in, 5
in relation to dental caries, 686
**Dietl's** crises, 655
**Differential** blood count, 521
**Diffuse** hyperplastic sclerosis, 366, 369
blood pressure in, 367
**Digestion**, organs of, diseases of, 389
**Digitalin** in auricular fibrillation, Eggleston's method of massive doses, 316
**" Digitalis** coupling," 316
**Digitalis** in auricular fibrillation, 315
in auricular flutter, 313
in heart block, 307
in heart disease, 348
in pulsus alternans, 313
in simple paroxysmal tachycardia, 311
treatment with, heart block after, 304
use previous to quinidine, 317
**Digitoxin** in heart disease, 349
**Dilatation** of auricles, 321
of heart, 320
acute, 321
compensatory, 320
diagnosis of, 322
differentiation from pericarditis, 358
during and after muscular exercise, 321
in gradual onset of cardiac failure, 320
physical signs, 321
X-ray appearances, 321
of left ventricle, primary, in aortic regurgitation, 340
in mitral regurgitation, 341
**Diphenylchlorarsine**, poisoning by, 265
**Diphtheria**, 66
acute endocarditis in, 328
acute rhinitis in, 197
anæsthesia of larynx in, 212
and lesions of phrenic nerve, 755
bronchitis in, 215
carriers, 66
differentiation from acute laryngitis, 200
follicular tonsillitis, 396
plague, 185
scarlet fever, 31
fatty degeneration of heart in, 323
faucial, 67, 69
laryngeal, 68
membranous laryngitis in, 201
method of spread of, 66
nasal, 68
of trachea, 213
paralysis of external tensors of vocal cords in, 210
polyneuritis in, 740
quarantine period in, 13
Schick test, 70
**Diphtherial paralysis**, 68
differentiation from myasthenia gravis, 920
**Diplococci** in acute endocarditis, 329
in pericarditis, 356
in rheumatic fever, 55
***Diplococcus intracellularis***, 93
***lanceolatus***, 72
***pneumoniæ***, 72, 238
**Diplopia**, 727
in botulism, 448
in disseminated sclerosis, 782
in encephalitis lethargica, 832
in syringomyelia, 786
in tuberculous meningitis, 130
**Disease**, fears of, in anxiety neurosis, 895, 900
nature of, etc., 1–8
treatment of, 4
**Disinfection** in infectious diseases, 13
of patient, 14
of room, 14
**Displacement** of liver, detection of, in ascites, 478
**Disseminated** sclerosis, 780
associated with vertigo, 751
differentiation from chronic mercurial poisoning, 683
combined subacute degeneration, 797
compression of cord, 770
Friedreich's ataxia, 798
hysteria, 907
paralysis agitans, 842
tumour of cerebellum, 857
**Dissociation** of auricle and ventricle rhythms in heart block, 305
*See also* Heart block.
of sensations in hæmorrhage into spinal cord, 777
**Dittrich's** plugs, 221
**Divers'** paralysis, 778
**Diverticulitis**, 457
**Döhle's** bodies, 29, 32
**Douches** in treatment, 7
***Dracunculus medinensis***, 557
**Dreyer's** formula for basal metabolism, 560, 561
**Dropsy.** *See* Œdema.
**Drugs**, antipyretic, 25
applied externally, toxic skin eruptions caused by, 980
ingestion of, toxic skin eruptions caused by, 979
in treatment of dyspepsia, 414
**Dry-cupping** in acute nephritis, 623
**Du Bois** standards of normal metabolism, 561
**Duchenne-Aran** type of muscular atrophy, 799
**Ductus** arteriosus, patent, 353, 354
**Dulness**, deep or relative, 286
præcordial, 286
superficial or absolute, 286
**Duodenal** contents, analysis of, in dyspepsia, 413
**" Duodenal hurry,"** 408
**Duodenal** ileus, 419
tube, Einhorn's, 500, 501
tube for administering large amounts of fluid in cyclical vomiting, 418
in hysterical vomiting, 418
in Raynaud's disease, 382. *See also* Nasal tube.
**Duodenum**, carcinoma of, 456
dilatation of, acute, 419
ulcer of, 421
recurrent, 427
differentiation from appendicitis, 454
chronic gastric ulcer, 425
**Dwarfism**, 595
**Dysarthria**, 723, 724
differentiation from aphasia, 820
**Dyschezia**, 435, 735
**Dysenteric** ulceration and amœbic abscess, 170
**Dysentery**, 162
amœbic, 166
colitis following, 166, 168
asylum, 450
bacillary, 163
differentiation from cholera, 181
chronic, 164
complicating relapsing fever, 147
diarrhœa in, 440
**Dysidrosis**, 1001
**Dyspepsia**, 408, 409
acid, 412
after gastro-jejunostomy, 427
appendix, 454
atonic, 411, 412
chronic, 409
differentiation from chronic gastric ulcer, 425
flatulent, 415
gall-stone, 498
in anxiety neurosis, 896, 901
in chronic alcoholism, 679
in pellagra, 696
nervous, 412
selective, 901
*sine materia*, 412
types of pain in, 410
**Dyspeptic** acrophagy, 415
symptom complex, spasmodic stricture œsophagus in, 402
symptoms in achylia gastrica, 419
in acute dilatation of duodenum, 419
**Dysphagia, hysterical**, 906
in achalasia, 403
in anæsthesia of larynx, 212
in anxiety neurosis, 896
in aortic aneurysm, 374

**Dysphagia** in carcinoma of œsophagus, 401
in goitre, 577
in œdematous laryngitis, 201
in tenth nerve lesions, 752
**Dyspituitarism**, 593
**Dyspnœa**, 188
in Adams-Stokes syndrome, 307
in adherent pericardium, 359
in anxiety neurosis, 896
in aortic aneurysm, 375
in cardiac disease, differentiation from asthma, 228
in compression of trachea, 214
in emphysema, 233
in empyema, 276
in goitre, 577
in mediastinitis, 384
in mitral disease, 342
in myocardial degeneration, 325
in obstruction of lung, 230
in œdematous laryngitis, 201
in paralysis of abductors of vocal cords, 208
in pulmonary embolism, 270
obstructive, 188

**Eberth's** bacillus, 80
**Ecchymoses**, 924
subconjunctival, in whooping cough, 48
**Echinococcus**. *See* Hydatid.
*Echinococcus granulosus*, 493
**Eclampsia**, acidæmia in, 548
**Ectasis**, alveolar, 231
**Ecthyma**, 928
**Ectoscopy**, 188
**Eczema**, 981
acute, differentiation from facial erysipelas, 54
chronic, differentiation from small-pox, 42
differentiation from cheiropompholyx, 1002
eczematoid ringworm, 958
lupus erythematosus, 971, 972
psoriasis, 991
erythemato-squamous, eczematoid ringworm resembling, 958
intertrigo, 929
differentiation from congenital syphilis, 121
eczematoid ringworm, 958
of internal origin, 984
pediculis capitis and, 965
pustular, differentiation from small-pox, 42
scrofulosorum, 953
seborrhœic, 984
differentiation from psoriasis, 991
**Eczematoid** ringworm of extremities, 958
**Effleurage** in massage, 7
**Effort**, response of heart to, 294
syndrome, ætiology of, 350
following "gassing," 265
physical signs, prevention and prognosis of, 352
symptoms of, 351
treatment of, 352
**Effusion**, blood-stained, in tuberculous pleurisy, 276
chyliform, 279
during treatment by artificial pneumothorax, 263
pericardial, 360
simulating pleurisy, 275
pleural, differentiation from chronic pneumonia, 246
empyema following, 272
**Egg** yolk in rickets, 694
**Eggleston's** treatment with massive doses of digitalin in auricular fibrillation, 316
**Ehrlich's** "606," 116, 118
*See also* Arsenobenzol.
**Einhorn's** duodenal tube, 500, 501
in treatment of diabetic coma, 576
**Ejaculation**, premature, in anxiety neurosis, 895
"**Elective** localisation" in peptic ulcer, 424
**Electrical** reactions of muscles, 732
treatment in lead palsy, 683
**Electricity** in treatment, 6
in acroparæsthesia, 383
in erythromelalgia, 383
in intermittent claudication, 383
in Raynaud's disease, 382
**Electro-cardiograph**, 303
in diagnosis of valvular heart disease, 346
hypertrophy of heart as shown by, 318
*See also* Heart, abnormalities of action of.

**Elephantiasis**, 555, 556
**Embolism**, 378
cerebral, 837
complicating failure of heart, 338
fat, of pulmonary capillaries, 270
in acute endocarditis, 329
in endocarditis lenta, 333
in kidneys, 380
in limbs, 380
in malignant endocarditis, 331
in spleen, 380
of cerebral arteries, 825
in mitral stenosis, 343
of mesenteric artery, 380
of new growth, 381
of pulmonary artery, 379
pulmonary, 269, 380
septic, 379
**Embolus** as cause of coronary aneurysm, 326
**Embryocardia**, 288
**Emetine**, bismuth iodide in treatment of amœbic dysentery, 168
subcutaneous injection of, in amœbic dysentery, 168
in amœbic infection of liver, 171
**Emotion**, causing gastroptosis, 405
causing reflex diarrhœa, 440
**Emotional** reaction, 891
**Emphysema**, 222, 231
compensatory, 232
in pulmonary tuberculosis, 250
differentiation from pneumothorax, 281
erythrocytosis and, 539
following mustard gas poisoning, 266
in chronic bronchitis, 219
interstitial, 231
pneumothorax following, 279
small-lunged, 232
surgical, 231
tricuspid regurgitation in, 343
vesicular, 216, 231
**Empyema**, 270, 272
and lardaceous disease of kidney, 646
causing bronchiectasis, 222
causing peritonitis, 509
cerebral abscess in, 837
differentiation from bronchiectasis, 224
phthisis, 259
tuberculosis of bronchial glands, 385
evacuation of, in children, 277
gangrene of lung following, 247
in hydatid disease of lung, 268
in lobar pneumonia, 241
in perinephric abscess, 640
in pulmonary tuberculosis, 250
of gall bladder, 496
pulsating, 274
resulting from foreign body in bronchi, 230
**Encephalitis**, acute, 829, 838
acute bulbar paralysis and, 811
chronic, 836
hæmorrhagic, in arseno-benzol poisoning, 123
lethargica, 831
causing Argyll-Robertson pupils, 726
chorea in, 864
differentiation from cerebral tumour, 856
disseminated sclerosis, 783
tuberculous meningitis, 132
experimental infection of animals with, 183
ophthalmoplegia in, 746
prevention of, 835, 836
spasmodic torticollis following, 882
syphilitica, 861
virus of, cause of paralysis agitans, 841
**Encephalopathy**, saturnine, 681
**Endarteritis**, 365
in malignant endocarditis, 331
syphilitic, 113
*See also* Syphilis.
**Endocarditis**, acute, 328
bacterial, subacute, 333
chronic, 334
differentiation from enteric fever, 88
gonococcal, 73
infective, clubbing of fingers in, 673
lenta, 333
right-sided, 329
in rheumatic fever, 57
**malignant**, 330

**Endocarditis,** malignant, aneurysm in, 326, 331
blood culture in, 331
blood transfusion in, 546
complicating failure of heart, 338
differentiation from malaria, 154
pyæmia, 52
splenic anæmia, 530
enlargement of spleen in, 530
in pneumonia, 241
myocarditis in, 322
pyæmia from, 51
pneumococcal, combined with pneumococcal meningitis, 332
purpura in, 542
**Endocrine** glands in psychopathology, 891
organs, diseases of, 558
**Endocrinopathic** syndromes, varied, 598
**Endothelioma** of brain, 850
**Endotoxins,** 16
**Enema,** opaque, 391
in diagnosis of carcinoma of intestine, 457
**Enemas** in constipation, 437
**Enophthalmos,** 720
*Entamœba coli,* 167
*histolytica,* 10, 166, 167, 168
*minuta,* 166
**Enteric** fevers, 79, 80
acute endocarditis in, 328
bronchitis in, 215
diarrhœa in, 440
diet in, 91
differentiation from acute gastritis, 421
food poisoning, 448
glanders, 137
influenza, 63
malaria, 155
Mediterranean fever, 173
miliary tuberculosis, 132
plague, 185
pneumonia, 241
pyæmia, 52
relapsing fever, 147
spirochætosis, 175
suppurative nephritis, 638
trench fever, 65
trichinasis, 913
typhus, 78
enlargement of spleen in, 550
epistaxis in, 199
fatty degeneration of heart in, 323
œdematous laryngitis in, 200
onset of peritonitis in, 511
pleurisy simulating, 275
resemblance of malignant endocarditis to, 332, 333
ulcer, perforation of, 84, 85, 91
ulcers of ileum causing acute peritonitis, 508
**Enteritis,** 442
catarrhal, 442
diarrhœa in, 440
follicular, 442
infantile, 444
phlegmonous, 449
**Enterogenous** cyanosis, 541
**Enterospasm,** 436
**Eosinophil** cells, 521
**Eosinophilia,** 523, 537
in differentiation of hydatid disease, 268, 495
in filariasis, 554
in myelocytic leukæmia, 535
in trichinasis, 913
**Ephelis,** 1010
ab igne, 973
**Epidemic** blackwater fever, 157
cerebro spinal meningitis, 93
dysentery, 162
encephalitis, 831
influenza, 61
poliomyelitis, 97
**Epidermolysis** bullosa, 1004
differentiation from pemphigus vulgaris, 1003
*Epidermophyton inguinale,* 957, 959
**Epididymis,** tuberculosis of, in phthisis, 255
**Epididymitis,** 73
in Mediterranean fever, 173
**Epilation** in treatment of ringworm, 959
**Epilepsy,** 827, 868
association with hypopituitarism, 595
differentiation from cerebral tumour, 857
**Epilepsy** in adults, resemblance of convulsions in children to, 875
Jacksonian, 872
in cerebral tumours, 854
major, 869
minor, 870
post-epileptic conditions, 870
spasm of glottis in, 211
status epilepticus, 871
tetany in, 585
treatment in epileptic colonies, 873
**Epileptic** fit, differentiation from hysterical fit, 907
**Epileptic** fits in chronic alcoholism, 679
**Epileptiform** or apoplectic attacks in general paralysis of insane, 862
convulsions in arseno-benzol poisoning, 123
in malaria, 153
**Epinephrin,** 589
**Epiphysitis** in congenital syphilis, 121
syphilitic, differentiation from rickets, 694
**Epistaxis,** 198
in enteric fever, 82
in malignant endocarditis, 333
in scurvy, 689
**Epizootics** of plague among rats, 183, 185
**Erb's** paralysis, 758
syphilitic paraplegia, 113, 775
**Ergotism,** tetany in, 585
**Erosions,** hæmorrhagic, in acute peptic ulcer, 422
**Eructation** in angina, 363
**Eruption.** *See* Rash
**Erysipelas,** 52
complicating relapsing fever, 147
delirium tremens and, 677
differentiation from frontal herpes, 1000
gonococcal infections, 74
following vaccination, 43
**Erythema** ab igne, 973
differentiation from facial erysipelas, 54
exudativum in rat-bite fever, 144
induratum, 953
infantile, of Jacquet, 930
in anthrax, 139
in hay fever, 227
in pellagra, 696
in small-pox, 39
in spirochætosis, 175
intertrigo, 929
associated with thrush, 394
multiforme, 968
differentiation from lupus erythematosus, 971, 972
pemphigus vulgaris, 1003
in rheumatic fever, 57
nodosum, 953, 969
in rheumatic fever, 57
papular, differentiation from small-pox, 42
pernio, 970
differentiation from lupus erythematosus, 972
**Erythemas,** the, 967
**Erythemato-squamous** skin eruptions, 988
**Erythematous** eruption in arseno-benzol poisoning, 123
**Erythræmia,** 522, 538
enlargement of spleen in, 550
**Erythrasma,** 961
**Erythroblasts,** 520
**Erythrocytes,** 520
**Erythrocytosis,** 522, 539
**Erythrodermia,** in leukæmia, 994
pruriginosa, 993
**Erythrodermias,** the, 992
**Erythromelalgia,** 783
in thrombo-angiitis obliterans, 365
**Esbach's** test, 614
**Euglobulin** in urine, 614, 616
in functional albuminuria, 661, 662
**Eunuchism,** 598
**Eunuchoidism,** 598
**Ewald's** test meal, 406
**"Exclamation** mark" hairs in alopecia, 1015
**Excreta,** disinfection of, 13
**Exercise,** graduated, in treatment of phthisis, 262
in obesity, 599
lack of, in rickets, 691
muscular, effect on digestion, 408
response of heart to, 294
to, in angina, 364
**Exercises** in constipation, 437

**Exfoliative** dermatitis, differentiation from small-pox, 42
**Exhaustion,** feeling of, in mitral disease, 341
**Exocardial** murmurs, 293 *See also* under Murmurs.
**Exophthalmic** goitre, 578
*See also under* Goitre, exophthalmic
**Exophthalmos** due to pressure from pituitary tumours, 594
in acute leukæmia, 536
in goitre, 580
**Exotoxins,** 16
**Expectorants** in treatment of chronic bronchitis, 220
**External** tensors of vocal cords, paralysis of, 210
**Extra** systoles, 307. *See also* Systoles, extra
**Eye,** error of refraction of, alopecia areata and, 1016
**Eyelids,** secondary vaccinia on, 44
**Eyes,** examination of, 726
soreness of, following gassing, 265

**Facultative** parasite, 10
**Fæces,** accumulated, differential diagnosis of cancer of kidney from, 653
blood in, in bilharziasis, 650
excess of fat in *See* Steatorrhœa
fat in, in pancreatic disease, 503
incontinence of, in pyrexia, 21
intestinal obstruction due to, 458, 460
lead in, tests for, 682
occult blood in, in carcinoma of stomach, 432
in duodenal ulcer, 425
in gastric ulcer, 425
tests for, 408
transmission of typhoid fever by, 80
undigested striped muscle fibres in, in pancreatic disease, 504
**Fainting** attacks, gastroptosis in, 405
**Family** periodic paralysis, 919
**Faradism** in treatment, 6
susceptibility to, in tetany, 586
*See also* Reaction of degeneration
**Farcy,** 136
**Fastigium,** 22
**Fasting** in treatment of diabetes, 571
obesity, 600
**Fat** embolism, 270
necrosis in acute pancreatitis, 505
**Fatigue,** extreme liability to, in anxiety neurosis, 895
**Fat-soluble** A vitamin, 685
in rickets, 691
**Fatty** degeneration in phthisis, 255
**Favus,** 956
**Feet,** eczematoid ringworm of, 958
**Fehling's** test for sugar, 565
**Femoral** thrombosis in phthisis, 255
causing pulmonary embolism, 269
**Fermentation** test for sugar, 567
**Ferric** chloride test for ketosis, 568
**Fever.** *See* Pyrexia.
**Fibrillation,** auricular, 302, 313
ventricular, 318
**Fibroid.** *See under* Heart, Lung *and* Phthisis
**Fibro-lipoma** of stomach, 434
**Fibroma,** intestinal obstruction caused by, 458
molluscum, 1017
of larynx, 206
of skin, multiple, 1017
**Fibro-myositis,** nodular, 912
**Fibrositis,** 910
in gonorrhœal rheumatism, 73
**Fidgets,** 924
*Filaria bancrofti,* 554, 555
*loa,* 557
*perstans,* 557
**Filariasis,** 554
**Filatow's** spots, 34
**Filter-passing** organisms, cause of common cold, 196
cause of influenza, 61
diseases caused by, 9
diseases of skin due to, 962
**Fingers,** clubbing of *See under* Clubbing
**Finsen** light treatment of lupus vulgaris, 950
**Fission** fungi, infection by, 9
**Fistula,** gastro-colic, in carcinoma, 431
**Fistula in ano,** association with phthisis, 255
tuberculous origin of, 456
**Fit,** epileptic, differentiation from hysterical fit, 907
hysterical, diagnosis of, 907
**Flagellation** in massage, 7
**Flatulence** after gastro-jejunostomy, 427
in angina, 363
in anxiety neurosis, 901
in dyspepsia, 412
in gastroptosis, 417
*See also* Aerophagy
**Flea** bites, differentiation from purpura, 544
**Fleas** from rats, conveyance of infection of bubonic plague by, 183
**Fleas,** transmission of infection by, 12
**Flies,** contamination of food by, causing enteric fever, 81
infecting agent in dysentery, 162
means of spread of cholera, 179
transmission of infection by, 12
**Flint's** murmur in aortic regurgitation, 293, 340, 346
**Floating** kidney, 654
**Fluctuation** in diagnosis of ascites, 478
**Fluids** in diet in dyspepsia, 414
**Flutter,** auricular, 312
**Fœtal** rhythm, 288
**Fœtus,** syphilitic, death of, 121
**Folliclis,** 953
**Folliculitis,** 941
decalvans, differentiation from alopecia areata, 1016
**Food,** contamination of, by flies, cause of enteric fever, 81
infant feeding, 445
poisoning, 447
differentiation from cholera, 181
**Foot-and-mouth** disease, 139
**Foot-drop** in peroneal muscular atrophy, 798
**Foramen** ovale, patent, 354
**Foreign** bodies causing jaundice, 476
causing obstruction of bronchi, 230
in bronchus, abscess of lung from, 245
causing bronchiectasis, 222
gangrene of lung following, 247
in larynx, 207
in trachea, 214
intestinal obstruction due to, 458
presence of, simulating laryngismus stridulus, 211
spasm of glottis, in, 211
**Fortification** spectrum in migraine, 878
**Fractures,** green-stick, in rickets, 694
**Fraenkel's** movements in treatment, 7
**Fragility** of red corpuscles, 549
*See also* Blood.
**Framboesia,** 124
**Freckles,** 1010
**Friction** in massage, 7
pleuro-pericardial, 275
sound, 195
in pericarditis with rheumatic fever, 57
in pleurisy, 273
over heart, 294
**Friedreich's** ataxia, 797
differentiation from chorea, 867
combined subacute degeneration, 797
peroneal muscular atrophy, 799
**Frohlich's** syndrome, 595
infantilism associated with, 600
**From's** syndrome, 737
**Frontal** lobe, localisation of function in, 814
**Fulness,** sensation of, in dyspepsia, pathology of, 410
**Functional** diseases, 1
*See also* Psycho-neuroses.
**Fundus** oculi, examination of, 725
pigmentation in congenital syphilis, 122
*See also* Ophthalmoscopic examination.
**Fungi,** higher, diseases of skin due to infection with, 955
**Funiculitis,** filarial, 555
**Funnel-shaped** orifice of mitral valve, 335
**Furunculosis,** 943

**Gaertner's** bacillus in food-poisoning, 447
**Gait,** examination of, 736
in hysteria, 906
in paralysis agitans, 842
staggering, in tumours of cerebellum, 855
"steppage," in polyneuritis, 740
**Galabin's** sign in heart block, 305
**Gall** bladder, cancer of, differentiation from aneurysm, 377
due to gall stones, 500
production by foreign bodies, 500

**Gall** bladder, distension of, 473
differentiation from movable kidney, 655
from impaction of gall stones, 499
empyema of, 496
fibrosis of, 500
obliteration of cavity of, by gall stones, 499
strawberry, 496
suppuration of, causing acute peritonitis, 508
drainage of, 501
" medical," 497
stone dyspepsia, 498
stones, 497
acute pancreatitis with, 505
cause of cholecystitis, 498
chronic pancreatitis, 506
jaundice, 476
suppurative cholangitis, 495
suppurative pylephlebitis, 502
differentiation from chronic gastric ulcer, 425
dyspepsia in, 411
impaction of, in common bile duct, 499
intestinal obstruction due to, 461
jaundice from, differentiation from catarrhal jaundice, 481
secondary to carcinoma, 500
**Galvanism**, susceptibility to, in tetany, 586
**Gametogony**, 150
**Gangrene**, arterio-sclerosis in, 368
cerebral abscess in, 837
diabetic, insulin in, 562
dry, from intermittent claudication, 363
in failure of heart from embolism, 338
in intermittent claudication, treated by sympathectectomy, 364
ischæmic, in thrombo-angiitis obliterans, 365
of bowel in dysentery, 164
of limb in diabetes, 569
of lung, 247. *See also* Lung, gangrene of.
symmetrical, 381
**Gas** gangrene, 108
**Gasserian** ganglion, injection with alcohol of, 748
**Gassing**, vomiting in, 418
**Gastric** crises of tabes dorsalis, differentiation from intestinal obstruction, 463
dyspepsia in, 411
ileus, 419
juice, prolonged secretion of, cause of dyspeptic pain, 411
origin of diarrhœa, 439
pain, differentiation from biliary colic, 500
**Gastritis**, acute, 420
suppurative, 421
chronic. *See* Peptic ulcer.
dyspepsia in, 411
infective, 420
**Gastro-colic** fistula in carcinoma, 431, 433
tetany in, 585
**Gastro-duodeno-pyloroplasty** in duodenal ulcer, 427
**Gastrogenous** diarrhœa, 439
**Gastro-jejunal** ulcer, 427, 428
**Gastro-jejunostomy**, diarrhœa following, 440
effect on HCl in gastric juice, 423
in duodenal ulcer, 427
sequelæ of, 427
**Gastroptosis**, 416
**Gastrostaxis**, 422
**Gastrostomy** in carcinoma of œsophagus, 402
in cicatricial stricture of œsophagus, 402
**Gaucher** type of splenic anæmia, 530
**General** paralysis of the insane, 113, 861
anæsthesia of larynx in, 212
differentiation from cerebral tumour, 857
delirium tremens, 678
neurasthenia, 888
**Genital** organs, hypoplasia of, in status thymico-lymphaticus, 588
**Genito-urinary** system in psychopathology, 891
tuberculosis, bovine infection in, 127
**German** measles, 36
**Giddiness**. *See* Vertigo.
**Gigantism**, 593
**Gingivitis**, 392
**Girdle** pain in tabes, 790
**Glanders**, 136
acute rhinitis in, 197
**Glands**, lymphatic, bronchial, enlarged, causing obstruction of bronchi, 230
compression by, causing jaundice, 476
enlarged, in splenic anæmia of infancy, 531
**Glands**, lymphatic, enlargement of, in acute leukæmia, 533
in Hodgkin's disease, 551
in portal fissure, obstruction by, causing ascites, 477
gastric, destruction of, in pernicious anæmia, 527
lardaceous disease of, 489
submaxillary, in mumps, 46
tonsillar, 395
enlargement of, in rheumatic fever, 59
tuberculosis of, differentiation from Hodgkin's disease, 553
carcinoma of intestine, 433
steatorrhœa in, 504, 515
tuberculous, differentiation from glandular fever, 50
mesenteric, differentiation from cancer of kidney, 653
inflammation of, in enteric fever, 81
sebaceous, affections of, 1014
sweat, affections of, 1012
varicose groin, in filariasis, 555
**Glandular fever**, 49
**Glegg's** paraffin and vaseline mixture in influenza, 64
treatment in nasal catarrh, 197, 198
**Glenard's** disease, 416, 473
movable kidney in, 655
**Glioma** of brain, 850
of spinal cord, 772
**Globus** hystericus, 906
**Glossitic** anæmia, 526 *See also* Anæmia, pernicious.
**" Glossy skin,"** 1009
**Glottis**, œdema of, differentiation from diphtheria, 70
spasm of, 211
**Glycosuria**, alimentary, 565, 569
benign, 570
clinical tests for, 565
complicating pregnancy, 575
eczema and, 984
in acromegaly, 595
in acute bulbar paralysis, 811
in chronic alcoholism, 679
in diabetes insipidus, 596
in diabetes mellitus, 562
in exophthalmic goitre, 580
over-activity of pituitary gland causing, 593
renal, 570
sapræmic, 562
*See also* Diabetes mellitus
**Glycuronic** acid in urine, 567
test of hepatic efficiency, 173
**Gmelin's** test 475
**Goat's** milk, spread of Mediterranean fever by, 172
**Goetsch** test, 580
**Goitre**, 576
colloid, 576
compression of trachea by, 214
endemic, tetany in, 585
exophthalmic, 577, 578
and diabetes mellitus, association of, 580
alopecia areata in, 1016
tachycardia in, 310
*See also* Thyroid gland.
**Gonads**, diseases of, 597
**Gonidia**, 10
**Gonococcal** infections, 72, 73
differentiation from rheumatic fever, 59
septicæmia, 73
*Gonococcus*, 73
in infective endocarditis, 331
in meningitis, 843
in peritonitis, 510
in rheumatoid arthritis, 668
**Gonorrhœa**, 73
cause of stricture of rectum, 457
**Gonorrhœal** rheumatism, 73
**Gout**, 663
acute, differentiation from gonococcal infections, 74
and granular kidney, 682
association with lead poisoning, 682
cause of primary contracted kidney, 631
differentiation from rheumatoid arthritis, 671
dyspepsia of, 411
eczema and, 984
irregular, 666
**Graduated** labour in treatment of tuberculosis, 262
**" Grand mal,"** 869

**Granular kidney** and gout, 682
association with lead poisoning, 682
*See also under* Kidney
**Granuloma** annulare, 998
pyogenicum, 946
**Gravel,** treatment of, 660
**Graves'** disease, 578
diarrhœa of, 440
*See also under* Goitre, exophthalmic.
**Grawitz** tumours, 652
**"Greedy colon,"** 435
**Grocco's** paravertebral triangle, 274
**"Growing pains"** in children, 58
**Guaiacum** test for blood, 408, 617
**Guanidine** and diseases of parathyroid glands, 585
**Guinea** worm, 557
**Gumboil,** 392
**Gumma,** abscess of lung from, 245
causing obstruction of bronchi, 230
mediastinal, 386
of liver, 490
*See also* Syphilis.
**Gums,** swollen, in scurvy, 689
**Gye, W. E.** Recent work on Carcinoma, 1020

**Habit** spasms, 880
differentiation from chorea, 867
**Hachure** in massage, 7
**Hæmagglutinins,** 16
**Hæmamœba,** 10
**Hæmatemesis,** cirrhosis of liver and, 425
in duodenal ulcer, 424
in gastric ulcer, 424
treatment of, 427
in portal cirrhosis, 485
painless, acute ulceration causing, 422
splenic anæmia and, 425, 529
**Hæmatidrosis,** 1013
**Hæmatoma,** subdural, chronic, 847
**Hæmatoporphyrin** in urine, 608
**Hæmatoporphyrinuria,** multiple neuritis and, 740
**Hæmaturia,** 616
differentiation from hæmoglobinuria, 539
essential, 647
in acute nephritis, 620
in bilharziasis, 650
in carcinoma of kidney, 652
in chronic parenchymatous nephritis, 625
in cystic disease of kidney, 654
in embolism of kidney, 380
in endocarditis lenta, 333
in relapsing fever, 146
in renal calculus, 657
in renal tuberculosis, 642
in spirochætosis, 173
**Hæmochromatosis,** cirrhosis of liver in, 484
**Hæmoclasic** crisis, 17
**Hæmocytometer,** 519
**Hæmoglobin,** estimation of, 520
**Hæmoglobinæmia,** 539
**Hæmoglobinuria,** 539
differentiation from acute nephritis, 622
epidemic, 539
malarial, 157
paroxysmal, 539, 540
**Hæmolysis,** 16
as cause of anæmia, 524
conditions associated with, 539
in congenital acholuric jaundice, 532
in paroxysmal hæmoglobinuria, 539
in pernicious anæmia, 527
**Hæmolytic jaundice,** 531
**Hæmopericardium,** 361
**Hæmophilia,** 544
purpura in, 542
**Hæmo-pneumothorax,** 279
**Hæmoptysis,** in *Ascaris lumbricoides* infection, 469
in bronchiectasis, 223
in broncho-pneumonia, 244
in chronic pneumonia, 246
in gangrene of lung, 247
in malignant endocarditis, 333
in mediastinal neoplasms, 387
in mitral disease, 342, 343
in neoplasms of lung, 267
in obstruction of bronchi, 230
in plastic bronchitis, 222
in pulmonary embolism, 270
in pulmonary tuberculosis, 252, 258, 264
in spirochætosis, 175

**Hæmoptysis,** treatment of, 264
**Hæmorrhage,** anæmia due to, 523
blood transfusion in, 546
causing tinnitus, 750
cerebellar, 829
cerebral, 821 *See also* Cerebral hæmorrhage.
extra-dural, 828
from aneurysm, 372
hyperplesia and, 370
in chronic interstitial nephritis, 629
in enteric fever, 83, 85, 87
in erythræmia, 538
in gastric ulcer, 424
treatment of, 427
in leukæmia, 534
in pernicious anæmia, 526
in pulmonary tuberculosis, 250
in scurvy, 688
in splenic anæmia, 529
into pons Varolii, 828
into spinal cord, 777
differentiation from spinal meningeal hæmorrhage, 808
meningeal, 821, 828
differentiation from hæmorrhage into spinal cord, 777
multiple, in scurvy, 688
*per rectum,* in intussusception, 465
renal, causes of, 647
retinal, in anæmia, 524
in chronic interstitial nephritis, 629
spinal meningeal, 808
sub-arachnoid, 828
diagnosis of, 736
sub-dural, 828
*See also* Hæmatemesis, Melæna, Hæmoptysis, Purpura.
**Hæmorrhagic** erosions in acute septic ulcer, 422
of stomach in dyspepsia, 412
nephritis complicating glandular fever, 50
rash in small-pox, 38
**Hæmorrhoids** in thrombosis of inferior vena cava, 380
**Hæmostatics** in hæmoptysis, 264
**Hæmothorax,** 278
massive collapse of lung associated with, 235
septic, 279
**Hæmozoin** in bone-marrow in malaria, 151
**Hair** and hair follicles, affections of, 1014
change of colour of, 1014
dystrophy of, in latent tetany, 580
**Haldane's** oxygen apparatus, 64, 234
*See also under* Oxygen.
**Hands,** eczematoid ringworm of, 958
**Hanot's** cirrhosis, 487
**Hay** fever, 224
prevention of, 228
**Hay's** test for bile salts in urine, 475
**Head,** ringworm of, 957
**Headache** after hysteria, 905
after gastro-jejunostomy, 428
due to quinidine, 317
following gassing, 265
in anxiety neurosis, 896
in cerebral meningeal hæmorrhage, 828
in cerebral tumours, 852
in gastroptosis, 417
in heat stroke, 27
in hypoglycæmia from insulin, 572
in masturbation, 900
in pyrexia, 20
in small-pox, 40
sick, 876
**Head-nodding** in rickets, 693
**Hearing,** nerve tracts associated with, 713
tests for, 725
**Heart,** abnormalities in action of, 303
action of, in pyrexia, 20
aneurysm of, 326
atrophy of, in chronic phthisis, 255
auscultation of, 287
-block, 304
acute, in acute endocarditis, 329
Adams-Stokes syndrome and, 306
as indicated by canter-rhythm, 288
branch-bundle, 304
complete, 305
in auricular fibrillation, 315
in chorea, 866
in myocardial degeneration, 304, 307, 324
sino-auricular, 307

**Heart**-block, slow pulse in, 827
**Heart**, brown atrophy of, 324
cessation of, 306
compensation of, 336
conditions of, in enteric fever, 82
in rheumatic fever, 56
congenital malformations of, 353
erythrocytosis and, 539
cysticercus of, 328
digitalis treatment of auricular fibrillation, 315
dilatation of, 321
adherent pericardium and, 358, 359
in beri-beri, 687
in simple paroxysmal tachycardia, 311
disease and pregnancy, 350
bronchitis in, 215
causing œdema of lungs, 236
complications, 343
hydrothorax in, 278
treatment of, 347
diseases of, 318
disordered action of, 350
displacement of, differentiation from hypertrophy, 320
in pleurisy, 276
estimation of capacity for effort, 294
examination of, 285
failure of, in simple paroxysmal tachycardia, 311
fatty overgrowth of, 326
fibroid degeneration of, 324
in simple paroxysmal tachycardia, 311
frequent action of, 310
*See also* Tachycardia
friction sounds over, 294
hydatid cysts of, 328
hypertrophy of, 318
adherent pericardium and, 359
dilatation in chronic interstitial nephritis, 629
in chronic interstitial nephritis, 630
in cystic disease of kidney, 654
*See also under* Hypertrophy
infrequent action of, 317
*See also under* Bradycardia.
inspection of, 285
latent jaundice in, 476
"law of the," 321
-lung, 338
muscle, abscess in, pyæmia from, 51
*See also* Myocardial degeneration *and* Myocarditis
neoplasm of, 327
orthodiagraphic measurements of, 296
palpation of, 286
parasites of, 328
percussion of, 286
pulse ratio for estimating cardiac efficiency, 294
response of, estimation of, in diagnosis of valvular disease, 346
right-sided failure of, in bronchiectasis, 224
in chronic pneumonia, 246
in emphysema, 233
rupture of, 325
sounds, 287
accentuation of, 287
of second sound in mitral regurgitation, 341
canter-rhythm in, 288
diminution of, 287
in left ventricular hypertrophy, 319
in myocardial degeneration, 324
mode of production of, 287
reduplication of, 287
triple rhythm in, 288
syphilis of, 328
tuberculosis of, 328
valvular disease of, 334
diagnosis of, 334, 344, 345
differentiation from aneurysm, 376
primary contracted kidney, 672
erythrocytosis and, 539
prevention of, 347
prognosis of, 346
right-sided, 344
lesions, relative frequency of, 336
vertical, 295
X-ray examination of, 295
*See also* Aortic, Mitral
**"Heart-lung** preparation," dilatation in, 320
**Heartburn**, 412
**Heat** apoplexy, 26
in treatment, 6
of dyspepsia, 414
loss, 22
production, 22
radiant, in treatment, 6
stroke, 26
**Heberden's** nodes in rheumatoid arthritis, 670
**Hebra's** prurigo, differentiation from urticaria papulosa, 978
**Hectic** fever, 21
**Heine-Medin's** disease, 97
**Heliotherapy** in lupus vulgaris, 950
in phthisis, 261
in tuberculous peritonitis, 515
**Heliotrope** cyanosis in pneumonic influenza, 62, 63
**Hemeralopia** in scurvy, 689
**Hemi-anæsthesia** in cerebral tumours, 854
**Hemianopia,** bitemporal, 745
homonymous, 745
due to pressure from tumours, 594
in migraine, 878
pressure from tumours causing, 594
**Hemichorea** *See* Chorea.
**Hemiplegia,** 813, 817
choreic, 867
crossed, 816
differentiation from writer's cramp, 884
in acute poliomyelitis, 99
in caisson disease, 779
in cerebral embolism, 837
in cerebral tumours, 853
in cerebral hæmorrhage, 824
in disseminated sclerosis, 782
in failure of heart, 338
in lead poisoning, 681
in mitral stenosis, 343
infantile, 819
right, association with aphasia, 820
**Henoch's** purpura, 543
differentiation from intestinal obstruction, 462
**Hepatic** colic, differentiation from intestinal obstruction, 463
efficiency, test of, 473
glycuronic acid test, 479
lævulose test, 473
tests, functional, in diagnosis of portal cirrhosis, 486
*See also under* Liver.
**Hepatisation** of lung, red and grey, 238
**Hepatitis,** acute, cause of catarrhal jaundice, 480
in syphilis, 490
amœbic, 169
**Hepatoptosis,** 416
**"Herald patch"** in pityriasis rosea, 988
**Hereditary** cerebellar ataxy, 839
**Heredity** in anxiety neurosis, 894
in diabetes mellitus, 562
in family periodic paralysis, 919
in progressive muscular dystrophy, 915
**Hernia,** diaphragmatic, 282
differentiation from pneumothorax, 281
strangulated, 459
**Herpes,** facialis, 1000
genitalis, 1000
differentiation from syphilis, 114
gestationis, 1005
in cerebro-spinal fever, 96
in lobar pneumonia, 239
in poliomyelitis, 99
in relapsing fever, 146
in spirochætosis, 175
labialis, 1000
preputialis, 1000
zoster, 998
and varicella, relationship between, 999
differentiation from facial erysipelas, 54
ophthalmicus, 748
**Heterogenetic** beats, 308
**Heterotopic** beats, 308
in simple paroxysmal tachycardia, 310
**Hexamine** in cholecystitis, 497
**Hiccough,** 764
in encephalitis lethargica, 833
**Hidradenitis,** 946
**High** frequency currents in treatment, 6
**Hilus** phthisis, 248, 256
*See also* Phthisis.

**Hip** joint disease, differentiation from sciatic neuritis, 762
**Hippocratic** succussion, 195
in pneumothorax, 281
**Hirschsprung's** disease, 466
**Hirsuties**, 1014
**Hirsutism**, adult, 592
**Histamine** shock, 17
**Hodgkin's** disease, 551
anæmia in, 523
differentiation from glandular fever, 50
enlargement of salivary glands in, 400
enlargement of spleen in, 550
lymphocytic leukæmia simulating, 536
of mediastinum, differentiation from pleurisy, 276
purpura in, 542
**Hofmann's** bacillus, 69
**Homogenetic** beats, 308
**Homogentisic** acid in urine in alkaptonuria, 608
**Homotopic** beats, 308
**Hook** worm, 471
**Hormones**, 558
in psychopathology, 891
**Horn**, cutaneous, 1006
**Hot-air** bath in treatment of chronic parenchymatous nephritis, 626
**Hot-cross** bun skull in rickets, 693
**Hour-glass** contraction of stomach, 425, 429
due to gastric ulcer, 424
stomach, formation of, 422
**Human** and bovine tubercle, distinction between, 126
**Hunger** œdema 697
osteomalacia, 695
**"Hunger pain,"** 410
in duodenal ulcer, 424
**Huntington's** chorea, 867
**Hutchinson's** teeth in congenital syphilis, 122
**Hydatid** cyst causing jaundice, 476
causing suppurative cholangitis, 495
differentiation from pancreatic cyst, 508
of heart, 328
of kidney, 648
of liver, 493
causing ascites, 477
changes in, 494
differentiation from hydronephrosis, 645
multilocular, 493
secondary, 493
simulating ascites, 478
pleurisy, 276
of lung, 268
suppurating, gangrene of lung following, 247
thrill, 494
**Hydræmic** atrophy in children, 694
nephritis, 612
plethora as cause of chlorosis, 525
**Hydrarthrosis**, intermittent, 672
**Hydroa**, 1005
æstivale, 608
**Hydrocarpus**, fatty acids of, in treatment of leprosy, 135
**Hydrocele**, filarial, 555
**Hydrocephalus**, acute, 130
chronic, 858
internal, acquired, 861
cause of hypopituitarism, 593
congenital, 859
secondary internal, 854
**Hydrochloric** acid, absence of, in gastric juice in pernicious anæmia, 527 *See also* Achylia
**Hydrogen** ions, concentration of in blood, 547
**Hydronephrosis**, 643
differentiation from pancreatic cyst, 508
in movable kidney, 655
in renal calculus, 657
simulating hydatid disease, 495, 649
**Hydropericardium**, 360
**Hydrophobia**, 102
differentiation from tetanus, 107
spasm of glottis in, 211
spurious, 103
virus of, method of spread after inoculation, 102
**Hydro-pneumothorax**, 279
in phthisis, 255
**Hydrothorax**, 276, 278
in chronic parenchymatous nephritis, 625
in failure of heart, 338
**Hygiene**, 4
**Hyperæmia** of skin, 924
**Hyperæsthesia** in appendicitis, 453
in compression of cord, 768
**Hypercholesterinæmia**, 498
**Hyperchlorhydria**, 407
in chronic indigestion, 413
**Hypergenitalism**, 598
**Hyperglycæmia** in acromegaly, 595
in diabetes mellitus, 564
in exophthalmic goitre, 580
**Hyperidrosis**, 1012
**Hyper-interrenopathy**, 592
**Hyperkeratoses** of skin, 1005
**Hypernephroma**, 652
of suprarenal capsule, adiposity in, 599
**Hyperpiesia**, 369
blood pressure in, 369
in primary contracted kidney, 631
symptoms of, 370
treatment of, 370
**Hyperpituitarism**, 593, 595
**Hyperplastic** sclerosis of arteries, diffuse, 366, 369
**Hyperpnœa**, 188
**Hyperpyrexia**, 19
in acute bulbar paralysis, 811
in cerebro-spinal fever, 95
in cholera, 180
in delirium tremens, 678
in dengue, 178
in heat-stroke, 27
in malaria, 152
in malignant tertian infection, 153
in rheumatic fever, 56, 57, 58
in tetanus, 106
in yellow fever, 177
**Hyper-resonance**, 191
**Hypersecretion** of gastric juice, 407
**Hyperthymismus**, 587
**Hyperthyroidism** and quinidine, 317
**Hypertonic** saline, intravenous injection of, in cholera 182
**Hypertonic** stomach, appetite causing, 405
**Hypertrophic** cervical pachymeningitis, 807
pulmonary osteoarthropathy, 672
**Hypertrophy** of auricles, 320
of heart, 318
anatomical changes, 319
concentric, 336
eccentric, 336
left sided, causes of, 319
physical signs, 319
right-sided, causes of, 319
in chronic bronchitis, 219
of left auricle in mitral stenosis, 342
of left ventricle in aortic stenosis, 339
of right ventricle in mitral stenosis, 342
**Hyphomycetæ**, 10, 144
**Hypnosis** in treatment of hysteria, 908
**Hypochlorhydria** in diarrhœa, 439
in rosacea, 975
*See also* Achylia gastrica.
**Hypogenitalism**, 598
**Hypoglycæmia** caused by muscular exertion, treatment of, 576
in cyclical vomiting, 418
in dyspepsia, 412
symptoms of, 572
**Hypoglycæmic** coma rapidly following diabetic coma 575
symptoms of, 575
**Hypophyseal** dystrophia adiposo-genitalis, 595
**Hypophysis**, symptoms due to tumours of, 815
**Hypopituitarism**, 593, 595
epilepsy associated with, 595
**Hypostatic** congestion in pyrexia, 20
**Hypothyroidism**, 577
as cause of arterio-sclerosis, 582
in myxœdema, 581
**Hypotonus** of stomach in dyspepsia, 411, 412
**Hysteria**, 902
causing reflex diarrhœa, 440
diagnosis of, 906
differentiation from chorea, 867
disseminated sclerosis, 783
epilepsy, 872
hydrophobia, 103
myasthenia gravis, 920
tetanus, 107
tetany, 586
disorders of cranial nerves in, 905
special senses in, 904

**Hysteria,** fits in, 904
mental condition in, 903
nervous symptoms in, 904
polyuria in, resembling diabetes insipidus, 597
reflexes in, 906
sensory and motor symptoms, 905
stigmata of, 903
treatment of, by persuasion, 908
by psycho-analysis, 909
by suggestion, 908
**Hysterical** fit, diagnosis of, 907
following epilepsy, 871
vomiting, 418

**Ice-bags,** 26
**Ichthyosis,** 1007
hystrix, 1007
**Icterus.** *See* Jaundice.
neonatorum, 477
**Idiopathic** or pernicious anæmia, 526
**Immune** body, 17
**Immunisation** in prevention of infection, 13
**Immunity,** 14
acquired, 11, 15
in influenza, 62
artificial, 15
hereditary disposition, 15
in typhus fever, 75
natural, 11, 14
**Impetigo,** bullosa, 927
circinata, 927
contagiosa, 927
contagious, pediculi capitis and, 965
of Bockhardt, 927, 938
**Impotence** in anxiety neurosis, 895
**Inanition** in ketosis, 568
**Inclusion** bodies in scarlet fever, 29, 32
**Incompetence** of valves of heart, 335
**Inco-ordination,** 732
in Friedreich's ataxia, 797
muscular, in tabes, 789
**Incubation** periods, 11
of acute poliomyelitis, 99
of anthrax, 138
of benign tertian and quartan infection, 152
of bubonic plague, 184
of cerebro-spinal fever, 95
of chicken-pox, 44
of cholera, 179
of dengue, 178
of diphtheria, 67
of dysentery, 164
of enteric fever, 82
of erysipelas, 63
of foot-and-mouth disease, 140
of hydrophobia, 102
of infectious disease, 12
of influenza, 62
of measles, 33
of Mediterranean fever, 172
of mumps, 46
of relapsing fever, 145
of rubella, 36
of sand-fly fever, 148
of scarlet fever, 29
of small-pox, 38
of syphilis, 110
of tetanus, 105, 107
of trench fever, 64
of trypanosomiasis, 159
of typhus fever, 75
of whooping-cough, 47
of yellow fever, 176
**Indican** in urine, 608
in pellagra, 696
**Indigestion,** acute, 409
chronic, 409
differentiation from angina, 363
*See also* Dyspepsia
**Induration** of lung, brown, 270
red, 270
**Infant** feeding in enteritis, 445
**Infants,** multiple abscesses of, 945
**Infantile** cerebral diplegia, 838
erythema of Jacquet, 930
paralysis. *See* Poliomyelitis, acute.
scurvy, 690
**Infantilism,** 600

**Infarcts,** 379
fibrosis of heart, secondary result of, 324
hæmorrhagic, or red, 379
in failure of heart, 338
in spleen in leukæmia, 534
pleurisy in, 271
pulmonary, 269
septic, 379
white, 379
*See also* Embolism.
**Infections,** asthma and, 227
bacterial, of kidney, 634
cause of thrombosis of inferior vena cava, 380
disinfection in, 13
isolation, 13
mixed, 12
nature of, 9
prevention of, 13
secondary, in primary contracted kidney, 631
transmission of, 12
**Infectious** diseases, 9
notification of, 14
**Infective** endocarditis, 328
*See also* Endocarditis, malignant.
**Inferior** vena cava, thrombosis of, 380
**Influenza,** 61
abdominal type, 63
acute rheumatism in, 197
as cause of acute encephalitis, 820
bronchitis in, 215
differentiation from enteric fever, 87
pneumonia, 241
scarlet fever, 31
small-pox, 41
trench fever, 65
method of spread of, 61
pericarditis in, 355
pleurisy in, 271
pneumonic type, 62
**Injury,** local, causing neuritis, 738
**Inoculation.** *See* Serums, Vaccines.
**Insanity,** fears of, in anxiety neurosis, 895
forms of, in chronic alcoholism, 679
manic-depressive, differentiation from anxiety neurosis, 897
**Insomnia** in anxiety neurosis, 895
treatment of, 899
in chronic alcoholism, 680
in encephalitis lethargica, 834
in neurasthenia, 887
treatment of, 889
**Insulin,** 563
after-treatment, 573
F. G. Banting's work on, 564
diarrhœa of, 440
evidence of some curative effect, 575
hypoglycæmia from, 572
in treatment of diabetes, 572
isolation of, 564
local irritation at time of injection of, 573
mode of preparation, 564
treatment, choice of case for, 575
**Inspection** of abdomen, 389
of chest, 187
of heart, 285
**Intelligence,** 723
**Intermittent** claudication, 382
in thrombo-angiitis obliterans, 365
hepatic fever, 499
hydrarthrosis, 672
**Internal** secretions, 558
in psychopathology, 891
**Inter-renal** system, 588
**Intertrigo,** 929
*See also* Eczema intertrigo.
**Interventricular** septum, deficiency of, combined with pulmonary stenosis, 353
**Intestinal** obstruction, 457
acute pancreatitis simulating, 506
acute, symptoms of, 460
causing peritonitis, 509
chronic, symptoms of, 461
effects of, 459
predisposing to tetany, 585
simulating peritonitis, 511
tetany in, 585
sand in mucous colitis, 450
stasis, peritoneal adhesions and, 516
worms, 466
**Intestine,** large, amœbic ulcers of, 166

**Intestines**, adenoma of, 456
carcinoma of, 456
disease of, 434
in failure of heart, 338
lardaceous disease of, 489
lymphosarcoma of, 456
tuberculosis of, 456
**Intolerance** to arseno-benzol, 119
**Intoxications** causing multiple neuritis, 740
**Intravenous** injection of quinine in malaria, 156
**Intra-ventricular** puncture in cerebro-spinal fever, 96
**Intubation** in carcinoma of œsophagus, 402
in diphtheria, 71
**Intussusception**, 464
differentiation from dysentery, 165
**Inunction** in syphilis, 119
**Iodide** of potassium in treatment of leprosy, 136
**Iodides** causing skin eruptions, 980
in treatment of syphilis, 117
**Iodine**, lack of, cause of endemic goitre, 576
treatment of goitre, 581
**Ionisation** in treatment, 5
**Iritis** in congenital syphilis, 122
in diabetes, 570
in dysentery, 165
in frontal herpes, 999
in gonorrhœal rheumatism, 73
in relapsing fever, 146
in syphilis, 112
**Irregularity**, phasic, vagal action causing, 318
"youthful," 304
**Irritant** gases, poisoning by, 265
**Islands** of Langerhans, 503, 563
histological changes in diabetes, 564
**Iso-glycuronic** acid in urine, 567
**Isolation** in prevention of infection, 13
in scarlet fever, 32
in small-pox, 42
in varicella, 45
**Itch**, 966. *See also* Scabies.
**Itching**. *See* Pruritus

**Jail** fever, 74
**Jarisch-Herxheimer** reaction, in poisoning by arseno-benzol, 123
**Jaundice**, 474
acholuric, 474, 531
acquired, 532
congenital, 531
resemblance to pernicious anæmia, 529
black, 474
bradycardia in, 317
catarrhal, 480
in new born, 477
causes of, 475
clinical tests for, 475
complicating relapsing fever, 147
differentiation from Addison's disease, 591
yellow colour due to carrotin, 474
dissociated, 474
familial, in new born, 477
hæmolytic, 476, 531
hepatic, toxic and infective, 476
in abscess of liver, 480
in acute pancreatitis, 506
in acute yellow atrophy of liver, 481, 482
in arseno-benzol poisoning, 123, 124
in carcinoma of liver, 492
in cholecystitis, 497
in hydatid disease of liver, 494
in mitral disease, 342
in new-born children, 477
in pneumonia, 241
in spirochætosis, 173
in syphilis, 490
infectious, 173
infective, in new born, 477
latent, 474, 476
in portal cirrhosis, 485
obstructive hepatic, 475, 476
sign of hepatic inefficiency, 473
Van den Bergh's test in, 475
yellow vision in, 474
**Jaw-winking**, 764
**Jejunal** ulcer, 428
after gastro-jejunostomy, 427
**Jejunostomy** in gastric ulcer, 427
**Jendrassik's** reinforcement, 734

**Jerks**, supinator, in multiple neuritis, 741
**Jews**, prevalence of diabetes mellitus among, 562
**Joints**, tuberculosis in. *See* Arthritis.
**Jugular** pulse, 302
**Juxta-pyloric** ulcers, 422
ulcer, chronic, symptoms of, 424
*See also* Gastric ulcer, Peptic ulcer, and Duodenal ulcer.

**Kala-Azar**, 161
cirrhosis of liver in, 484
**Kaposi's** disease, 1009
**Keratitis** in congenital syphilis, 122
neuro-paralytic, 748
**Keratodermia** ble morrhagica, 990
**Keratosis**, 1006
follicularis, 1006
pilaris, 1006
in chronic arsenical poisoning, 684
**Kernig's** sign, 130, 132, 732
in cerebro-spinal fever, 95
in spinal meningitis, 804
**Ketosis**, 549, 568
acidæmia in, 548
association with alkalæmia, 549
clinical tests for, 568
in acute yellow atrophy of liver, 482
in cyclical vomiting, 418
**Kharsivan**, 116, 118
**Kidney** and **Kidneys**,
bacterial infections of, 634
calculus in, 656
formation of, 657
congestion of, in failure of heart, 338
cystic disease of, 653
associated with cystic disease of liver, 492
cysts of, 653, 654
simulating ascites, 478
decapsulation of, in treatment of chronic parenchymatous nephritis, 627
diseases of, 611
chronic, differentiation from valvular heart disease, 345
differentiation from cerebral tumour, 856
embolism in, 380
embryonic tumours of, 651
enlargement of, in diabetes mellitus, 568
epithelial carcinoma of, 651
examination of, 603
excision of, in treatment of hydronephrosis, 645
in treatment of tubercle, 642
flea-bitten, in malignant endocarditis, 331
functions of, tests of, 609
in chronic interstitial nephritis, 628
in suppurative nephritis, 638
in trypanosomiasis, 160
granular, 627, 632
association with lead poisoning, 682
gout predisposing to, 667
histological structure of, 611
hydatid disease of, 648
in enteric fever, condition of, 82
in leukæmia, 534, 536
inflammation of, causes of, 612
lardaceous disease of, 489, 611, 645
movable, dyspepsia in, 411
movable or prolapsed, 416, 603, 654
myelin, 624
primary contracted, 366, 630
and cerebral hæmorrhage, 821
in gout, 664
ptosis of, 416
secondary contracted, 627
differentiation from chronic parenchymatous nephritis, 626
senile arterio-sclerotic, 325, 633
in emphysema, 243
threshold of, for sugar, 564
tuberculosis of, 640
tumour of, causing jaundice, 476
differentiation from distended gall bladder, 497
white, contracting, 624
large, 624
*See also* Nephritis, Bright's disease.
**Kjeldahl's** method for estimation of nitrogen in urine, 605
**Klebs-Loeffler** bacillus, 66, 69
**Klumpke's** paralysis, 758

**Kneading** in massage, 7
**Knee** jerk, 734
disappearance in multiple neuritis, 741
**Koch's** new tuberculin (T.R.), 261
**Koplik's** spots, 34, 35
**Korsakow's** disease, 741
**Kronig's** isthmus in apical phthisis, 252
**Kyphosis** in Friedreich's ataxia, 798

**Labio-glosso-laryngeal** paralysis, 809
**Labyrinthitis,** associated with vertigo, 751
**Lactation** predisposing to tetany, 584
**Lactic** acid in test meal in carcinoma of stomach, 434
**Lactose** in urine, 567
**Lævulose** test for hepatic efficiency, 473
in urine, 563, 567
**Lamblia,** presence of, in amœbic dysentery, 167
**Laminectomy,** exploratory, in compression of cord, 770
**Landry's** paralysis, 803
acute poliomyelitis simulating, 100
differentiation from chronic myelitis, 775
**Langerhans,** Islands of, 563
**Lardaceous** degeneration in syphilis, 114
in leprosy, 134
disease complicating phthisis, 255
differentiation from carcinoma of liver, 492
chronic parenchymatous nephritis, 626
in chronic interstitial nephritis, 628
of kidney, 611, 645
of liver, 489
in syphilis, 490
**Laryngeal** crisis, spasm of glottis in, 211
diphtheria, 68, 69
muscles, paralysis of, 207
*See also* Vocal cords
nerve, recurrent, lesions of, causing complete paralysis of vocal cords, 208
superior, injury to, anæsthesia of larynx in, 212
nerves, compression of, in mediastinal neoplasms, 386
paralysis in beri-beri, 687
stridor, 212
**Laryngismus** stridulus, 211, 875
associated with tetany, 586
differentiation from thymic asthma, 587
in rickets, 693
**Laryngitis,** acute catarrhal, 199
catarrhal, differentiation from diphtheria, 70
chronic catarrhal, 201
differentiation from laryngeal tuberculosis, 203
syphilitic laryngitis, 205
chronic hypertrophic, 202
complicating measles, 35
in coryza, 197
membranous, 201
œdematous, 200
spasm of glottis in, 211
stridulosa, 200
differentiation from membranous laryngitis, 201
treatment of, 200
syphilitic, 204
differentiation from laryngeal tuberculosis, 203
tuberculous, 202
differentiation from chronic catarrhal laryngitis, 202
paralysis of adductors of vocal cords, 210
syphilitic laryngitis, 203
in phthisis, 55
**Laryngo-fissure,** operation of, 207
**Larynx,** anæsthesia of, 212
carcinoma of, differentiation from chronic catarrhal laryngitis, 202
diseases of, 199
foreign bodies in, 207
lupus of, 204
obstruction of, differentiation from asthma, 228
compression of trachea, 214
paralysis of, in alcoholic neuritis, 741
perichondritis of, 205
tumours of, 206
ulceration of, in enteric fever, 85
**Lavage** in carcinoma of stomach, 434
in dilatation of stomach, 429
*Laverania malariæ,* 150
**Lead,** action on anterior horn cells of peripheral nerves, 740
colic, differentiation from intestinal obstruction, 463
line, 681
paralysis, 680
differentiation from progressive muscular atrophy, 801
poisoning, 680
anæmia in, 523
as cause of primary contracted kidney, 631
**Left** ventricle, small, in mitral stenosis, 342
**Legal's** test for ketosis, 568
*Leishmania canimum,* 161
*donovani,* 160, 161
*infantum,* 161
*tropica,* 161
**Leishmaniasis,** 160
**Lemon** juice, fresh, in scurvy, 690
**Lenticular** degeneration, progressive, 840
nuclei, degeneration of, paralysis agitans in, 841
**Lentigo,** 1010
**Leontiasis,** 134
**Lepra** anæsthetica, 134
cells, 134
maculosa, 134
mutilans, 135
nodosa, 134
prevention of, 135
tuberculosa, 134
**Leproma,** 134
**Leprosy,** 133
**Leptomeningitis,** acute, 804
chronic, 806
*Leptospira hebdomadis,* 176
*ictero-hæmorrhagica,* 174
*icteroides,* 176
**Leptothrix,** 10, 1014
in gangrene of lung, 247
of mouth, 392
**Leucopenia,** 522, 537
in pernicious anæmia, 528
in splenic anæmia, 530
**Leucocytes,** 520
*See also* Leukæmia.
**Leucocythæmia,** 522, 523, 532
**Leucocytosis,** 522, 523, 537
after perforation of typhoid ulcer, 85
digestion, 537
in amœbic dysentery, 167
in amœbic hepatitis, 171
in elliot syndrome, 352
in empyema, 276
in erysipelas, 54
in glandular fever, 50
in gout, 665
in influenza, 63
in lead poisoning, 681
in leukæmia, 533
in lobar pneumonia, 240
in pyæmia, 51
in pyrexia, 22
in rat-bite fever, 144
in splenic anæmia of infancy, 531
in whooping-cough, 49
**Leucodermia,** 1012
**Leucomelanoderma,** alopecia areata associated with, 1016
**Leucopenia** in influenza, 63
**Leukæmia,** 522, 523, 532
acute, 536
differentiation from Kala-azar, 161
glandular fever, 50
splenic anæmia, 530
leucoid, myelocytic, myeloid, or spleno-medullary, 533
lymphatic, lymphocytic, or lymphoid, 535
enlargement of salivary glands in, 400
nodular, 536
purpura in, 542
**Leukæmic** erythrodermia, 994
**Leukanæmia,** 533
**Lice,** infecting agent in relapsing fever, 145, 146
spread of typhus fever by, 75
transmission of infection by, 12
trench fever by, 64
*See also* Pediculosis.

**Lichen** circumscripta, 939
planus, 995
differentiation from papular syphilide, 111
psoriasis, 991
syphilis, 114
urticaria papulosa, 978
scrofulosorum, 953
tropicus, 945, 1013
urticatus, 977
**Light** touch, sensibility to, 729
in treatment, 6
treatment in angina, 364
in lupus vulgaris, 950
ultra-violet, in treatment of alopecia areata, 1017
in rickets, 691, 695
**Lightning** pains in tabes, 788
stroke, ventricular fibrillation in, 318
**Limbs,** arteries of, embolism in, 380
**Lineæ** albicantes, 1009
gravidarum, 1009
**Lingual** tonsil, painting of, in phthisis, 263
**Lipæmia,** 568
retinalis, 568
**Lipiodol,** injection of, in spinal compression, 769
**Lipoma** of stomach, 434
**Lips,** secondary vaccinia on, 44
**Liquids** in diet in dyspepsia, 414
**Livedo** reticulata, 973
**Liver,** abscess of, 479
aspiration of, 171
causing acute peritonitis, 508
ascites, 477
complicating bronchiectasis, 224
differentiation from malaria, 154
in suppurative pancreatitis, 505
in suppurative pylephlebitis, 502
tropical, 167
differentiation from hydatid, 495
resemblance to suppurative cholangitis, 496
action of organic arsenic compounds on, in arseno-benzol poisoning, 123
actinomycosis in, 141
acute yellow atrophy of, 481
differentiation from yellow fever, 177
in arseno-benzol poisoning, 123
jaundice from, differentiation from catarrhal jaundice, 484
latency of, 483
multiple nodular hyperplasia in, 481
necrosis in, 482
purpura in, 542
subacute atrophy in, 482
amœbic infection of, 169
atrophy of, 472, 488
capsule of, thickened, in cirrhosis, 484
carcinoma of, 491
*See also* Carcinoma of liver
cells, degeneration of, in chronic enteritis, 444
cirrhosis of, 483
*See also under* Cirrhosis of liver.
conditions in enteric fever, 82
congestion of, 479
cystic disease of, 492
diseases of, 472
dropped, 416
efficiency of, tests for, in arseno-benzol poisoning, 124
enlarged and infiltrated with leucocytes in leukæmia, 534, 535
enlarged and tender, in simple paroxysmal tachycardia, 311
enlargement of, 472
differentiation from cancer of kidney, 652
in abscess, 480
in carcinoma, 491, 492
in catarrhal jaundice, 481
in hydatid disease, 495
in mitral disease, 342
in splenic anæmia, 529
fatty, complicating phthisis, 255
fatty degeneration of, 488
in cyclical vomiting, 418
fatty infiltration of, 488
in chronic enteritis, 444
function, tests of, 473
ascites in, 473
glycuronic acid test, 473
impairment of, causing jaundice, 475
**Liver,** function, in catarrhal jaundice, 481
jaundice in, 473
gumma of, 490
hobnailed, 483
hydatid of, 493
*See also under* Hydatid of liver.
in acute leukæmia, 534
in arseno-benzol poisoning, 123
lardaceous disease of, 489
*See also under* Lardaceous.
new growths in, 491
nutmeg, 479
in failure of heart, 338
pigment in, cirrhosis and, 484
ptosis of, 416
pulsating, in tricuspid regurgitation, 344
puncture in diagnosis of amœbic abscess, 171
pus in, in melioidosis, 137
syphilis of, 489
in new born, 477
tuberculosis of, 491
**Loa** loa, 556
**Lobar** pneumonia. *See* Pneumonia, Pneumothorax, etc
**Localisation,** "elective," in peptic ulcers, 424
**Lockjaw,** 105 *See also* Tetanus
**Locomotor** ataxy, 786
in syphilis, 113
*See also* Tabes
**Loewi's** mydriatic test, 503
**Ludwig's** angina, 395
**Lugol's** solution in treatment of goitre, 581
**Lumbago,** 910
differentiation from small-pox, 41
**Lumbar** abscess, 512
puncture in acute poliomyelitis, 101
in cerebral meningeal hæmorrhage, 828
in cerebral pachymeningitis, 848
in cerebral tumours, danger of, 857
in cerebro-spinal fever, 96
in diabetes insipidus, 597
in spinal meningitis, 806
in treatment of uræmic convulsions, 623
in tuberculous meningitis, 143
*See also* Cerebro-spinal fluid
**Luminal** in treatment of reflex spasms of limbs, 771
**Lung** and **Lungs,**
abscess of, 245
from foreign body in bronchi, 230
in pneumonia, 241
pneumothorax in, 279
actinomycosis in, 142
amœbic, 167
brown induration of, in failure of heart, 338
changes in, in congenital syphilis, 121
collapse of, 230, 234
massive, 235
in paralysis of diaphragm, 755
compression of, causes, 235
condition of, in enteric fever, 82
decortication of, for empyema, 278
diseases of, 231
ascites in, 477
erythrocytosis and chronic diseases of, 539
fibroid, 222, 230, 245
gangrene of, 247
complicating bronchiectasis, 224
differentiation from fœtid bronchitis, 221
in laryngeal perichondritis, 205
in pneumonia, 241
neoplasm of lung causing, 266
pneumothorax in, 279
hydatid of, 268
*See also* Hydatid.
neoplasms of, 266
causing pneumonia, 266
simulating pleurisy, 276
œdema of, 236
hypostatic, 236
in beri-beri, 687
in hypoglycæmic coma, 576
pneumonic or gangrenous, neoplasm of lung causing, 266
resonance, normal, 191
rupture of, spontaneous pneumothorax in, 279
septic infarcts in, pyæmia from, 51
structure of, 193
**syphilis of, 264**
tuberculosis of, 248
*See also* Phthisis.

**Lupus** erythematosus, 971
differentiation from alopecia areata, 1016
psoriasis, 991
rosacea, 975
verrucosus, 951
vulgaris, 947
differentiation from lupus erythematosus, 950, 972
of larynx, 204
**Lymph** in pleurisy, 272
scrotum, 556
**Lymphadenoma**, 551
*See also* Hodgkin's disease.
**Lymphanæmia**, 536
**Lymphangioma** of skin, 1018
**Lymphangitis**, streptococcal, 555, 931
**Lymphatic** glands. *See* Glands, lymphatic.
**Lymphatic** system, diseases of, 551
**Lymphoblastic** erythrodermia, 994
**Lymphocytes**, 521
excess of, in tuberculous pleurisy, 276
increased, in lymphocytic leukæmia, 535
**Lymphocythæmia**, 535
*See also* Leukæmia.
**Lymphocytosis**, 537
in influenza, 63
in status thymico-lymphaticus, 587
**Lymphomyelocytes** in lymphocytic leukæmia, 536
**Lymphorrhages** in myasthenia gravis 919
**Lympho-sarcoma**, mediastinal, 386
of tonsils, 398
**Lysis** in pyrexia, 22
in lobar pneumonia, 240
**Lyssaphobia**, 103

**McBurney's** point, 453
**Macrophages**, 16, 521
in dysentery, 165
**Madura** foot, 143
*Madurella mycetomi*, 143
**Magnesium** sulphate in diagnosis of gall stones, 500, 501
"*Main d'accoucheur*," 585
*Main en griffe*, 757, 800
**Maize**, and pellagra, 696
**Malaria**, 149
differentiation from Addison's disease, 591
cholera, 181
enteric fever, 88
Kala-azar, 162
liver abscess, 171
Mediterranean fever, 173
plague, 185
pneumonia, 241
pyæmia, 52
relapsing fever, 147
sand-fly fever, 148
suppurative nephritis, 638
trench fever, 65
yellow fever, 177
enlargement of spleen in, 549
malignant endocarditis in, 330
mixed infections, 153
paroxysmal hæmoglobinuria in, 540
**Malarial** hæmoglobinuria, 157
treatment of general paralysis of insane, 864
**Malignant** disease, cause of thrombosis of inferior vena cava, 380
polyneuritis in, 740
endocarditis, 330
*See also* Endocarditis, malignant.
growths, chyluria due to, 556
pustule, 138
small-pox, 41
tertian infection, 153
**Malingering** and epilepsy, 872
and hysteria, differentiation between, 907
**Mallein** in glanders, 137
**Malta** fever, 171
**Manganese** poisoning, chronic, 684
**Mania** in chronic mercurial poisoning, 683
in pellagra, 695
**Manic** depressive insanity, differentiation from anxiety neurosis, 897
**Marmite** in treatment of beri-beri, 688
**Marrow.** *See* Bone-marrow.
**Massage** in treatment, 7
of constipation, 437
**Mass** reflex, 714
**Mast** cells, 521
increased in myelocytic leukæmia, 535
**Mastitis** complicating mumps, 47
**Masturbation** in anxiety neurosis, 895
fears in connection with, treatment of, 900
**Maturation** of spots in chicken-pox, 45, 46
**Measles**, 33
acute rhinitis in, 197
bronchitis in, 215
differentiation from cerebro-spinal fever, 97
scarlatina and rubella, 36
scarlet fever, 31
typhus, 78
fatty degeneration of heart in, 323
hæmorrhagic, 35
papular, differentiation from small-pox, 42
pleurisy in, 271
purpura in, 542
quarantine period in, 13
**Meckel's** diverticulum, 459
**Mediastinal growths**, 386
compression of trachea by, 213
differentiation from aneurysm, 376
pleurisy, 277
**Mediastinitis**, 356, 359, 384
fibrosa, 356, 384
**Mediastino-pericarditis**, 356, 384
**Mediastinum**, diseases of, 384
**Mediterranean** fever, 171
differentiation from enteric fever, 88
**Medulla oblongata**, diseases of, 808
tumours of, 812
**Megaloblasts**, 520
**Megalocytes**, 520
in pernicious anæmia, 527
**Megrim**, 876
**Melæna**, 440
in duodenal ulcer, 424
in gastric ulcer, 424
in portal cirrhosis, 485
**Melanæmia**, 151
**Melanin** in urine, 608
**Melioidosis**, 137
**Menière's** syndrome, 752
**Meningeal hæmorrhage**, 821, 828
*See also* Hæmorrhages, meningeal
**Meninges**, spinal, diseases of, causing compression, 767
inflammation of, symptoms, 769
new growths of, causing compression of cord, 768
or extra-dural, new growths of, symptoms of, 769
**Meningitis**, 843
basal, posterior, 93, 96
benzoin test in, 738
cerebro-spinal, 93
encysted and loculated, 96
*See also* Cerebro-spinal fever.
differentiation from acute poliomyelitis, 100
broncho-pneumonia, 244
cholera, 181
delirium tremens, 678
encephalitis lethargica, 834
enteric fever, 88
intestinal obstruction, 463
typhus, 78
infective, differentiation from cerebral meningeal hæmorrhage, 828
in malignant endocarditis, 331
micro-organisms found in, 843
pneumococcal, 845
and pneumococcal endocarditis combined, 332
serosa circumscripta, 768
spinal, 804
differentiation from tetanus, 107
suppurative, 843
differentiation from cerebral abscess, 838
syphilitic, 845
causing compression of cord, 767
differentiation from cerebral tumours 856
paroxysmal headache due to, differentiation from migraine, 879
tuberculous, 129
**Meningococci** in malignant endocarditis, 330
*Meningococcus*, 93
**Meningo-encephalitis** in Malta fever, 173
in relapsing fever, 146
in trypanosomiasis, 158

**Meningo-myelitis** in trypanosomiasis, 158
syphilitic, 773
**Menopause,** premature, 598
**Menorrhagia** in lead poisoning, 682
**Mental** anxiety, dyspepsia in, 412
**Mental** changes in cerebral tumours, 853
in disseminated sclerosis, 782
encephalitis lethargica, 834
depression in influenza, 62
impairment in infantile cerebral diplegia, 839
symptoms in anxiety neurosis, 895
treatment of, 899
**Meralgia** paræsthetica, 762
**Mercurial** poisoning, chronic, 683
**Mercury** in congenital syphilis, 122
in syphilis, 117
in syphilitic myelitis, 776
myocarditis, 350
intra-muscular injections of, 119
oral administration of, in syphilis, 119
**Mesenteric** artery, embolism of, 386
glands. *See under* Glands.
**Metabolism,** basal, 558
in Addison's disease, 590
increased in exophthalmic goitre, 558, 578
in goitre, 577
in leukæmia, 533
in obesity, 599
in polycythæmia rubra, 538
low, in under-nutrition, 571
nomogram relating, 559, 560
carbohydrate, physiology of, 562
diseases of, 558
in diabetics, 571
in psychopathology, 891
in pyrexia, 20, 22
normal, standards of, 561
**Metallic** tinkling, 195
in phthisis, 254
in pneumothorax, 280
**Metastatic** gonococcal infection, 73
**Methæmoglobin** in urine, 617
*See also under* Urine.
**Methæmoglobinæmia,** 541
clubbing of fingers in, 673
**Metrocyte,** 521
**Microblasts,** 520
**Micrococci,** infection by, 9
*Micrococcus melitensis,* 171
*tetragenus* in peritonitis, 510
in pleurisy, 271
**Microcytes,** 520
*Microfilaria diurna,* 557
*nocturna,* 551
**Microphages,** 16, 321
*Microsporon Audouini,* 958
*furfur,* 961
*minutissimum,* 961
**Mid-diastolic** murmurs, 288
in mitral stenosis, 291
**Migraine,** 876
ophthalmoplegic, 879
**Mind,** unconscious, 892
**Mikulicz's** disease, 400
**Miliaria,** 1013
in pyrexia, 19
in rheumatic fever, 56
papulosa, 1013
rubra, 945, 1013
**Milium,** 1014
**Milk,** channel of infection in diphtheria, 66
in enteric fever, 80
in scarlet fever, 28
**"Milk spots"** following pericarditis, 358
**Mind,** unconcious, 892
**Mitral** disease, tricuspid regurgitation in, 343
regurgitation, 341
in chorea, 866
murmurs in, 290, 291
"simple," in gonorrhœal rheumatism, 73
stenosis, 342
achalsia of cardia in, 403
as indicated by canter-rhythm, 288
auricular fibrillation in, 313
measurement of hypertrophy in, 319
mode of production of, 285
murmurs in, 288—291
tricuspid stenosis and, 344
valve, results of inflammation 335
**Moebius'** sign in exophthalmic goitre, 580
**Mogiphonia,** 213
**Mollities** ossium, 673
**Molluscum** contagiosum, 963
**Monckeberg's** sclerosis, 367
**Monilethrix,** 1014
**Mononuclear** cells, large, 521
excess of, in pleurisy, 276
increase of, in malaria, 155
**Monoplegia,** 813
**Moogrol** in treatment of leprosy, 136
**Morbilli,** 33
**Morbus** cœruleus, 354
**Morphœa,** 1008
**Mors** thymica, 587, 588
**Mosquito** in malaria, 149
infecting agent in yellow fever, 176
in filariasis, 554
transmission of dengue by, 178
infection by, 12
**Motor** nerve fibres, toxins of tetanus transmitted by, 105
nerves, examination of, 730
**Moulds,** 10
**Mouth,** cleansing of, in pyrexia, 24
diseases of, 392
temperature, normal, 18
**Movable** kidney, 416, 603, 654
**Mucous** secretion in neoplasms of lung, 266
**Mulberry** rash of typhus fever, 76
**Multi-glandular** syndromes, 601
**Multiple** abscesses of infants, 945
**Mumps,** 46
differentiation from glandular fever, 50
pancreatitis in, 506
quarantine period in, 13
**Murexide** test for uric acid in urine, 607
**Murmur** and **Murmurs,** 288
aortic diastolic, in aortic regurgitation, 340
aortic, double, differentiation from rub in pericarditis, 357
auriculo-systolic, 289
not heard in auricular fibrillation, 315
cardio-pulmonary, 293
in hypertrophy, 320
causes of, 288
character of, 292
diastolic, 289
in acute endocarditis, 329
early, in rheumatic fever, 293
exocardial, 293
differentiation from valvular disease, 345
Flint's, in aortic regurgitation, 340, 346
functional, 292, 345
hæmic, 292, 345
in aneurysm, 301, 374
in aortic regurgitation, 290, 291, 340
in arterial aneurysm, 289
in arterio-venous aneurysm, 289
in malignant endocarditis, 332
in mitral stenosis, 288—291, 342
in patent ductus arteriosus, 355
in pulmonary regurgitation, 291, 292
mid-diastolic, in rheumatic fever, 293
pre-systolic, 289
or early diastolic, due to heart block, 305
significance of, 293
systolic, 288, 289
exocardial, 293
in acute endocarditis, 329, 330
in aortic stenosis, 289, 339
in congenital heart disease, 355
in mitral regurgitation, 290, 342
in pulmonary stenosis, 344
in rheumatic fever, 293
in tricuspid regurgitation, 291, 343
to be disregarded, 293
tune of, 289
to-and-fro, in aortic regurgitation, 340
**Muscles,** atrophy of, progressive, 799
in beri-beri, 687
degeneration of, in acute poliomyelitis, 98, 99
diseases of, 910
parasitic diseases of, 912
re-education of, in acute poliomyelitis, 101
wasting of, in compression of cord, 768
**Muscular** atrophy, 914
progressive, differentiation from lead palsy, 682

**Muscular** dystrophy, progressive, 914
differentiation from progressive muscular atrophy, 801
distal type, 917
facio-scapulo-humeral type, 916
pseudo-hypertrophic type, 916
scapulo-humeral type, 916
exercise, effect on digestion, 408
exhaustion, 920
wasting in neuritis, 739
weakness in encephalitis lethargica, 834
work predisposing to malignant endocarditis, 330
**Musculospiral** paralysis, differentiation from lead palsy, 682
**Mustard** gas, poisoning by, 265
**Myalgia**, 910
**Myasthenia** gravis, 919
**Myasthenic** reaction, 920
**Myelin kidney**, 624
**Mycosis** fungoides, 1019
**Mydriatic** test, Loewi's, 503
**Myelitis**, 772
acute, differentiation from compression of cord, 769
infective, 773, 774
resembling spinal compression, 768
chronic, 775
differentiation from hæmorrhage into spinal cord, 777
Landry's paralysis, 803
neuritis, 742
spinal meningitis, 805
tabes, 792
in syphilis, 112, 113
syphilitic, 773, 776
differentiation from primary spastic paraplegia, 794
spinal tumour, 770
**Myeloblasts**, 521
in leukæmia, 535
in lymphocytic leukæmia, 535
**Myelocytes**, 521
basophil, 521
granular, 521
in leukæmia, 533
**Myelocytic** leukæmia, 533
**Myeloma** in vertebræ, 767
multiple, fracture of bones in, 674
**Myocardial** degeneration, 323
brown atrophy in, 323
fatty, 323
in pernicious anæmia, 526
fibroid, 324
heart block in, 304, 307, 324
in emphysema, 232
paroxysmal breathlessness in, 349
pulsus alternans in, 313
simple paroxysmal tachycardia in, 311
symptoms of, 325
**Myocarditis**, 322
acute, 322
chronic, fibroid degeneration in, 324
in rheumatic fever, 57
interstitial, fibroid degeneration in, 324
suppurative, 322
syphilitic, 328
treatment of, 350
**Myoclonus**, 730, 880
**Myoma** of skin, 1018
of stomach, 434
**Myopathy**, 914
*See also* Muscular dystrophy.
**Myositis**, 911
in gonorrhœal rheumatism, 73
ossificans, 912
**Myotonia** atrophica, 918
congenita, 917, 918
**Myxœdema**, 581
*See also* Thyroid gland.
**Myxo-sarcoma** in peritoneum, 517

**Nails**, affections of, associated with alopecia areata, 1016
dystrophy of, in latent tetany, 586
favus of, 956
tinea of, 958
**"Napkin rash,"** 929, 930
differentiation from congenital syphilis, 121
**Nasal** catarrh, acute, 196
mucous membrane, infection of, in primary asthma, 224
sinuses, infection of, paroxysmal headache due to, differentiation from migraine, 879
tube, use of, in cyclical vomiting, 418
in diabetic coma, 576
**Nascent** iodine in treatment of phthisis, 263
**Nasopharyngeal** ulceration in trypanosomiasis, 161
**Naso-pharynx**, primary focus of infection in influenza, 62
**Nauheim** treatment, 7, 327
**Nausea** following gassing, 265
in dyspepsia, 412
in gastroptosis, 417
in hypoglycæmia from insulin, 572
**Neck**, stiffness of, in cerebral meningeal hæmorrhage, 828
**Necrosis**, coagulative, 379
**Negri** bodies, 102
**Nematoda**, 466
**Neoarsphenamine**, 116
**Neodiarsenol**, 116, 118
**Neo-kharsivan**, 116, 118
**Neoplasms** causing neuritis, 739
Gye and Barnard's recent work on, 1020
mediastinal, 386
*See also* Mediastinal growths
of heart, 327
of liver, 491
of lung, 266
simulating pleurisy, 276
of peritoneum, 517
of skin, 1017
of tonsil, 398
*See also* Tumours, Carcinoma, Sarcoma, etc
**Neo-salvarsan** *See* Novarsenobenzol.
**Nephrectomy** in renal calculus, 660
**Nephritis**, 611
acute, 618
casts in, 620
in influenza, 63
in scurvy, 689
interstitial, 619
azotæmic type, 612, 622
chronic, colitis in, 450, 626
diarrhœa in, 440
interstitial, 627
parenchymatous or tubal, 624
course and cause of death in, 626
diet, rich in protein, in treatment, 627
salt free, in treatment, 627
differentiation from primary contracted kidney, 632
urine in, 625
complicating cholera, 181
scarlet fever, 30
embolic, in malignant endocarditis, 331
Gaskell's classification of, 619
glomerulo-tubular, 619
hæmorrhagic, 624
complicating glandular fever, 50
in arseno-benzol poisoning, 123
hydræmic type, 612, 622
in pneumonia, 241
lardaceous disease of kidney and, 646
metastatic, 639
secondary to septic tonsils, 398
Sippy's diet causing, 426
suppurative, 637
toxic, 619
*See also* Bright's disease, Kidney, etc.
**Nephrolithiasis**, 656
**Nephroptosis**, 416
**Nephrostaxis**, 647
**Nephrotomy** in renal calculus, 660
in treatment of pyonephrosis, 637
**Nerve** and **Nerves**, circumflex, lesions of, 755
brachial plexus, lesions of, 757
cranial, disorders of, in hysteria, 905
examination of, 726
deafness, 728, 750
diseases of, 738*
eighth, examination of, 728
lesions of, 750
eleventh or spinal accessory, lesions of, 753
external popliteal, lesions of, 760
fibres, effect of diphtheria on, 67
fifth or trigeminal, examination of, 727
lesions of, 747

**Nerve** and **Nerves**, internal popliteal, lesions of, 760
median, lesions of, 757
musculo-spiral, lesions of, 756
ninth or glosso-pharyngeal, examination of, 728
lesions of, 752
olfactory, lesions of, 743
optic, lesions of, 744
*See also* Optic neuritis.
phrenic, lesions of, 755
posterior thoracic, lesions of, 755
roots, posterior, degeneration of, in diabetes, 569
sciatic, lesions of, 760
secondary degeneration of, 739
seventh or facial, examination of, 728
lesions of, 748
spinal, lesions of, 754
tenth, pneumogastric or vagus, lesions of, 752
third, fourth and sixth, lesions of, 746
twelfth or hypoglossal, lesions of, 753
ulnar, lesions of, 756
upper cervical, lesions of, 754
vestibular, lesions of, associated with vertigo, 751
**"Nervous breakdown,"** 891
**Nervous** dyspepsia, 412
exhaustion in anxiety neurosis, 896
system, anatomy and physiology of, 699
central, development of, 701
clinical examination of, 722
diseases of, 698
effects of pyrexia on, 20
in psychopathology, 891
involuntary, 720
cranio-bulbo-sacral, 720
functional disturbances of, 720
sympathetic, 720
motor, examination of, 730
**Neuralgia**, 884
complicating diabetes, 569
in lesions of fifth nerve, 748
intercostal, differentiation from biliary colic, 500
pleurisy, 275
trigeminal, 885
**Neurasthenia**, 886
following gassing, 265
in encephalitis lethargica, 834
**Neurasthenic** symptoms in anxiety neurosis, treatment of, 898
**Neuritis**, 738
alcoholic, 679, 741
differentiation from beri-beri, 688
general paralysis, 863
arsenical, 684, 741
associated conditions of, 742
brachial, 708
complicating diabetes, 569
diabetic, 742
family hypertrophic, 742
gouty or rheumatic, 740
interstitial, 739
in beri-beri, 687
in dysentery, 168
in Mediterranean fever, 172
in pneumonia, 241
lead, 680
local, causing neuralgia, 885
multiple or peripheral, 740
ataxic, 741
benzoin test in, 738
differentiation from acute poliomyelitis, 100
Landry's paralysis, 803
progressive muscular atrophy, 801
tabes, 792
in diabetes, 569
in dysentery, 165
optic, 744
in suppurative meningitis, 844
parenchymatous, 739
retrobulbar, 744
in diabetes, 570
sciatic, 761
differentiation from tumours of femur, 762
*See also* Polyneuritis and Nerves.
**Neuroblastoma** of suprarenal capsules, 592
**Neurofibromas**, 743, 856
**Neuroma**, 743
of skin, 1018
of ulnar nerve, 756
traumatic, 757
**Neuronophagia**, 98
in encephalitis lethargica, 832
**Neurosis**, anxiety, 894
**Neuro-syphilis**, benzoin test in, 738
**Night-blindness** in scurvy, 689
**Night-sweating** in phthisis, 263
**Nitric** acid test for albuminuria, 614
**Nitrobenzol**, workers dealing with, methæmoglobinæmia in, 541
**Nitrogen** excretion increased in pyrexia, 20
**Node**, auriculo-ventricular, 285
sino-auricular, 284
**Nodes**, Aschoff's, 322
in glanders, 137
**Nodular** leprosy, 135
**Nodules**, rheumatic, 56
subcutaneous, in rheumatism in children, 58
**Noma**, 394
**Nomogram** relating basal metabolism, 559, 560
**Normoblasts**, 520
**Notch**, in gastric ulcer, 425
**Notification** of infectious diseases, 14
**Novarsenobenzol**, 116, 118
in disseminated sclerosis, 784
in rat-bite fever, 144
in relapsing fever, 148
in syphilis, 116
**Novarsenobillon**, 116, 118
in syphilitic myelitis, 776
**Nucleo-protein**, euglobulin, in urine, 616
in urine, 614
**Numbness** in anxiety neurosis, 896
in multiple neuritis, 741
**Nummular** sputum, 252
**Nystagmus**, 727, 763
cause of, 716
in albinism, 1011
in alcoholic neuritis, 741
in disseminated sclerosis, 782
in encephalitis lethargica, 832
in Friedreich's ataxia, 797
in infantile cerebral diplegia, 839
in rickets, 693
in syringomyelia, 786
in tumours of cerebellum, 855
vertical, 716

**Obesity**, 598
associated conditions, 599
diabetes mellitus and, 562
dyspepsia of, 411
in fatty liver, 488
**Obstruction** at valvular orifices causing murmurs, 288
*See also* Stenosis.
bronchial or tracheal, in syphilis of lung, 264
due to gastric ulcer, 423
intestinal, 457
acute, symptoms of, 460
chronic, symptoms of, 461
effects of, 459
in carcinoma, 456
**Occipital** cortex, lesions of, 745
lobe, localisation of function in, 815
**Occult** blood in stools, 408
examination of, in dyspepsia, 413
*See also under* Fæces.
**Ochronosis**, 1011
**Œdema**, angelo-neurotic, 383
*See also* Angeio-neurotic œdema.
cachectic, complicating diabetes, 570
cardiac, in chronic bronchitis, 219
in failure of heart, 337
in simple paroxysmal tachycardia, 311
in tricuspid regurgitation, 343
differentiation from sclerema neonatorum, 1009
formation, test for, 610
hunger, 697
in congenital heart disease, 354
in heart disease, treatment of, 349
in mediastinitis, 384
in primary contracted kidney, 632
in trichiniasis, 913
of larynx, 200
of legs in beri-beri, 687
of lungs in hypoglycæmic coma, 576
renal, in chronic parenchymatous nephritis, 625
in acute nephritis, cause of, 620
in lardaceous disease of kidney, 646
in suppurative nephritis, 638
treatment of, 639
**war**, 697

**Oertel's** method in treatment of fatty overgrowth of heart, 327
**Œsophageal** pain in angina, 363
**Œsophagectasia,** 403
**Œsophagitis,** 401
**Œsophagoscope,** use of, 402
**Œsophagus,** carcinoma of, 401
clubbing of fingers in, 673
cicatricial stricture of, 402
dilatation of, 402
idiopathic, 403
diseases of, 401
epithelioma of, causing obstruction of bronchi, 230
in achalasia, 402, 403
obstruction of, 401
in carcinoma of stomach, 431
in mediastinal neoplasms, 386
pressure diverticula of, 404
spasmodic stricture of, 402
traction diverticula of, 404
*Oidium albicans,* 394
*Onchomelania japonicum,* 650
**Onchosphere,** 467
**Onychomycosis,** 958
**Ophthalmia** complicating relapsing fever, 147
**Ophthalmic** reaction, Calmette's, 257
in hay fever, 225
**Ophthalmoplegia,** 746
**Opisthotonus** in cerebro-spinal fever, 95
in spinal meningitis, 805
in tetanus, 105
in tetany, 585
**Opotherapy** in treatment, 5, 558
*Oppler-Boas bacilli* in vomit in chronic dilatation of stomach, 429
**Opsonins,** 17
**Optic** atrophy, 725
due to pressure from tumours, 594
in acute nephritis, 622
in arteriosclerotic retinitis, 368
in diabetes, 570
in infantile cerebral diplegia, 839
in lead poisoning, 681
in syphilis, 113
in syphilitic meningitis, 846
in tabes, 791
primary and secondary, 745
chiasma, lesions of, 745
disc, œdema of, in pachymeningitis interna hæmorrhagica, 848
neuritis, 744
in delirium tremens, 678
in lead poisoning, 681
in malignant endocarditis, 331, 333
in suppurative meningitis, 844
in tuberculous meningitis, 130
*See also* Papillitis, Papillœdema.
radiations, lesions of, 745
thalamus, syndrome of, 814
tract, lesions of, 745
**Oral** sepsis, 392
alopecia areata and, 1016
eczema and, 984
in dyspepsia, 413
in pernicious anæmia, 527
in rheumatoid arthritis and osteoarthritis, 668
malignant endocarditis and, 330
**Orchitis** complicating parotitis, 46
filarial, 555
in Mediterranean fever, 173
**Orcin** test for pentose in urine, 567
**Organo-therapy** in treatment, 5
*Ornithodorus moubata,* 145
*talaje,* 145
*venezuelensis,* 145
**Orthodiagraphic** measurements of heart, 296. *See also* X-rays.
**Orthopnœa,** 188
in emphysema, 233
in mitral disease, 342
in myocardial degeneration, 325
in œdema of lungs, 236
venesection for, 348
**Orthotonus** in cerebro-spinal fever, 95
**Osler's** spots, 333
**Osteitis** deformans, 675
**Osteo-arthritis,** 668
differentiation from osteitis deformans, 676
**Osteogenesis** imperfecta, 675
**Osteomalacia,** 673
differentiation from osteitis deformans, 676
fracture of bones in, 674
hunger, 695
tetany in, 585
**Osteomyelitis,** acute, from pyæmia, 51
differentiation from rheumatic fever, 59
in gonorrhœal rheumatism, 73
**Osteophytes** in rheumatoid arthritis, 669
**Osteoporosis,** 674
**Otitis** media, abscess of brain and, 836
adenoids and, 399
complicating enteric fever, 86
measles, 35
scarlet fever, 30
whooping-cough, 49
**Otosclerosis** causing tinnitus, 750
**Otorrhœa.** *See* Otitis.
impetigo contagiosa associated with, 927
**Ovaries,** cyst of, differentiation from pancreatic cyst 508
lardaceous disease of, 489
resembling chronic peritonitis, 513
simulating ascites, 478
tumour, causing jaundice, 476
differentiation from cancer of kidney, 653
hydronephrosis, 645
movable kidney, 655
**Over-exertion,** causing gastroptosis, 416
**Overwork,** dyspepsia in, 412
**Oxygen** apparatus, Haldane's, 64, 234
chamber, treatment in, of after-effects of "gassing," 266
deficiency of, in arterial blood, in pneumonic influenza, 62
in heart disease, 348
intratracheal insufflation of, in treatment of tetanus, 108
in treatment of broncho-pneumonia, 245
of chronic bronchitis, 218, 220
of emphysema, 234
of erythrocytosis, 539
of lobar pneumonia, 242
of methæmoglobinæmia, 541
of œdema of lungs, 237
of pneumonic influenza, 64
of pneumothorax, 282
of poisoning by irritant gases, 265
*Oxyuris vermicularis,* 470

**Pachydermia** laryngis, 202
**Pachymeningitis,** cerebral, 847
external, 807
interna hæmorrhagica, 807, 847
internal, 806
**Paget's** disease of bone, 675
**Pain** after gastro-jejunostomy, 427
at back of neck in hyperpiesia, 370
colicky, in carcinoma of intestine, 456
"hunger," 410
in duodenal ulcer, 424
hysterical, 905
in angina pectoris, 361
in carcinoma of stomach, 432
in cerebral abscess, 837
in chest following gassing, 265
in gastric ulcer, 424
treatment of, 427
in limbs in small-pox, 40
in neuritis, 739
in suppurative meningitis, 844
neuralgic, differentiation from angina, 363
œsophageal, in angina, 363
præcordial, differentiation from angina, 363
referred, in angina, 363
sensibility to, 729
substernal, in aortic regurgitation, 341
**Palpitations,** 307
following gassing, 265
in adherent pericardium, 359
in anxiety neurosis, 896
in hypoglycæmia from insulin, 572
in obesity, 599
**Palsy.** *See* Paralysis.
**Pancreas,** accessory, 434
carcinoma of, 507
concretions in, 507
cysts of, 508

**Pancreas,** deficiency of external secretion of, 503
of internal secretion of, 504
diseases of, 503
functional tests of, 503
in diabetes mellitus, macroscopical changes, 567
tumour of, causing jaundice, 476
differentiation from movable kidney, 656
**Pancreatic** deficiency in cœliac disease, 448
**Pancreatitis,** acute hæmorrhagic, 505
differentiation from intestinal obstruction, 462
simulating peritonitis, 511
chronic, 506
from gall stones, 500
with cholecystitis, 496
complicating mumps, 47, 506
hæmorrhagic, causing pancreatic cysts, 508
laparotomy in, 506
suppurative, 496, 505
**Papillitis,** 744
in acute nephritis, 622
**Papilloedema,** 726, 744
in acute nephritis, 622
in cerebral tumours, 853
in pachymeningitis interna hæmorrhagica, 848
in syphilitic meningitis, 846
**Papilloma** of choroid plexus, 850
of larynx, 206
**Papular** erythema, differentiation from small-pox, 42
skin eruptions, 995
**Papulo-erythematous** eruption in arseno-benzol poisoning, 123
**Parabasal** body in trypanosomes, 158
**Paracentesis** of chest, 277, 282
of pericardium, 359, 360
**Parakeratosis,** 985
variegata, 997
**Paralysis** agitans, 840
resemblance of chronic manganese poisoning to, 684
symptoms like, in encephalitis lethargica, 834
diphtheral, 68
facial, 749
in relapsing fever, 146
of respiratory movements in botulism, 448
progressive bulbar, 809
pseudo-bulbar, 811
**Paramelitensis** fever, 173
*Par meningococci,* 94
**Paramyoclonus** multiplex, 880
**Paraplegia,** senile, 804
spastic, primary, 793
in compression of cord, 768
secondary, 794
**Parasites,** 10
animal, diseases of skin due to, 964
in heart, 328
in kidney, 648
in muscles, 912
in urinary organs, 648
**Parasiticides,** 960
**Parathyroid** glands, diseases of, 584
in simple paroxysmal tachycardia, 310
in treatment when ionised calcium in serum is low, 584
*See also* Tetany.
**Paratyphoid** fevers, 92
differentiation from food poisoning, 448
**Parietal** lobe, localisation of function in, 814
**Parkinson's** disease, 840
**Parotitis,** 44
differentiation from plague, 185
in pneumonia, 241
secondary, 400
*See also* Mumps.
**Paroxysmal** hæmoglobinuria, 540
tachycardia, 309
simple, 310
**Pasteur's** dry cord method, 104
**Pathogenic** bacteria, 10
**Pectoriloquy,** 195
**Pediculosis,** 964. *See also* Phtheiriasis.
*Pediculus capitis,* 964
*pubis,* 965
*vestimentorum,* 965
**Pel-Ebstein's** disease, 553
**Peliosis rheumatica,** 57, 542
**Pellagra,** 695
**Pelvic** lesions, differentiation from sciatic neuritis, 762
organs, inflammatory lesions of, causing acute peritonitis, 508
veins, thrombosis of, in pulmonary embolism, 270
viscera, operations on, pulmonary embolism and thrombosis after, 269
**Pemphigus** acutus malignus, 1002
contagiosus tropicus, 927, 1002
differentiation from small-pox, 42
foliaceus, 1002, 1004
gestationis, 1005
leprosus, 1002
neonatorum, 928, 1002
differentiation from congenital syphilis, 121
syphiliticus, 1002
vegetans, 1004
vulgaris, 1003
**Pentose** in urine, 567
**Peptic** ulcer, 421
active, 422
acute, morbid anatomy of, 422
nature of, 421
callous, 422
chronic, 422
nature of, 421
differentiation from appendicitis, 455
carcinoma, 433
*See also* Stomach, ulcer of.
**Peptone** in treatment of rheumatoid arthritis, 672
**Peptones** in urine, 616
**Percussion,** auscultatory, 196
in massage, 7
**Perforation** in carcinoma of stomach, 433
of peptic ulcer, 424, 426
acute pancreatitis simulating, 506
chronic, 422
or duodenal ulcers, differentiation from intestinal obstruction, 462
treatment of, 427
of typhoid ulcer, 84, 85, 89, 91
**Peribronchial** phthisis, 248
**Pericardial** effusions, 360
differentiation from hypertrophy, 320
simulating pleurisy, 276
rub, 356
**Pericarditis,** 355
differentiation from valvular heart disease, 315
empyema in, 277
hæmorrhagic, 356
in chronic parenchymatous nephritis, 625
in lobar pneumonia, 241
in rheumatic fever, 57
suppurative, 356
tuberculous, 356
**Pericardium,** adherent, 359
paracentesis of, 359, 360
**Perichondritis** of larynx, 205
in enteric fever, 85
**Perihepatitis,** 501
causing ascites, 477, 486
in splenic anæmia, 530
in syphilis of liver, 490
**Perinephric** abscess, 639
causing peritonitis, 509
**Perineuritis** in gonorrhœal rheumatism, 73
**Periostitis** in congenital syphilis, 121
in gonorrhœal rheumatism, 73
**Peripheral** neuritis. *See* Neuritis.
**Perisplenic** abscess, 512
**Perisplenitis,** 501, 550
in splenic anæmia, 530
**Peristalsis,** visible, in Hirschsprung's disease, 466
in intestinal obstruction, 461
**Peritoneum,** adhesions of, 516
diseases of, 508
effusions of, 516
new growths in, 517
wounds of, peritonitis following, 509
**Peritonitis,** acute, 508
circumscribed, 511
differentiation from intestinal obstruction, 462
chronic, 513
differentiation from pneumonia, 241
suppurative nephritis, 638
gonococcal, 509
in carcinoma of liver, 492
of stomach, 431

**Peritonitis** in cholecystitis, 496
in chronic parenchymatous nephritis, 625
in gastric ulcer, 422
in phthisis, 255
in pneumonia, 241
in typhoid fever, 84, 85
pneumococcal, 509, 511
suppurative, in infantile enteritis, 445
tuberculous, 514
adhesive, fibrinous or plastic type, 514
ascitic type, 514
caseous type, 514
causing ascites, 477
differentiation from enteric fever, 88
tabes mesenterica type, 514
**Pernicious** anæmia. *See* Anæmia, pernicious.
**Peroneal** muscular atrophy, 798
**Personality,** dual, in hysteria, 904
**Persuasion** in treatment of hysteria, 908
**Pertussis** *See* Whooping-cough.
**Pes** cavus in Friedreich's ataxia, 798
in peroneal muscular atrophy, 798
**Petechiæ**, 924
**"Petit mal,"** 870. *See also* Epilepsy.
**Pétrissage** in massage, 7
**Peyer's** patches, infiltration of, in enteric fever, 81, 84
ulceration of, in paratyphoid fevers, 93
**Pfeiffer's** bacillus, 61
**Phagocytes**, 16
**Phagocytosis**, 10, 16
**Pharyngitis,** catarrhal, 395
chronic, 390
granular, 399
ulcerative, complicating enteric fever, 85
**Phenolsulphonepthalein** test for renal function, 610
**Phenylhydrazine** test for sugar, 567
**Phlebitis,** 378
accompanying gout, 665
**Phlebotomus** fever, 148
**Phosgene,** poisoning by, 265
**Phosphates** in urine, 605
**Phosphorus,** poisoning by, fatty degeneration of heart in, 323
simulating acute yellow atrophy of liver, 483
**Phtheiriasis,** 964
differentiation from Addison's disease, 591
prurigo, 997
**Phthisis,** 248
and lardaceous disease of kidney, 646
basal, primary, 251
causing fatty liver, 488
chronic, differentiation from bronchiectasis, 224
chronic bronchitis, 220
differentiation from Addison's disease, 591
chronic pneumonia, 246
glanders, 137
hydatid disease of lung, 268
neurasthenia, 888
pneumothorax, 281
dyspepsia of, 411
fatty degeneration of heart in, 323
fibroid, 250, 251, 256
following mustard gas poisoning, 266
gangrene of lung in, 245
hilum, 248, 256
œdematous laryngitis in, 200
peribronchial, 248
pleurisy and, 271
pneumonic, 256
acute, 249
pneumothorax in, 278
tricuspid regurgitation in, 343
*See also* Tuberculosis.
**Phylaxis,** antigen in, 17
**Picric** acid test for albuminuria, 614
**Pigmentation** of mucous membrane in Addison's disease, 591
**Pigmentation** of skin, alterations in form of, 1010
in Addison's disease, 590
in chronic arsenical poisoning, 684
in exophthalmic goitre, 580
in Hodgkin's disease, 553
in pellagra, 696
in pernicious anæmia, 528
in rheumatoid arthritis, 670
in syphilis, 111
**Piles.** *See* Hæmorrhoids.
**Pineal** body, tumours of, 598
**gland, diseases of,** 597
adiposity in, 599
**Pineal** gland, symptoms due to tumours of, 815
**Pituitary** extract, 593
in treatment, 596
of diabetes insipidus, 596
gland, changes in, in adiposis dolorosa, 599
diseases of, 592
acromegaly, 594
posterior lobe, cause of diabetes insipidus, 596
hypertrophy or adenomatous enlargement of, 593
increase in sugar in blood due to, 564
pressure on, by tumours outside, 593
neighbourhood symptoms of enlargements, 593
symptoms due to tumours of, 815
tumours of, 593
**Pityriasis** capitis, 934
circinata, 934
differentiation from pityriasis rosea, 988
rosea, 988
differentiation from macular syphilide, 110
rubra, 992
differentiation from psoriasis, 991
pilaris, 994
simplex, 934
**Plague,** 183
bubonic, 184
pneumonic, 183, 184
purpura in, 542
septicæmic, 184
*Planorbis*, intermediate host of *Schistosoma mansoni*, 650
**Plasma** bicarbonate. *See* Alkali reserve
chloride, low, in tetany, 585
*Plasmodium falciparum*, 150
*malariæ*, 150
*vivax* in malaria, 150
**Platelet** counting, 520
**Platelets,** absence of, in aplastic anæmia, 529
diminution in number of, in influenza, 63
in hæmophilia, 544
in purpura, 542
in typhus fever, 75
**Pleura,** diseases of, 271
**Pleural** reflex syncope, 278
**Pleurisy,** 271
chronic pneumonia following, 245
complicating failure of heart, 338
diaphragmatic, 273, 274
differentiation from biliary colic, 500
pneumonia, 241
dry, 271
gonococcal, 73
in chronic parenchymatous nephritis, 625
in hydatid disease of lung, 268
in lobar pneumonia, 238, 241
in phthisis, 255
in pulmonary embolism, 270
tuberculosis, 250
in rheumatic fever, 57
interlobar, 273, 274
mediastinal, 273, 274
loculated, 272
pulsating, 274
with effusion, 271
with effusion, differentiation from chronic pneumonia, 246
**Pleuritic** rub, 195, 273
**Pleurodynia,** 910
differential diagnosis from pleurisy, 275
**Pleuro-pericardial** friction, 195, 275
**Pleuro-pneumonia,** 238
**Plumbism,** 680
**Plummer's** bag, use in achalasia of cardia, 404
**Pluri-glandular** syndromes, 558, 601
**Pneumococcal** endocarditis and pneumococcal meningitis combined, 332
infections, 72
empyema in, 277
pericarditis in, 355
pleurisy in, 271
peritonitis, 509, 511
septicæmia, 72, 238
causing peritonitis, 509
*Pneumococcus*, 72, 238
and myelitis, 772
in acute encephalitis, 830
endocarditis, 329
in broncho-pneumonia, 243

*Pneumococcus* in gangrene of lung, 247
in infective endocarditis, 331
in malignant endocarditis, 330
in meningitis, 843
in pericarditis, 356
in peritonitis, 510
in pleurisy, 271
in pulmonary tuberculosis, 248
in rheumatoid arthritis, 668
types of, 72
**Pneumo-hydropericardium,** 360
**Pneumokoniosis,** 126, 245
**Pneumonia,** abscess of lung from, 245
actinomycosis causing, 142
acute, differentiation from typhus, 78
œdema of lungs with, 236
cerebral abscess in, 837
chronic, 241, 245
complicating acute myelitis, 774
bronchiectasis, 224
cholera, 181
failure of heart, 338
relapsing fever, 147
delirium tremens and, 678
differentiation from acute poliomyelitis, 100
influenza, 63
liver abscess, 171
pleurisy, 275
yellow fever, 177
empyema in, 277
Friedländer, 243
hypostatic, 243
herpes facialis in, 1000
in chronic parenchymatous nephritis, 626
in hydatid disease of lung, 268
in laryngeal perichondritis, 205
in perinephric abscess, 640
infantile convulsions in, 876
inhalation, 243
lobar, 237
caseous, 249
causing bronchiectasis, 222
chronic pneumonia following, 245
gangrene of lung following, 247
pneumococcus in, 72
lobular, 243
malignant endocarditis in, 330
pleurisy in, 271
pneumococcal, 238
pneumothorax in, 279
pulmonary embolism and thrombosis in, 269
scrofulous, 256
septic, 243
traumatic, 237
white, 264
*See also* Broncho-pneumonia.
**Pneumonic** influenza, 62
phthisis, acute, 249, 256. *See also* Phthisis
**Pneumopericardium,** 360
**Pneumothorax,** 279
artificial, in bronchiectasis, 224
in pleural effusions, 277
in treatment of phthisis, 262
partial, in treatment of phthisis, 263
closed, 280
in lobar pneumonia, 241
in neoplasms of lung, 267
in pulmonary tuberculosis, 250, 255
open, 280
resulting from foreign body in bronchi, 230
simulating subphrenic abscess, 513
valvular, 280
**Poikiloblasts,** 520
**Poikilocytes,** 520
**Poisoning,** arsenical, chronic, 683
arseno-benzol, 473, 777
by irritant gases, 265
œdema of lungs from, 236
by nitrobenzol, causing methæmoglobinæmia, 541
food, 445
lead, 680
manganese, chronic, 684
mercurial, chronic, 683
trinitrotoluene, causing acute yellow atrophy of liver, 481
*See* Alcoholism.
**Poisons** and nephritis, 619
bacterial, causing jaundice, 476
causing multiple neuritis, 740
chemical, causing jaundice, 476
**Polariscope** in test for sugar, 567
**Polioencephalitis,** 830
acute, 97
**Poliomyelitis,** acute, 97
bulbar paralysis and, 811
differentiation from cerebro-spinal fever, 97
chronic myelitis, 775
encephalitis lethargica, 835
hæmorrhage into spinal cord, 778
Landry's paralysis, 803
progressive muscular atrophy, 801
progressive muscular dystrophy, 917
rheumatic fever, 59
rickets, 694
scurvy, 690
tuberculous meningitis, 132
**Pollen,** subcutaneous injection of extracts of, in treatment of hay fever, 228
**Polychromasia,** 520
in scurvy, 689
**Polycystic** kidney, 492, 653
**Polycythæmia** rubra, 522, 537
chronic bronchitis with, 219
following "gassing," 265
hypertonica, 538
in cholera, 180
in congenital heart disease, 353
in hyperpnœa, 369
with splenomegaly, 538
**Polygraph,** 302
advantages of, 304
**Polymorphonuclear** cells, 521
increased, in myelocytic leukæmia, 533
leucocytes, excess of, in pleurisy, 276
**Polyneuritis,** acute febrile, 742
differentiation from subacute combined degeneration, 742, 796
*See also* Neuritis, multiple
**Polyorrhomenitis,** 501
**Polypi** in ulcerative colitis, 451
intestinal, intussusception and, 465
**Polypnœa,** 188
**Polyuria,** 603
in diabetes mellitus, 562
in polycystic kidney, 654
in primary contracted kidney, 631
**Pompholyx,** eczematous ringworm resembling, 958
**Pons Varolii,** hæmorrhage into, 828
symptoms of lesions of, 815
**Portal** pyæmia, 51
as cause of abscess of liver, 479
system, relief of, in cirrhosis, through collateral circulation, 485
vein, thrombosis of, 502
**Position,** sense of, 729
examination of, in compression of cord, 768
**Posture,** examination of, 736
in hysteria, 906
**Post-mortem** wart, 126
**Post-tussive** suction, 195
in phthisis, 254
**Potassium** iodide, in treatment of actinomycosis, 143
in treatment of leprosy, 136
in treatment of syphilitic myocarditis, 350
poisoning by, purpura in, 542
**Præcordium,** uncomfortable sensation in anxiety neurosis, 896
**Precipitin** reaction in hydatid disease of liver, 495
**Precipitins,** 16
**Precocity** in hypergenitalism, 598
**Pregnancy,** albuminuria of, 661
dyspepsia in, 411
chorea and, 864
glycosuria complicating, 575
heart disease and, 350
influence on prognosis of phthisis, 259
lead poisoning and, 682
nephritis and, 619
osteomalacia and, 673
predisposing to tetany, 584
pyelitis and, 635
vomiting in, 418
**Premature** heart beats, 307
*See also* Systoles, extra.
**Premycosic** erythrodermia, 993
**Pressure** in massage, 7
**Pre-systolic murmurs,** 289
*See also* Murmurs.
**Price-Jones** blood cell distribution curve, 521

**Prickly** heat, 945
**Primary** contracted kidney, 366, 630
lateral sclerosis, 793
secondary infections in, 631
**Progeria,** 600
**Proglottides,** 467
**Progressive** bulbar paralysis, 809
lenticular degeneration, 840
muscular atrophy, 799
differentiation from progressive muscular dystrophy, 917
infantile hereditary, 801
in syringomyelia, 784
resemblance of lead poisoning, to, 682
muscular dystrophy, 914
**Proptosis** in exophthalmic goitre, 580
**Prostrate,** enlarged, cause of hydronephrosis, 643
suppurative pyelo-nephritis associated with, 638
**Protein** diet, high, causing albuminuria, 661
excess of, in cerebro-spinal fluid, 769
testing in allergic state, 225
therapy in asthma, 229
**Protein-sensitisation,** eczema and, 984, 987
**Protein,** specific dynamic action of, 599
shock therapy in rheumatoid arthritis, 672
**Proteins,** foreign, sensitiveness to, dyspepsia in, 412
**Protozoa,** 10
diseases of skin due to infection with, 962
**Prurigo,** 965, 996
senilis, 965
**Pruritus,** body louse causing, 965
forms of, 997
differentiation from prurigo, 996
in jaundice, 474
in psoriasis, 989
**Pseudo-angina,** 363
**Pseudo-bulbar** paralysis, 811
**Pseudo-hermaphroditism,** female, 592
**Pseudo-lævulose** in urine, 567
**Pseudo-leukæmia,** 551
**Pseudo-myxoma** peritonei, 517
**Pseudo-pelade,** differentiation from alopecia areata, 1016
**Pseudo-tuberculosis** following mustard gas poisoning, 266
**Pseudo-uræmia,** eclamptic, 621
**Psilosis,** 448
**Psittacosis,** 93
**Psoas** abscess causing peritonitis, 509
**Psoriasis,** 989
differentiation from lupus erythematosus, 972
pityriasis rosea, 988
pityriasis rubra, 991
rupia, 111
squamous syphilide, 111
palmaris in syphilis, 112
**Psychasthenia** *See* Anxiety Neurosis
**Psycho-analysis** in treatment of hysteria, 909
**Psychopathology,** anxiety neurosis, 894
conditioned reflex, 892
emotional reaction, 891
unconscious mind, 892
**Psychology,** 889
**Psychoneuroses,** 889
**Psychopathology,** 891
**Psychosis** polyneuritica, 741
**Psychotherapy** in hysteria, 907
in treatment of spasmodic torticollis, 883
**Ptomaine** poisoning, 447
**Ptomaines,** production of, by bacterial action, 11
**Ptosis,** 720, 726
bilateral, in tabes, 789
causes of, 747
in botulism, 448
**Puberty,** premature, 592
**Public** health, 4
**Puerperal** fever, malignant endocarditis in, 330
**Pulmonary** abscesses in melioidosis, 137
artery, embolism of, 379
and aorta, transposition of, 353
embolism and thrombosis, 269
infarcts, 269
œdema, acute, in rheumatic fever, 57
osteoarthropathy, hypertrophic, 672
regurgitation, 344
murmurs in, 291, 292
stenosis, 344
and deficient septum ventriculorum, association of, 354
murmurs in, 292
**Pulmonary** tuberculosis, 248
*See also* Phthisis.
**Pulse,** alternation of, in angina, 364
anacrotic, 299
in aortic stenosis, 339
anacrotic or bisferiens, in aortic stenosis, 340
dicrotic, conditions associated with, 299
differentiation from pulsus bisferiens, 299
hardness of, 300
conditions favouring, 301
hyperdicrotic, 299
incompressible, 300
in pyrexia, 20
pressure, 298
radial, 297
rate and rhythm, 299
ratio for estimating cardiac efficiency, 294
slow, in cerebral tumours, 853
in jaundice, 474
soft or compressible, 300
conditions favouring, 301
tidal or predicrotic wave, 299
venous, 301
auricular form, 302
ventricular form, 302
volume of, 298, 300
water-hammer, in aortic regurgitation, 340
**Pulses,** radial, inequality of, in aneurysm, 374
**Pulsus** alternans, 308, 313
differentiation from bradycardia, 317
in angina, 364
bigeminus, 308
differentiation from bradycardia, 317
bisferiens, differentiation from dicrotic pulse, 299
celer in aortic regurgitation, 340
irregularis perpetuus, 314
paradoxus, 300
in pericarditis, 357
tardus in aortic stenosis, 339
trigeminus, 308
**Punctate** basophilia, 520
**Puncture,** intra-ventricular, in cerebro-spinal fever, 96
*See also* Lumbar Puncture
**Pupil** reflexes, 726
**Pupils,** enlarged, in neurasthenia, 887
inequality of, 726
in aneurysm, 375
**Purin** bodies in diet, 664, 666
**Purpura,** 542
differentiation from scurvy, 690
small-pox, 41
fatty degeneration of heart in, 323
fulminans, 542
after scarlet fever, 31
hæmorrhagica, 542
in relapsing fever, 146
in measles, 35
possible identity of aplastic anæmia with, 529
rheumatica, 542
simplex, 542
variolosa, 39, 40
**Pus** in urine. *See* Pyuria.
**Pustular** eczema, differentiation from small-pox, 42
**Pustule,** in small-pox, 39, 40
malignant, 138
**Pyæmia,** 11, 51
abscess of lung from, 245
acute, differentiation from rheumatic fever, 59
cerebral abscess in, 837
complicating bronchiectasis, 224
differentiation from enteric fever, 88
glanders, 137
gout, 666
malaria, 154
suppurative nephritis, 638
empyema in, 272
in acute myelitis, 774
malignant endocarditis in, 330, 334
pneumococcal, 241
portal, 51, 479
resemblance of malignant endocarditis to, 332, 334
**Pyæmic** abscesses of liver, 479
**Pyelitis,** 635
with bacilluria, 635
with renal calculus, 657
**Pyelography,** 603

**Pyelonephritis**, 637
in renal calculus, 657
pyelitis associated with, 635
suppurative, enlarged prostate associated with, 638
**Pylephlebitis**, 502
adhesive, 502
suppurative, as cause of abscess of liver, 479, 502
conditions simulating 503
in appendicitis, 452
resemblance to suppurative cholangitis, 496
**Pyloric** stenosis, 405, 428
as cause of dilatation of stomach, 428
congenital hypertrophic, 430
in carcinoma, 431
physical signs of, 428
**Pyogenic** organisms, secondary infection with, in Malta fever, 173
**Pyonephrosis** complicating bilharziasis, 650
differentiation from hydronephrosis, 645
in renal calculus, 657
**Pyo-pneumothorax**, 272, 279
in empyema, 272
in gangrene of lung, 247
in pulmonary tuberculosis, 250, 255
resulting from foreign body in bronchi, 230
**Pyorrhœa** alveolaris, 392
*See also* Oral sepsis
**Pyramidal** fibres in spinal cord, 707
**Pyrexia**, 11, 18
**Pyrosis**, 412
**Pyuria**, chemical test for, 636
in perinephric abscess, 640
in pyelitis, 636
in tubercle of kidney, 642
with renal calculus, 657

**Quarantine**, periods of, in various infectious diseases, 13
**Quartan** fever, 151
**Quinidine**, hyperthyroidism and, 317
in auricular fibrillation, 316
in auricular flutter, 313
intolerance of, 317
use of digitalis previous to, 317
**Quinine** causing skin eruptions, 980
causing tinnitus, 750
in treatment of malaria, 155
prophylactic use of, in malaria, 155
susceptibility to, in blackwater fever, 157
**Quinsy**, 396
**Quotient**, respiratory, effect of insulin on, 563
in basal metabolic rate, 560

**Rabies**, 102
**Rachitis**, 691
*See also* Rickets.
**Radium** in carcinoma of œsophagus, 402
in lympho-sarcoma, 388
in treatment, 6
of cheloid, 1017
of goitre, 581
of ichthyosis, 1007
of neoplasms of lung, 268
of xerodermia pigmentosa, 1010
**Râles**, 194
**Rash** caused by drugs applied externally, 980
ingested, 979
in acute poliomyelitis, 99
in cerebro-spinal fever, 96
in chicken-pox, distribution of, 15
in congenital syphilis, 121
in dengue, 178
in encephalitis lethargica, 832
in enteric fever, 82, 83, 84
in food poisoning, 447
in glanders, 137
in influenza, 63
in measles, 34
in paratyphoid fevers, 92
in rubella, 37
in scarlet fever, 29
in small-pox, 38, 39
in syphilis, 110
in typhus fever, 76
intestinal worms causing, 471
toxic, in cholera, 181
**Rat-bite** fever, 144
**Rats**, fleas from, conveyance of infection of bubonic plague by, 183
*Rattus norvegicus*, 174
*Rattus rattus*, plague spread by, 183
**Raynaud's** disease, 384
angina in, 363
diffused scleroderma associated with, 1008
in paroxysmal hæmoglobinuria, 540
**Reaction**, accelerated, in anaphylaxis, 18
immediate, in anaphylaxis, 18
myasthenic, 920
of blood, 547
acapnia in, 585
of degeneration, 733
in acute poliomyelitis, 99
in lead neuritis, 681
in neuritis, 739
in peroneal muscular atrophy, 799
partial, in progressive muscular atrophy, 799
pathetic, 738
precipitin, in hydatid diseases of liver, 495
Wassermann, 114, 117
**Reactions**, colloidal, in cerebro-spinal fluid, 738
skin, in allergic state, 225
to tuberculin, local and general, 257
**Recklinghausen's** disease, 1017
**Rectal** temperature, normal, 18
**Rectum**, adeno-papilloma of, 457
carcinoma of, 457
disturbances of reflexes of, 735
inflammatory strictures of, 457
polypoid growths of, 457
syphilis of, 457
**Red** corpuscles, fragility of, in congenital acholuric jaundice, 532
Cross shells, poisoning by, 265
gum, 1013
**Reduplicated** first sound in canter-rhythm, 287
first and second sounds in heart-block, 305
second sound in canter-rhythm, 287
mitral stenosis, 290
**Referred** pain in angina, 363
**Reflex** and **Reflexes**.
abdominal, 733
action in cerebrum, 716
activity in spinal cord, 713
centres in cerebellum, mid-brain, pons and medulla, 714
conditioned, 892
corneal, 728
diminution or loss of, in fifth nerve lesions, 748
cremasteric, 733
deep, 734
gastro-colic, 435
gastro-ileal, 445
in hysteria, 906
mass, 714
plantar, 734
pupil, 726
superficial, 733
**Refraction** of eye, error of, alopecia areata and, 1016
**Regurgitation** at valvular orifices causing murmurs, 287
of valves of heart, 355
tricuspid, 343
*See also* Heart, valvular disease of.
**Relapsing** fever, 144
differentiation from trench fever, 65
yellow fever, 177
enlargement of spleen in, 550
epistaxis in, 199
**Remissions** in disseminated sclerosis, 784
**Renal** asthma, 227
calculus, 656
formation of, 657
*See also* Kidney.
**Resection** of rib for empyema, 277
in pneumothorax, 282
**Respiration**, cog-wheel, in apical phthisis, 253
effect of pyrexia on, 20
inversion of, in broncho-pneumonia, 244
**Respiratory** exchange, increased in exophthalmic goitre, 579
in pyrexia, 22
movement, paralysis of, in botulism, 448
quotient, effect of insulin on, 563
system in psychopathology, 891
*See also* Metabolism.
**Rest** in treatment, 5

**Reticulo-endothelial** system, formation of bilirubin by, 475
**Retina,** changes in, in congenital syphilis, 122
detachment of, in chronic parenchymatous nephritis, 625
**Retinal** appearances in arterio-sclerosis, 367
hæmorrhages in anæmia, 524
in leukæmia, 535
in malignant endocarditis, 331, 333
in pernicious anæmia, 528
in splenic anæmia, 529
**Retinal** artery, thrombosis of, in arteriosclerosis, 368
**Retinitis,** arteriosclerotic, 368
in syphilis, 112
leukæmic, 535
renal, in secondary contracted kidney, 629
**Retraction,** systolic, 286
**Retropharyngeal** abscess, 400
**Re-vaccination,** 44
**Rheumatic** arthritis, chronic, 668
confusion with lightning pains of tabes, 788
fever, 55
acute myocarditis in, 322
anæmia in, 523
and chorea, 864
association with erythema multiforme, 969
auricular fibrillation in, 313
causing aortic disease, 338
differentiation from acute poliomyelitis, 100
glanders, 137
gonococcal infections, 74
gout, 666
Henoch's purpura, 543
Mediterranean fever, 173
pyæmia, 52
relapsing fever, 147
small-pox, 41
early systolic murmurs in, 293
endocarditis in, 328
heart block in, 304, 307
in children, 58
subcutaneous nodules in, 58
joint affections in, 56
malignant endocarditis in, 330
pericarditis in, 355
pleurisy in, 271
secondary to septic tonsils, 398
sweating in, 56
gout, 668
nodules, 56
**Rheumatism,** acute and subacute. *See* Rheumatic fever.
chronic, 668
gonorrhœal, 73
muscular, 910
differentiation from pleurisy, 275
tetanus, 107
nodose, 668
scarlatinal, 31
**Rheumatoid** arthritis, 668
association of psoriasis with, 990
**Rhinitis,** acute, 196
bronchitis and, 215
in hay fever, 227
chronic, 198
atrophic, 198
hypertrophic, 198
impetigo contagiosa associated with, 927
in congenital syphilis, 121
**Rhinorrhœa** in pituitary tumours, 594
**Rhinoscleroma,** 955
**Rhonchi,** 194
in acute bronchitis, 217
**Rhythm,** triple, in heart sounds, 288
**Rib,** resection of. *See* Resection of Rib
**Rickets,** 691
delayed, associated with chronic interstitial nephritis, 627
diet in, effect of excess of cereals in producing, 691
differentiation from congenital internal hydrocephalus, 860
enlargement of spleen in, 550
laryngismus stridulus in, 211
late or adolescent, 691
predisposing to tetany, 584
**Rickettsia** body, 64
*Rickettsia prowazeki,* 75
**Rickety** rosary, 692
**Rigidity** in paralysis agitans, 841
**Rigors** in cerebral abscess, 837
in diphtheria, 67
in erysipelas, 53
in malignant endocarditis, 332
in pyrexia, 21
**Ringworm,** 956
differentiation from alopecia areata, 1016
cheiropompholyx, 1002
eczematoid, of extremities, 958
fungi of, 958
**Rinné's** test, 725
**Risus** sardonicus in tetanus, 105
**Roentgen** rays. *See* X-rays.
**Romberg's** test in tabes, 789
**Roof-tiling** appearance in apical phthisis, 253
**Room,** disinfection of, 14
**Root** phthisis, 249
*See also* Phthisis.
**Rosacea,** 974
differentiation from lupus erythematosus, 972
in chronic alcoholism, 679
**Roseola,** 36
**Rotch's** sign, 356
**Rotheln,** 36
**Rothera's** test for ketosis, 568
**Round** worms, 466
**Rub,** pericardial, 294, 356
pleuritic, 195, 273
**Rubella,** 35
differentiation from scarlatina and measles, 36
scarlet fever, 31
quarantine period in, 13

**Sabouraud's** pastilles, 959
**Sachs-Georgi** reaction, 115
**Sacro-iliac** disease, differentiation from sciatic neuritis, 762
**St. Vitus's** dance, 864
**Salicylate** poisoning, 60
**Salicylates** causing tinnitus, 750
in gout, 667
in rheumatic fever, 56, 58, 59
**Salicyl-sulphonic** acid test for albuminuria, 614
**Saliva,** excess of, in aerophagy, 415
**Salivation** from mercurial treatment in syphilis, 120
in chronic mercurial poisoning, 368
**Salivary** glands, enlargement of, chronic, 400
**Salvarsan.** *See* Arseno-benzol.
**Sanatorium** treatment of phthisis, 260
**Sand-fly** fever, 148
**Sapræmia,** 51
**Saprophytes,** 10
**Sarcina,** infection by, 9
**Sarcoma,** fracture of bones in, 674
in osteitis deformans, 676
mediastinal, 386
of brain, 850
of lung, 268
of peritoneum, 517
of spinal cord, 772
of thyroid gland, 577
of tonsils, 398
**Sarcomatosis,** differentiation from idiopathic purpura, 543
purpura in, 542
*Sarcoptis hominis,* 964, 966
**Saturnine** encepha opathy, 681
**Scabies,** 966
differentiation from urticaria papulosa, 978
**Scarlatina,** 28
acute endocarditis in, 328
Dick test in, 32
differentiation from diphtheria, 69
measles and rubella, 36
smallpox, 41
empyema in, 272
malignant endocarditis in, 330
pleurisy in, 271
purpura in, 542
quarantine period in, 13, 32
rheumatic fever associated with, 55
**Scarlet** fever, 28
**Scarring** of skin in chicken-pox, 44
in smallpox, 42
**Schick** test, 70
*Schistosoma japonicum,* 650
*Schistoma hæmatobium,* 649
*Mansoni,* 650

**Schizocytes**, 520
**Schizogony**, 150
**Schizomycetæ**, infection by, 9
**Schott** treatment, 327
**Sciatica**, 761
**Sciatic** neuritis, 761
**Sclavo's** serum, 139
**Sclerema** in infantile enteritis, 445
neonatorum, 1008
**Sclerodactylia**, 1007
Raynaud's disease associated with, 381
**Sclerodermia**, 1007
circumscribed, 1008
diffused, 1007
Raynaud's disease associated with, 381
**Sclerosis**, amyotrophic, lateral, differentiation from compression of cord, 770
coronary, dilatation of heart in, 320
disseminated *See* Disseminated sclerosis.
hyperplastic, diffuse, of arteries, 366, 369
in primary contracted kidney, 631
of arteries, 366
in central nervous system, meaning of, 699
Monckeberg's, 367
primary lateral, 793
**Scoliosis** in Friedreich's ataxia, 798
in syringomyelia, 786
**Scorbutus**, 688
*See also* Scurvy.
**Scrofulodermia**, 947
**Scrum-pox**, 927
**Scurf**, 934
**Scurvy**, 688
differentiation from Henoch's purpura, 543
rickets, 694
fatty degeneration of heart in, 323
infantile, 690
differentiation from acute poliomyelitis, 100
purpura in, 542
**Sebaceous** cysts, 1014
glands, affections of, 1014
**Seborrhœic** dermatitis, 939
differentiation from pityriasis rosea, 988
eczema, 987
lesions, differentiation from squamous syphilide, 111
state, infections associated with, 931
skin diseases which it predisposes to, 933
**Sedimentation**, 16
**Seminal** emissions in anxiety neurosis, 895, 900
**Senile** degeneration of arteries, 366
blood pressure in, 366
**Senoran's** evacuator, 429
**Sense** of position, 729
loss of, in disseminated sclerosis, 782
of vibration, 730
**Sensibility**, cutaneous, impaired in multiple neuritis, 741
to light touch, 729
to pain, 729
to temperature, 729
and pain, loss of, in syringomyelia, 785
**Sensory** cortex, 814
**Sepsis**, focal, in diabetes, treatment of, 572
in fibrositis, treatment of, 911
in pernicious anæmia, treatment of, 528
oral. *See* Oral sepsis.
**Septicæmia**, 11, 50
anthrax, 139
complicating bronchiectasis, 224
differentiation from Mediterranean fever, 173
small-pox, 41
suppurative nephritis, 638
gonococcal, 73
in filariasis, 555
in melioidosis, 137
malignant endocarditis in, 330
pericarditis in, 355
pleurisy in, 271
pneumococcal, 72, 238
causing peritonitis, 509
terminal, causing acute endocarditis, 328
**Septic** rashes, differentiation from scarlet fever, 31
**Sera**, antitoxic and antibacterial, in treatment, 5
**Serous** membranes, pigmentation of, in Addison's disease, 590
**Serratus magnus**, paralysis of, 755
**Serum**, anti-anthrax, 139
anti-dysenteric, 165, 166
anti-icteroides, 178
antistreptococcal, in malignant endocarditis, 334
antitoxic, in diphtheria, 70
in scarlet fever, 32
diagnosis in acute poliomyelitis, 100
disease, 18
prophylactic, in gas gangrene, 109
in tetanus, 108
salvarsanised, in tabes, 793
therapeutics, 16
treatment in acute poliomyelitis, 101
in cerebro-spinal fever, 97
in enteric fever, 91
in erysipelas, 54
in gas gangrene, 109
in hæmophilia, 545
in lobar pneumonia, 242
in pyæmia, 52
in septicæmia, 50
in tetanus, 107
in typhus fever, 79
**Serum-globulin** in urine, 615
**Sexual** matters, fears in relation to, in anxiety neurosis, 895, 900, 901
**Shingles**, 998
*See also* Herpes zoster
**Shock**, anaphylactic, 18
diastolic, 286
histamine, 17
**Shortness** of breath *See* Breathlessness
**Siderosis**, 216
**Sigma** reaction, 115
**Sigmoidoscope** in diagnosis of carcinoma of colon or rectum, 457
of diverticulitis, 457
of dysentery, 165
use of, in muco-membranous colitis, 457
in ulcerative colitis, 451
**Signe de Musset** in aortic regurgitation, 341
**Silica**, cause of predisposition to tubercle, 126
**Silicosis**, 216
**Silver** salvarsan, 116, 118
**Singer's** nodules, 202
**Sinking** sensation of, in dyspepsia, pathology of, 410
**Sino-auricular** block, 307
bradycardia in, 318
node, 284, 304
**Sinus** arrhythmia, 304
vagal action causing, 318
irregularity or arrhythmia, 304
thrombosis in infantile enteritis, 445
**Sippy's** diet in peptic ulcer, 426
**Sitting** height *See* Body length
**Skin**, abscesses of, in melioidosis, 137
atrophic and dystrophic conditions of, 1007
bullous eruptions of, 1002
diseases of, 923
caused by external infection, 592
classified according to their morphology, 967
due to filter-passing organisms, 962
due to infection with animal parasites, 964
due to *infection* with higher fungi, 955
due to infection with protozoa, 962
hair and hair follicles, affections of, 1014
hyperkeratoses of, 1005
in psychopathology, 892
lesions of, in chronic parenchymatous nephritis 626
lymphangioma of, 1018
myoma of, 1018
neuroma of, 1018
new growths in, 1017
papular eruptions of, 995
pigmentation of *See* Pigmentation.
primary lesions of, 924
hæmorrhages, 924
papules, 924
pustules, 924
vesicles, 924
pruritus of, complicating diabetes, 569
reactions in allergic state, 225
staphylococcal and other infections associated with seborrhœic state, 931
streptococcal infections of, 926
toxic eruptions of, 979
tuberculosis of, 947
tuberculous ulcers of, 947
vesicular eruptions of, 998

**Skodaic** resonance, 191, 274
in hæmothorax, 279
**Sleeping** sickness, 158
**Small-pox,** 37
differentiation from chicken-pox, 45
measles, 35
relapsing fever, 147
scarlet fever, 32
yellow fever, 177
fatty degeneration of heart in, 323
modified, differentiation from chicken-pox, 45
purpura in, 542
quarantine period in, 13
variola hæmorrhagica pustulosa, 40
**Smell,** nerve tracts associated with, 713
tests for, 725
**Snuffles** in congenital syphilis, 121
**Sodium** benzoate in gout, 667
bicarbonate test in ketosis, 568
biurate in gout, 664
sulphate in treatment of dysentery, 165
**Soft** chancre, 954
palate, paralysis of, in diphtheria, 68
sore, 954
differentiation from plague, 185
on penis, differentiation from syphilis, 114
**Soldiers'** heart, 350
**Sore** throat, catarrhal, 395
sensation of, 398
**Southey's** tubes in chronic parenchymatous nephritis, 625
in treatment of dropsy, 627
**Spasm,** facial, 763
muscular, localised, 763
of glottis, 211
of jaw, 763
of limbs, reflex, treatment of, 771
of ocular muscles, 763
phonic, 212
**Spasmus** nutans in rickets, 693
**Special** senses, examination of, 724
nerve tracts associated with, 712
**Speech,** 718, 723
affection of, in Friedreich's ataxia, 797
defects of, in disseminated sclerosis, 782
in general paralysis of insane, 863
in progressive bulbar paralysis, abnormality of, 809
scanning, in cerebellar lesions, 715
*See also* Aphasia, dysarthria
**Specific** fevers, 23
**Sphincter,** cardial *See* Cardia
control, 735
impaired in compression of spinal cord, 768
**Sphincters,** loss of control of, in acute infective myelitis, 774
in combined subacute degeneration, 796
in disseminated sclerosis, 781
**Sphygmograph,** use of, 298
**Sphygmomanometer,** the, 300
**Spina** bifida, hydrocephalus associated with, 859
**Spinal** caries *See* Tuberculosis of spine
**Spinal** cord, arrangement of nerve cells and fibres in, 702
cauda equina, lesions of, 766
cervical enlargement of, lesions of, 765
complete lesion of, 722
compression of, 766
differential diagnosis of, 769
symptoms of, 768
treatment of, 770
diseases of, 764
fracture dislocation of, causing compression, 767
hæmorrhage into, 777
*See also* Brown Sequard syndrome.
inflammation of, 772
lesions of, differentiation from polyneuritis, 742
lumbar enlargement of, lesions of, 766
reflex activity in, 713
relation of spinal segments to spinous processes of vertebræ, 766
results of bilateral transverse lesion as affecting conduction, 765
results of unilateral transverse lesion as affecting conduction, 765
subacute combined degeneration of, 794
in pernicious anæmia, 527
differentiation from tabes, 792
**Spinal** cord, thoracic region, lesions of, 766
tumour and meningitis of, differentiation from progressive muscular atrophy, 801
tumours of, 772
meningeal hæmorrhage, 808
meningitis, 804
differentiation from spinal meningeal hæmorrhage, 808
tumour, differentiation from combined subacute degeneration, 797
**Spine.** *See* Vertebræ.
**Spirilla,** infection by, 9
*Spirillum minus,* 144
*morsusmuris,* 144
*Spirochæta duttoni,* 145
*obermeieri,* 145
*pallida,* 109
*recurrentis,* 145
**Spirochætes,** infection by, 9
in ulcerative stomatitis, 393
**Spirochætosis** ictero-hæmorrhagica, 173
causing acute yellow atrophy of liver, 481
differentiation from yellow fever, 177
jaundice in, 476
*Spironema berberum,* 145
*pallidum,* 109
in general paralysis of insane, 862
in spinal cord in tabes, 787
*pertenue* in framboesia, 124
*refringens,* 109
*venezuelense,* 145
**Spleen,** abscess of, causing acute peritonitis, 500
active congestion of, 550
amœbic abscesses in, 167
diseases of, 549
embolism in, 379
enlargement of, chronic, 550
differentiation from cancer of kidney, 653
movable kidney, 656
in acholuric jaundice, 531
in *Bilharzia japonicum* infection, 650
in endocarditis lenta, 333
in enteric fever, 82
in erythræmia, 538
in Hodgkin's disease, 552
in Kala-azar, 161
in leukæmia, 534
in lymphocytic leukæmia, 536
in malaria, 152
in malignant endocarditis, 331
in mixed infection, 154
in pernicious anæmia, 528
in portal cirrhosis, 485
in splenic anæmia, 530
in splenic anæmia of infancy, 531
in spleno-megalic cirrhosis, 487
gumma of, 551
"hard-bake" in Hodgkin's disease, 552
in acute leukæmia, 533
in failure of heart, 338
infarcts in, 550
in leukæmia, 532
in malignant endocarditis, 331
lardaceous degeneration of, 489, 550
parasites of, 551
passive congestion of, 550
ptosis of, 416
pus in, in melioidosis, 137
regulation of blood volume by, 550
removal of, in acholuric jaundice, 532
in purpura, 544
in splenic anæmia, 530
sago, 550
tuberculosis of, 551
erythrocytosis secondary to, 539
**Splenic** anæmia, 529
cirrhosis of liver in, 484
differentiation from malignant endocarditis, 334
enlargement of spleen in, 529
of infancy, 530
veins, thrombosis of, splenic anæmia and, 530
**Splenitis,** 550
**Splenoptosis,** 416
**Spondylitis** deformans in rheumatoid arthritis, 670
resembling hunger osteomalacia, 695
**Sponging,** 25
**Spongiosis,** 985
**Spores,** formation of, 9

**Spores** of Malassez in pityriasis, 935
**Sporogony,** 151
**Sporotrichosis,** 143
*Sporotrichum Beurmanni,* 143
**Spotted** fever, 93
**Sprue,** 448
steatorrhœa in, 504
**Sputum,** bile and hydatid skins in, 268
elastic tissue in, in phthisis, 257
examination of, in phthisis, 257
transmission of pneumonic plague by, 183
tubercle bacilli in, in phthisis, 257
**Squatting** test in beri-beri, 688
**Squint** in tetany, 585
**Staphylococcal** infections of skin associated with seborrhœic state, 931
**Staphylococci,** differentiation from streptococci in infections of skin, 926
in acne varioloformis, 945
in acute endocarditis, 329
in amœbic abscess, 170
in appendicitis, 452
in boils, 943
in broncho-pneumonia, 243
in cholecystitis, 496
in gangrene of lung, 247
infection by, 9
in infective endocarditis, 330
in Ludwig's angina, 395
in meningitis, 843
in myelitis, 772
in pericarditis, 356
in peritonitis, 510
in pityriasis capitis, 935
in pleurisy, 271
in pulmonary tuberculosis, 248
in suppurative cholangitis, 495
in sycosis, 941
secondary infection by, 13
**Star** figure in arteriosclerotic retinitis, 368
**Starvation,** slow pulse-rate in, 317
**Stasis,** colonic, 435
ileal, 435
intestinal, 436, 438
**Status** epilepticus, 871
lymphaticus, 587
thymico-lymphaticus, 587
**Steam,** disinfection by, 13
**Steatorrhœa,** congenital, 504
in abdominal tuberculosis, 504, 515
in congenital pyloric stenosis, 430
in jaundice, 474
in pancreatic disease, 503
*Stegomyia fasciata,* 178
**Stellwag's** sign in exophthalmic goitre, 580
**Stem** length *See* Body length
**Stenosis** at valvular orifices causing murmurs, 288
of valves of heart, 335
pyloric, clubbing of fingers in, 673
"relative," of mitral valve, 293
*See also* Obstruction, and Heart, valvular disease of.
**Stereognosis,** 730
**Sterno-mastoid,** paralysis of, 753
**Stiff-neck,** 910
**Still's** disease, 671
**Stimulants,** 26
**Stomach,** acute dilatation of, 418
acute distension of, differentiation from angina, 364
alterations in size and position of, 405
arrangement of muscular coat of, 405
blood in, examination of, 408
carcinoma of, 430
achalasia of cardia secondary to, 403
*See also under* Carcinoma of stomach.
contents of, examination of, 406
dilatation of, chronic, 428
in carcinoma, 431
in gastric ulcer, 423
in pneumonia, 241
predisposing to tetany, 585
diseases of, 404
dropped *See* Gastroptosis.
examination of, 404
functional disorders of, 408
glands of, destruction in pernicious anæmia, 527
hour-glass contraction of, 422, 425, 429
hypertonic, 405, 428
hypotonic or dropped, 405
**Stomach,** hypotonus of, dyspepsia in, 411, 412
in failure of heart, 338
lardaceous disease of, 489
lavage of, in carcinoma, 434
in dilatation of stomach, 429
leather-bottle, 431
lymphadenoma of, 434
tumours of, benign, 434
causing jaundice, 476
ulcer of, 421
achalasia of cardia secondary to, 403
acute, formation of, 423
carcinoma and, 431, 433
causing acute peritonitis, 510
differentiation from appendicitis, 454
experimental production of, 423
operations for, 427
perforated, causing intraperitoneal abscess, 512
*See also* Peptic ulcer
**Stomatitis,** 392
aphthous, 393
catarrhal, 393
gangrenous, 394
in chronic mercurial poisoning, 683
ulcerative, 393
**Stools,** excess of fat in *See* Steatorrhœa.
fatty, in congenital pyloric stenosis, 430
occult blood in, examination of, in dyspepsia, 413
**Strabismus** in cerebro-spinal fever, 95
**Strangulated** hernia, 459
**Strangulation** in intussusception, 465
of intestine by bands or through apertures, 459
**"Strawberry tongue"** in scarlet fever, 90
**Streptococcal** fissure, 930
infections of skin, 926
lymphangitis, relapsing, 931
**Streptococci** in acute endocarditis, 329
differentiation from staphylococci in infections of skin, 926
in alopecia areata, 1016
in appendicitis, 452
in cholecystitis, 496
in gangrene of lung, 247
in infective endocarditis, 330
in Ludwig's angina, 395
in malignant endocarditis, 330
in meningitis, 843
in myelitis, 772
in peptic ulcer, 424
in pericarditis, 356
in peritonitis, 510
in pleurisy, 271
in pulmonary tuberculosis, 248
in rheumatic fever, 55
in rheumatoid arthritis, 668
in suppurative cholangitis, 495
*Streptococcus brevis,* 442
hæmolytic, in broncho-pneumonia, 243
cause of pernicious anæmia, 526
in purpura, 542
in amœbic abscess, 170
infection by, 9
*longus* in herpes zoster, 999
*pyogenes,* 52
in lobar pneumonia, 238
secondary infection by, 13
**Streptothrix,** 10, 125
aerobic, in actinomycosis, 140
in splenic anæmia, 530
*Streptothrix maduræ,* 143
**Striæ et maculæ atrophicæ,** 1009
**Stricture** of rectum from gonorrhœa, 457
of ureter, cicatricial, cause of hydronephrosis, 643
**Strictures** from ulceration, intestinal obstruction due to, 458
**Stridor,** 194
in aortic aneurysm, 374
in compression of trachea, 214
in laryngeal diphtheria, 68
in obstruction of lung, 230
in œdematous laryngitis, 201
in paralysis of abductors of vocal cords, 208
laryngeal, congenital, 212
**Strobila,** 467
**Strophanthin** in auricular fibrillation, 315
in heart disease, 349
in simple paroxysmal tachycardia, 31
**Strophanthus** in heart disease, 349

**Strophulus**, 1013
albidus, 1014
**Strychnine** poisoning, differentiation from tetanus, 107
**Subacute** combined degeneration, 794
*See also* Spinal cord.
**Subconscious** mind, 892
**Submaxillary** glands in mumps, 46
**Subnormal** temperatures, conditions in which found, 23
**Subtertian** infection, 149
**Sudamina**, 1013
in pyrexia, 19
in rheumatic fever, 56
**Sugar** in arterial blood, insulin and, 563
in urine. *See* Glycosuria.
in venous blood, insulin and, 563
tolerance, diminished, in rheumatoid arthritis, 669
low, in myasthenia gravis, 920
lowered, in acromegaly, 595
**Suggestion** in hysteria, 902, 908
**Sulphæmoglobinæmia**, 541
clubbing of fingers in, 673
**Sulpharsenol**, 116, 118
in syphilitic myocarditis, 350
**Sulphates** in urine, 605
**"Sulphur granules"** in actinomycosis, 140
**Sun-bathing** in treatment of phthisis, 261
**Sunlight**, exposure to, in treatment of tuberculous peritonitis, 515
**Sunstroke**, 26
**Superior** vena cava, pressure on, in tuberculosis of bronchial glands, 385
**Supersensitiveness** (anaphylaxis), 17
**Suppuration** and lardaceous disease of kidney, 645
chronic, anæmia in, 523
**Suppurative** pylephlebitis, 502
**Suprarenal** glands, diseases of, 588
increase in sugar in blood due to, 564
in leukæmia, 534
*See also* Addison's disease.
**Sweat**, excess of, 1012
glands, affections of, 1012
**Sweating**, 19
in anxiety neurosis, 896
in hypoglycæmia from insulin, 572
in rheumatic fever, 56
**Swedish** movements in treatment, 7
**Sycosis** coccogenica, 941
differentiation from tinea sycosis, 958
nuchæ, 942
**Sympathetic** fibres, cervical, 720
paralysis of, 758
ganglia, chromatolysis of cells of, in pellagra, 696
**Symptoms**, meaning of, 2
**Syncope** in aortic regurgitation, 341
in mediastinal neoplasms, 387
local, in Raynaud's disease, 381
pleural reflex, 278
**Syndromes**, endocrinopathic, varied, 598
multiglandular, 601
pluri-glandular, 601
**Synovitis**, gonococcal, 73
differentiation from rheumatic fever, 59
in rheumatic fever, 56
**Syphilide**, congenital, associated with thrush, 395
papular, differentiation from pityriasis rosea, 988
roseolar, differentiation from pityriasis rosea, 988
scaly, differentiation from psoriasis, 991
varieties of rash and differential diagnosis, 41, 110
**Syphilis**, 109
acute endocarditis in, 328
anæmia in, 523
aortitis in, angina pectoris in, 365
arteritis, 113, 365
causing adhesive pylephlebitis, 502
bronchiectasis, 222
general paralysis of insane, 861
portal cirrhosis, 484
progressive muscular atrophy, 799
tabes dorsalis, 787
cerebro-spinal, cerebro-spinal fluid in, 737
congenital, 109, 120
acute rhinitis in, 197
enlargement of spleen in, 550
**Syphilis**, differentiation from disseminated sclerosis, 783
encephalitis lethargica, 835
glanders, 137
Hodgkin's disease, 553
leprosy, 135
osteitis deformans, 676
endarteritis, 113
causing lesions of valves of heart, 336
epiphysitis, differentiation from rickets, 691
differentiation from scurvy, 690
fibroid degeneration of heart in, 324
gastric ulcer due to, 422
heart block in, 304
treatment of, 307
infantile cerebral diplegia and, 839
in splenic anæmia, 530
lardaceous disease of kidney and, 645
leucodermia in, 1012
meningitis in, 843, 845
herpes zoster in, 999
myelitis in, 772, 773
treatment of, 776
œdematous laryngitis in, 200
of heart, 328
of larynx, 204
of liver, 490
differentiation from carcinoma, 492
of lung, 264
of muscles, 912
of rectum, 457
of trachea, 213
ophthalmoplegia in, 746
paralysis of abductors of vocal cords in, 209
parenchymatous, 109, 113, 788
paroxysmal hæmoglobinuria in, 540
possible cause of diabetes mellitus, 562
primary lesions, 109, 110
relation to aneurysm, 372
routine treatment of, 117, 118
secondary lesions, 109, 110
secondary, differentiation from scarlet fever, 31
tertiary, 112
*See also* Gumma.
**Syringomyelia**, 784
differentiation from compression of spinal cord, 770, 786
hydrocephalus associated with, 859
**Systoles**, extra, 307
in angina, 363
in mitral regurgitation, 341
in myocardial degeneration, 325
in simple paroxysmal tachycardia, 311
**Systolic** blood pressure, 300
murmurs, 288, 289
*See also* Murmurs, systolic
retraction in adherent pericardium, 359

**Tabby** cat striation in fatty degeneration of heart, 323
in pernicious anæmia, 526
**Tabes**, benzoin test in, 738
causing diarrhœa, 440
cervical, 790
differentiation from pellagra, 697
from polyneuritis, 742
dorsalis, 786
anæsthesia of larynx in, 212
gastric crises of, differentiation from intestinal obstruction, 463
spasm of glottis in, 211
gastric crises in, 789, 790
dyspepsia in, 411
juvenile, differentiation from Friedreich's ataxia, 798
mesenterica, 514
paralysis of abductors of vocal cords in, 209
**Tache** méningitique, 805
*Tache cérébrale*, 130
**Tachycardia**, 310
in alcoholic neuritis, 741
in exophthalmic goitre, 580
in Malta fever, 173
paroxysmal, 309
simple, 310
*Tænia echinococcus*, 493
*saginata (mediocanellata)*, 466, 467
*solium*, 466
**Talipes** equinus after acute poliomyelitis, 100
**Tapeworms**, 466
**Tapôtement** in massage, 7

**Tartar** emetic in treatment of Kala-azar, 162
**Taste,** nerve tracts associated with, 713
tests for, 725
**Teeth,** defective, in rickets, 692
defects of, in latent tetany, 586
infection of, in primary asthma, 227
X-ray examination of, 392
*See also* Oral sepsis.
**Teichopsia** in migraine, 878
**Temperament,** 723
**Temperature,** rectal, in diagnosis of phthisis, 257
registration of, 19
sensibility to, 729
**Temperatures,** collapse, 19
normal, range of, 19
subnormal, 19
conditions in which found, 23
in alcoholism, 677
**Temporal** lobe, localisation of function in, 814
**Tendon** jerks, absent in myotonia atrophica, 918
loss of, in peroneal muscular atrophy, 799
**Tendons,** sheaths of, inflammation in rheumatic fever, 56
**Teno-synovitis** in dysentery, 165
in gonorrhœal rheumatism, 73
**Test,** Casoni's, in hydatid disease of liver, 495
diadochokinesis, 732
Dick, 32
finger-nose-finger, 732
heel-knee, 732
meal, 406
Ewald's, 406
fractional, 407
in chronic dilatation of stomach, 429
in duodenal ulcer, 425
in diagnosis of carcinoma of stomach, 433
peptic ulcer, 425
in dyspepsia, 413
lactic acid in, in carcinoma of stomach, 434
Schick's, 70
Van den Bergh's. *See* under Van den Bergh
**Tests** of function of kidney, 609
of liver, 473
of pancreas, 503
for bile pigment, 475
**Testes,** amœbic abscesses in, 167
atrophy of, in myotonia atrophica, 918
histological changes in Addison's disease, 590
tumours of, 598
**Tetania** parathyreopriva, 584, 585
**Tetanus,** 104
antitoxin, 107
cephalic, 106
differentiation from hydrophobia, 103
spinal meningitis, 805
tetany, 586
modified or local, 106
neonatorum, 104
spasm of glottis in, 211
splanchnic, 106
traumatic, 104
**Tetany,** 584
acapnia in, 585
alkalæmia in, 547
differentiation from tetanus, 107
epidemic, 585
in myxœdema, 582
in osteomalacia, 674
in rickets, 693
laryngismus stridulus in, 211
latent, 586
*See also* Parathyroid glands.
**Tetrachlorethane** poisoning, acute yellow atrophy of liver in, 481
**Thermic** fever, 26
**Thermogenesis,** 22
**Thermolysis,** 22
**Thiothrix,** 10
**Thomsen's** disease, 917
**Thoracic** duct, rupture or obstruction of, as cause of chylous ascites, 478
**Thoracoplasty,** 263
**Thoracoscope** in treatment of adhesions in artificial pneumothorax, 263
**Threshold,** renal, for sugar, low, in glycosuria, 571
**Thrill,** hydatid, 494
in heart disease, 286
in mitral stenosis, 342
in stenosis of pulmonary valves, 344
systolic, in congenital heart disease, 355
**Throat** cough, 398
sore, catarrhal, 395
tickling in, in whooping-cough, 49
**Thrombo-angiitis** obliterans, 365
erythromelalgia in, 383
in intermittent claudication, 382
**Thrombocytopenia,** 542
**Thrombo-phlebitis** in thrombosis of inferior vena cava, 380
**Thrombosis,** 378
arterio-sclerosis in, 367
femoral, 380
in phthisis, 255
in pulmonary embolism, 269
jugular, 380
of cavernous sinus, 380
of central retinal artery in arteriosclerosis, blindness following, 368
of cerebral arteries, 825
of cerebral capillaries in arseno-benzol poisoning, 123
of cerebral sinuses, 380, 848
of inferior vena cava, 380
of lateral sinus, 380
of longitudinal sinus, 380
of pelvic veins, 380
in pulmonary embolism, 269
of portal vein, 502
of splenic and portal veins, erythrocytosis secondary to, 539
of splenic veins, splenic anæmia in, 530
of veins accompanying gout, 665
pulmonary, 269
sinus, in infantile enteritis, 445
**Thrush,** 394
**Thymic** asthma, 587
**Thymol** in treatment of intestinal worms, 472
**Thymus** gland, diseases of, 586
enlargement of, 587
in myasthenia gravis, 919
in leukæmia, 534
tuberculosis of, 587
**Thymus-suprarenal-pituitary** compensatory syndrome, 601
**Thyroid** extract in treatment of alopecia areata, 1016
circumscribed sclerodermia, 1008
in cretinism, 584
in myxœdema, 583
in obesity, 599
gland, changes in, in adiposis dolorosa, 599
diseases of, 576
carcinoma, 577
cretinism associated with, 576, 583
encapsuled adenoma, 577
myxœdema associated with, 576, 581
operations for, 577
sarcoma, 577
toxic adenoma, 577, 578
enlargement of, in exophthalmic goitre, 579
increase in sugar in blood due to, 564
in leukæmia, 534
lardaceous disease of, 489
removal of, tetany after, 585
*See also* Goitre.
**Thyroideum** siccum in treatment of myxœdema, 583
**Thryo-toxic** fibrillation, 317
**Thyroxin,** 578
**Tic** or **Tics,** 880
douloureux, 885
general convulsive, 881
hysterical, in aerophagy, 415
**Tick** fever, 145
**Tickling** in throat, 398
in whooping-cough, 49
**Ticks,** transmission of infection by, 12
**Timme's** syndrome, 601
**Tinea** circinata, 957
differentiation from pityriasis rosea, 988
psoriasis, 991
marginata, 956
of the nails, 958
sycosis, 956
tonsurans, 956
versicolor, 961
differential diagnosis of Addison's disease from, 591
**Tingling** in anxiety neurosis, 896
in multiple neuritis, 740
**Tinnitus** aurium, 750
in acoustic tumours, 856

**Tinnitus** in neurasthenia, 887
quinine treatment of malaria causing, 156
**Tone,** muscular, variations in, 732
**Tongue,** in pyrexia, 20
paralysis of, 753
secondary vaccinia on, 44
soreness of, in pernicious anæmia, 528
**Tonsillectomy,** 398
abscess of lung following, 245
in rheumatic fever, 59
**Tonsillitis,** 395
differential on from glandular fever, 50
follicular, 396
differentiation from diphtheria, 69
quinsy, 397
scarlet fever, 31
malignant endocarditis in, 330
psoriasis and, 990
rheumatic fever following, 55
**Tonsils** as primary focus of rheumatic fever, 55
chronic enlargement of, 397
diseases of, 395
faucial, 395
infection of, in primary asthma, 227
lingual, 395, 398
painting of, in phthisis, 263
neoplasms of, 398
pharyngeal, 395, 398
secondary vaccinia in, 44
septic, chronic, 398
**Tophi** in gout, 665
**Torticollis,** spasmodic, 881
**Toxæmia,** 11
alimentary, 438
intestinal, eczema and, 984
**Toxalbumins,** production of, by bacterial action, 11
**Toxic** eruptions of skin, 979
idiopathies, 225, 384
eczema and, 984
**Toxins,** bacterial, action of, in infectious disease, 11
in liver causing ascites, 477
of tetanus transmitted by motor nerve fibres, 105
**Trachea,** compression of, 214
diseases of, 213
foreign bodies in, 214
new growths in, 213
obstruction of, 214
in diphtheria, 68
pressure on, in tuberculosis of bronchial glands, 385
stricture of, 214
syphilitic stenosis of, differentiation from syphilitic laryngitis, 205
**Tracheal** tugging in aortic aneurysm, 374
**Tracheitis,** 213
**Tracheotomy** for removal of foreign bodies in larynx, 207
in laryngeal perichondritis, 205
in paralysis of abductors of vocal cords, 209
**Transfusion** of blood, 545
in malignant endocarditis, 334
**"Transitional"** cells, 521
**Transverse** colon, ptosis of, 416
**Trapezius,** paralysis of, 753
**Traube's** plugs, 221
space, 274
**Trauma,** causing neuritis, 738
thrombosis of inferior vena cava, 380
diabetes mellitus following, 562
**Traumatic** tetanus, 104
**Tremor** and **Tremors,** 730, 921
fibrillary, 731, 921
in amyotrophic lateral sclerosis, 802
in progressive muscular atrophy, 800
in syringomyelia, 784
in anxiety neurosis, 896
in cerebellar lesions, 715
in chronic alcoholism, 679
in encephalitis lethargica, 834
in exophthalmic goitre, 580
in general paralysis of insane, 863
in paralysis agitans, 841
in progressive lenticular degeneration, 840
in writers' cramp, 884
intention, in disseminated sclerosis, 782
mercurial, 683
senile, differentiation from paralysis agitans, 842
types of, 730
**Trench** fever, 64
**Trichiniasis,** 912
differentiation from enteric fever, 88
*Trichocephalus dispar,* 471
**Trichomonas,** presence of, in amœbic dysentery, 167
**Trichomycetæ,** infection by, 10
*Trichophyton acuminatum,* 959
*megalosporon,* 959
*endo-ectothrix,* 959
*endothrix,* 959
*microsporon,* 958
*rubrum,* 959
*violaceum,* 959
**Trichorrhexis** nodosa, 1014
*Trichuris trichiura,* 471
**Tricuspid** regurgitation, 343
murmurs in, 291, 292
stenosis, 344
murmurs in, 291, 292
*See also under* Murmurs.
**Trinitrotoluene** poisoning causing acute yellow atrophy of liver, 481
**Trismus,** 763
in tetanus, 105
**Trophic** changes, 735
in hysteria, 906
in lesions of fifth nerve, 748
in neuritis, 738
disturbances in syringomyelia, 784
in tabes, 791
**Trophonucleus** in trypanosomes, 158
**Trophozoites** of malaria parasite in blackwater fever, 157
**Tropical** abscess, 169, 170, 479
of liver, resemblance to suppurative cholangitis, 496
**Trousseau's** sign in latent tetany, 586
**Trypanosoma,** 10
*Trypanosoma gambiense,* 158
*rhodesiense,* 158
**Trypanosomiasis,** 158
**Tryparsamide** in treatment of trypanosomiasis, 160
**Tryptophane,** 685
**Tsetse** flies, infecting agent in trypanosomiasis, 158
transmission of infection by, 12
**Tuberculides,** the, 951
papulo-necrotic, 953
**Tuberculin** in asthma, 229
in diagnosis of phthisis, 257
of tuberculous peritonitis, 515
in treatment of phthisis, 261
of tubercle of kidney, 642
of the tuberculides, 952
**Tuberculosis,** 125
abdominal, steatorrhœa in, 504, 515
anæmia in, 523
causing ascites, 477, 486
cerebral abscess in, 837
colony, 260
differentiation from broncho-pneumonia, 244
enteric fever, 88
leprosy, 135
Mediterranean fever, 173
distinction between human and bovine tubercle 127
enlargement of spleen in, 550
general or miliary, 128, 129
, complicating phthisis, 255
genito-urinary, bovine infection in, 127
laryngeal, 202
*See also* Larynx.
of bladder, 641
of bronchial glands, 385
*See also under* Glands.
of heart, 328
of intestine, 456
of kidney, 640
differentiation from renal calculus, 642, 659
of liver, 491
of lung, 248
*See also* Phthisis.
of muscles, 914
of skin, 947
of spine, 767
causing compression, 767
differentiation from perinephric abscess, 640
of spleen, 551
of suprarenal capsules in Addison's disease, 590
of thymus, 587
of trachea, 213
of ureter, 641

**Tuberculosis,** pericarditis in, 355
pleurisy and, 271
polyneuritis, 740
pulmonary, 248
verrucosa, 951
**Tuberculous** adenitis, differentiation from glandular fever, 50
broncho-pneumonia, 249
*See also* Phthisis.
consolidation of lung simulating pleurisy, 276
infection in pneumokomioses, 245
meningitis, 129
peritonitis, 514
*See also* Peritonitis.
ulcers of ileum causing acute peritonitis, 508
ulcers of skin, 947
**Tubular** breathing, 194
**Tularæmia,** 185
**Tumour** and **Tumours,**
acoustic, 850
symptoms of, 856
extra-cerebellar, 852
in carcinoma of stomach, 431
in portal fissure, obstruction by, causing ascites, 477
intra-cerebellar, 852
mediastinal, 386
of brain, 849
*See also* Cerebral tumour
of medulla oblongata, 812
of spinal cord, 772
presence of, in intussusception, 464
pressure from, causing adhesive pylephlebitis, 502
**Turban-Gerhardt** classification of apical phthisis, 252
**Tylosis** palmaris et plantaris, 1006
**Tympany** in chest percussion, 191
**Typhoid** fever. *See* Enteric fever.
spine, 86
**Typhus** exanthematicus, 74
fever, 74, 79
differentiation from measles, 35
plague, 185
relapsing fever, 147
scarlet fever, 31
fatty degeneration of heart in, 323
purpura in, 542
**Typus** inversus in pyrexia, 21

**Ulceration,** nasopharyngeal, in trypanosomiasis, 161
**Ulcerative** endocarditis, 330
*See also* Endocarditis, malignant
**Ulcers,** atheromatous, 366
gastro-jejunal, 427, 428
juxta-pyloric, 422
of colon causing acute peritonitis, 508
of duodenum. *See under* Duodenum *and* Peptic
of large intestine, amœbic, 166
of skin, tuberculous, 947
of stomach. *See under* Stomach *and* Peptic.
peptic, 421 *See also under* Peptic.
perforating, in tabes, 791
varicose, in thrombosis of inferior vena cava, 380
**Ulnar** nerve, lesion of, differentiation from progressive muscular atrophy, 801
**Ultra-microscopic** organisms, 9
virus cause of influenza, 61
**Ultra-violet** light in treatment of phthisis, 261
**Umbilication** of vesicle in small-pox, 39
**Uncinate** seizures in cerebral tumours, 854
in pituitary tumours, 594
**Unconscious** mind, 892
**Undernutrition** in treatment of diabetes, 571
slow pulse rate in, 317
**Uræmia,** asthenic or chronic, 629
acidæmia in, 548
course of, 630
in suppurative nephritis, 638
blood transfusion in, 546
carbon dioxide in alveolar air in, 629
in acute myelitis, 774
in acute nephritis, 621
in cholera, 181
in cystic disease of kidney, 654
in endocarditis lenta, 333
in gout, 665
in hydronephrosis, 645
in primary contracted kidney, 633
**Uræmia** in typhus fever, 77
latent, 630, 658
paroxysmal headache due to, differentiation from migraine, 879
Sippy's diet causing, 426
**Uræmic** asthma, 227
convulsions, treatment of, 623
dermatitis, 979
**Urea,** administration by mouth in treatment of chronic parenchymatous nephritis, 627
concentration test, 610
in acute nephritis, 620
in chronic interstitial nephritis, 627
in blood, estimation of, 610
in acute nephritis, 620
in chronic parenchymatous nephritis, 625
increase of, in tetany, 585
in typhus fever, 77, 78
increased in suppurative pyelo-nephritis, 638
in secondary contracted kidney, 629
in senile arterio-sclerotic kidney, 634
retention of, in myocardial degeneration, 325
quantitative estimation of, 604
**Ureter,** tuberculosis of, 641
**Ureters,** catheterisation of, 603
**Urethra,** stricture of, cause of hydronephrosis, 643
**Urethritis,** malignant endocarditis in, 330
**Uric** acid and urates in urine, 606
endogenous, 664
excess of, in blood, 664
exogenous, 664
**Uridrosis,** 1013
**Urinary** bladder *See* Bladder.
**Urine,** albumoses in, 616
bacilli in, 634
Bull's orcin test for pentose, 567
bile pigment in, tests for, 475
bile salts in, Hay's test for, 475
blood in, 616 *See also* Hæmaturia.
calcium oxalate in, 606
carboluria associated with ochronosis, 1011
casts in, various types of, 617, 618
chlorides in, 605
colouration by medical and chemical substances, 608
cylindroids in, 618
detection and causes of albumin in, 613, 615
*See also* Albuminuria
diastase in, estimation of, 610
in pancreatic disease, 504
effects of pyrexia on, 20
euglobulin in, 614, 616
examination of, 603
fallacies in detection of albumin in, 613
glycuronic acid in, 567
guaiacum test for blood in, 617
hæmatoporphyrin in, 608
incontinence of, in bacilluria, 634
in hæmoglobinuria, 539
lactose in, 567
lævulose in, 563, 567
melanin in, 608
methæmoglobin in, 616
in hæmoglobinuria, 540
method of making acid or alkaline, 609
murexide test, 607
nitrogen in, estimation of, Kjeldahl's method, 605
nucleo-protein in, 614
nucleo-protein, euglobulin, in, 616
obstructive suppression of, 657
oxy hæmoglobin spectrum in, 617
pentose in, 567
peptones in, 616
phosphates in, 605
pigments of, 607
pseudo-lævulose in, 567
pus in *See* Pyuria.
reaction of, 609
retention of, in infective myelitis, 774
in pyrexia, 21, 24
in spinal meningitis, 805
serum-globulin in, 615
specific gravity of, 604
sugar in. *See* Glycosuria.
sulphates in, 605
temperature of, normal, 18
urea concentration test, 610. *See also* Urea.
uric acid and urates in, 606

**Urobilin** in urine, 608
in acholuric jaundice, 531
in pernicious anæmia, 528
**Urobilinogen** in urine, 608
**Urochrome** in urine, 608
**Uroerythrin** in urine, 608
**Urticaria,** 975
due to quinidine, 317
in hay fever, 227
in *Schistoma japonicum* infection, 650
in spirochætosis, 175
in toxic idiopathies, 225
papulosa, 977
pigmentosa, 978
**Uterus,** lardaceous disease of, 489
pregnant, simulating ascites, 478
tuberculosis of, in phthisis, 255
tumours of, causing jaundice, 476

**Vaccination,** 15, 42
by glycerinated calf lymph, 43
**Vaccines,** bacterial, 15
in treatment, 6
gonorrhœal, in treatment of keratodermia blennorrhagica, 991
in malignant endocarditis, 334
treatment of acne vulgaris, 937
of actinomycosis, 143
of bacilluria, 635
of enteric fever, 89
of furunculosis, 943
of gonococcal synovitis, 74
of hay fever, 228
of hydrophobia, 104
of Mediterranean fever, 173
of nasal catarrh, 197
of rabies, 104
of rheumatoid arthritis, 671
of septicæmia, 50
of sycosis, 942
prophylactic in enteric fever, 89
in influenza, 63
in plague, 185
in typhoid fever, 89
**Vaccinia,** 42
aberrant, 44
secondary, 44
**Vagabond's** disease, 965
**Vagal** inhibition in aortic regurgitation, 341
stimulation in myocardial degeneration, 324
**Vagi,** pressure on, in paroxysmal tachycardia, 311
**Vaginitis,** malignant endocarditis in, 330
**Vagus** affections in alcoholic neuritis, 741
examination of, 728
inhibition of, digitalis causing, 316
injury to, anæsthesia of larynx in, 212
paralysis of, quickened action of heart in, 310
pressure on, causing achalasia of cardia, 403
**Valves** of heart, diseases of, 334. *See also* Murmur, Regurgitation, Stenosis, Heart, valvular disease of.
**Van den Bergh's** test in acholuric jaundice, 531
in acute yellow atrophy, 482
in carcinoma of liver, 492
in catarrhal jaundice, 480
in cirrhosis, 485
in jaundice, 475
in pernicious anæmia, 527
**Varicella,** 44. *See also under* Chicken-pox.
gangrenosa, 929
**Varicose** veins in thrombosis of inferior vena cava, 380
ulcers in thrombosis of inferior vena cava, 380
**Variola,** 37 *See also* Small-pox.
**Varioloid,** 41
differentiation from chicken-pox, 45
**Vegetations** in acute endocarditis, 328
**Veins,** auscultation of, 303
obstruction of, in mediastinal neoplasms, 386
varicose, in thrombosis of inferior vena cava, 380
**Venereal** warts, 962
**Venesection** in cardiac disease, 348
in cerebral hæmorrhage, 824
in erythræmia, 538
in simple paroxysmal tachycardia, 312
**Venous** hum, 303
**Ventricle and auricle** rhythms, dissociation in heart block, 305. *See also* Heart block.
left, hypertrophy of, in aortic stenosis, 339
small, in mitral stenosis, 342
**Ventricular** extra systoles, 307, 308
fibrillation, 318
**Verruca,** 963
necrogenica, 126, 951
**Vertebræ,** diseases of, 769
malignant growth of, differentiation from chronic myelitis, 775
new growths of, causing compression of cord, 767
secondary deposits in, 767
tuberculous caries of, differentiation from chronic myelitis, 775
causing compression of cord, 767
**Vertigo,** 728
causes and treatment of, 751
following gassing, 265
in anxiety neurosis, 896
in cerebral tumours, 853
in hypoglycæmia from insulin, 572
**Vesiculæ** seminales, tuberculosis of, in phthisis, 255
**Vesicular** murmur, 192
diminished, 193
skin eruptions, 998
**Vesiculitis,** 73
**Vibices,** 924
**Vibration,** sense of, 730
in compression of spinal cord, 768
diminution of, in disseminated sclerosis, 781
in massage, 7
*Vibrio, cholera,* 179
*Vibrion septique,* 108
**Vincent's** angina, 394, 397
differentiation from diphtheria, 69
follicular tonsillitis, 396
spirochæte, 397
**Virilism,** 592
**Virus,** action of, in recipient, 10
fixé, in antirabic treatment, 104
**Visceral** pains in dyspepsia, pathology of, 409
**Visceroptosis,** 416
**Viscosity** of blood increased in hyperpiesia, 369
**Visible** peristalsis, 428
**Vision,** acuity of, 724
disturbances of, in cerebral tumours, 854
fields of, in arteriosclerotic retinitis, 368
nerve tracts associated with, 712
tests of fields of, 724
**Visual** acuity, defects of, in disseminated sclerosis, 782
**Vital** capacity, 189
estimation of, in diagnosis of phthisis, 257
**Vitamins,** calcifying, 685
in relation to dental caries, 686
in rickets, 691
**Vitiligo,** 1012
alopecia areata associated with, 1016
**Vitiligoidea,** 1018
**Vocal** cords, paralysis of, complete, 208
in laryngeal tuberculosis, 203
perverted action of, differentiation from paralysis, 209
fremitus, tactile, 190
resonance, 195
*See also* Laryngeal.
**Voice,** auscultation of, 195
hoarseness of, in tumours of larynx, 206
**Voluntary power,** systematic investigation of, 732
**Volvulus** of intestine, 458
**Vomicæ** in pulmonary tuberculosis, 249
**Vomit,** coffee grounds, 406
**Vomiting** after gastro-jejunostomy, 427
bilious in acute dilatation of duodenum, 420
cyclical, 418
due to quinidine, 317
hysterical, 418
in cerebral tumours, 853
in dyspepsia, pathology of, 411
in empyema, 276
in gastric ulcer, 424
in gastroptosis, 417
in heat stroke, 27
in intussusception, 465
morning, following gassing, 265
stercoraceous or fæcal, in intestinal obstruction, 460
**Von Graefe's** sign in exophthalmic goitre, 580
**Von Jaksch's** anæmia, 530
**Von Pirquet's** cutaneous reaction, 257
**Vulva,** pruritus of, complicating diabetes, 569
**Vulvo-vaginitis** of children, *Bacillus coli communis* in, 74
gonococcus in, 74
staphylococcus in, 74

**Wart,** 963
post-mortem, 126
**Warts,** venereal, 962
**Wassermann** reaction, 114, 115
in acute myelitis, 774
in cerebro-spinal fluid, 738
in congenital syphilis, 121
in diagnosis of compression of spinal cord, 770
in diagnosis of tabes, 787
in encephalitis lethargica, 835
in estimation of effect of treatment of syphilitic myelitis, 776
in gastric ulcer, 425
in leprosy, 135
in syphilis, course of treatment influenced by, 117, 119
in syphilitic meningitis, 846
in tabes dorsalis, 787
positive, causes of, 115
in paroxysmal hæmoglobinuria, 540
provocative dose of arseno-benzol, 110
**Water brash,** 412
contamination of, causing typhoid fever, 80, 89
means of spread of cholera, 179
**Water-fever** " in poisoning by arseno-benzol, 123
**Water-hammer** pulse in aortic regurgitation, 340
**Water-soluble** B, 685, 686
**Weakness** in neuritis, 739
**Weight,** loss of, in gastroptosis, 417
in myotonia atrophica, 918
**Weil-Felix** test in typhus, 78
**Weil's** disease, 173
**Wens,** 1014
**Wet** pack, 25
**Whipworm,** 471
**Whooping-cough,** 47
bronchitis in, 215
cough in, due to inflamed lingual tonsils, 398
laryngismus stridulus in, 211
quarantine period in, 13
**Widal's** test, 16, 81, 87
**Winter** cough, 219
**Wool-sorter's** disease, 139
**Word** blindness, 718, 723
deafness, 718, 723
forgetfulness, 724
**Worms,** intestinal, 466
**Wrist-drop** in multiple neuritis, 741
in musculo-spiral nerve lesions, 756
**Writers'** cramp, 883

**Xanthelasma,** 1018
**Xanthoma,** 1018
complicating diabetes, 569
*Xenopsylla astia* (rat flea), 184
**Xerodermia,** 1007
albidum, 1010
pigmentosa, 1009
**Xerophthalmia** due to lack of fat-soluble A, 686
**X-ray appearances in examination or diagnosis.**
**Abdomen,** 391
acute yellow atrophy of liver, 483
appendicitis, 455
carcinoma of colon or rectum, 456
diverticulitis, 457
duodenal ulcer, 425
gall-stones, 475
gastroptosis, 417
kidney, 603
muco-membranous colitis, 450
peritoneal adhesions, 516
stomach, 405
carcinoma, 433, 434
chronic dilatation, 429
duodenal ulcer, 425
gastric ulcer, 425
gastroptosis, 417
hour-glass contraction, 430
tuberculous peritonitis, 515
ulcerative colitis, 451
subphrenic abscess, 513
aerophagy, 416
cerebral tumours, 857
**X-ray appearances in examination or diagnosis.**
**Lungs and Mediastinum,** 190
abscess, 245
aneurysm, 375
chronic phthisis, 255
compression of spinal cord, 769
diaphragmatic hernia, 282
hilus phthisis, 257
hydatid, 268
lobar pneumonia, 241
mediastinal neoplasms, 387
mediastinitis, 384
neoplasm, 268
phthisis, 253, 257
pleurisy, 275
pneumothorax, 281
tuberculosis of bronchial glands, 385
**X-rays in diagnosis** of foreign body in bronchi, 231
foreign bodies in larynx, 207
gas gangrene, 108
gout, 666
**X-rays appearances in examination or diagnosis.**
**Circulation,** 295
adherent pericardium, 360
aneurysms, 326
congenital disease, 355
dilatation, 321
effort syndrome, 352
heart, 295
hypertrophy, 318, 320
inform tteat claudication, 383.
pericarditis, 358
valvular disease, 344, 346
obstruction of œsophagus, 402
pituitary disease, 594
pituitary tumours, 594, 595
renal calculus, 659
rheumatoid arthritis, 669, 671
rickets, 694
status thymico-lymphaticus, 587
teeth, 392
tumours of spinal cord, 772
vertebræ, disease of, 769
**X-rays in treatment,** 6
by artificial pneumothorax, 262
of acne vulgaris, 948
of cheloid, 1017
of chronic enlargements of salivary glands, 400
of circumscribed sclerodermia, 1008
of eczema, 986
of goitre, 577, 581
of Kala-azar, 162
of lichen planus, 996
of neoplasms of lung, 268
of rhinoscleroma, 955
of ringworm, 959
of sarcoma of peritoneum, 517
of splenic anæmia, 530
of sycosis, 942
of tuberculosis verrucosus, 951
of warts, 964
of xerodermia pigmentosa, 1010
**X-ray, deep, in treatment** of actinomycosis, 143
of Hodgkin's disease, 554
of leukæmia, 535
of pituitary tumours, 858
of tumours of spinal cord, 772
**Xylyl** bromide, poisoning by, 265

**Yaws,** 124
**Yeast** in treatment of beri-beri, 688
" **Yellow Cross** " shells, poisoning by, 265
**Yellow** fever, 175
differentiation from plague, 185
relapsing fever, 147
fatty degeneration of heart in, 323
vision in jaundice, 474

**Zammit** test, 173
**Zenker's** degeneration in enteric fever, 82
in pyrexia, 22
in typhus fever, 75
**Zona,** 998 *See also* Herpes Zoster.
**Zygotes,** 151

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## INDEX.

PAGE

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