UNIVERSAL
LIBRARY
OU_172370
UNIVERSAL
LIBRARY

ENCYCLOPEDIA AND DICTIONARY

OF

MEDICINE AND SURGERY

Printed by NEILL & CO., LIMITED, *Edinburgh*,

FOR

WILLIAM GREEN & SONS.

January 1909.

GREEN'S ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

EDITED BY

J. W. BALLANTYNE, M.D., F.R.C.P.E.

VOL: X

THIERSCH-ZYMOTIC

WILLIAM GREEN & SONS
EDINBURGH & LONDON

EDITORIAL NOTE

THIS, the tenth, volume of Green's *Encyclopedia and Dictionary of Medicine and Surgery*, carries the subject headings from *Thiersch's Method* to *Zymotic*, and brings the whole work to a conclusion. Within these ten volumes will be found almost all the articles which appeared originally in the thirteen volumes of the *Encyclopædia Medica*, along with a large number of new articles, long and short, dealing with subjects such as PHYSIOLOGY, MATERIA MEDICA, PHARMACOLOGY, PRESCRIBING, the TRAINING OF MIDWIVES, and the MEDICAL EXAMINATION OF SCHOOL CHILDREN, which in the former work were not treated in detail. The promises made in the Introduction to Volume I. have all been fulfilled: a Medical Dictionary, containing thousands of definitions, has been incorporated with the *Encyclopædia Medica*; an elaborate system of Cross References has been carried through; important advances in medical and surgical practice have been taken notice of, sometimes in the form of new articles, sometimes as additions, marked by square brackets, to existing articles, and sometimes by short paragraphs or definitions cross-referenced to the original contributions; and additional illustrations have been supplied. In one particular alone the performance falls short of the promise: the appearance of the Supplementary Volume to the *Encyclopædia Medica* and to the present work, which it was hoped would take place in the present year (1908), must be postponed to the Spring or Summer of 1909; it will still, however, be published within five years after the date (1904) on the title-page of the Index Volume of the *Encyclopædia Medica*, and will, therefore, be correctly designated a *Quinquennium of Medicine and Surgery*.

The present volume contains 1059 subject-headings; of these, sixty-four are articles of more than 1000 words in length, fifty-four are of medium size (less than 1000 words, but not less than ten lines in length), and 941 are short paragraphs, mostly of the nature of definitions, of less than ten lines.

The sixty-four long articles deal as usual with a great variety of subjects. There is, for instance, a group of contributions treating of the morbid conditions of the UTERUS, Developmental Errors, Displacements, Inflammations, Tumours, and Insanity associated with Uterine Disease; these contributions cover nearly sixty pages of the *Encyclopedia*. Another long article (forty-two pages) deals with PATHOLOGICAL CHANGES IN THE URINE; another with TUBERCULOSIS; another with the WRIST-JOINT; another with WHOOPING-COUGH; another with YELLOW FEVER, and so on. Two closely associated subjects fall to be considered near to each other in alphabetical sequence, TOXICOLOGY and DANGEROUS TRADES. Other matters dealt

with in these longer articles are the THYMUS GLAND, the THYROID GLAND, the TONGUE, the TONSILS, the TRACHEA, TREMORS, TROPICAL FEVERS, TUMOURS, TYPHOID and TYPHUS FEVER, ULCERS, UNCONSCIOUSNESS, UNDULANT FEVER, URÆMIA, URTICARIA, VACCINATION, VAGINA, VARICELLA, VENEREAL DISEASE, VENTILATION and WARMING, VERRUGA PERUANA, VERTIGO, VICE, VISCERAL PAIN, FIELDS OF VISION, WATER, WOUNDS, X-RAYS, and YAWS.

Among the subjects treated of in the fifty-four medium-sized articles may be named *Thymol*, *Therapeutics of the Thyroid Gland*, *Tonic*, *Total Necrosis*, *Toxine*, *Tragacanth*, *Training*, *Trauma*, *Trendelenburg Posture*, *Tricephalus*, *Trypsin*, *Turpentine*, *Unsound Food*, *Urea*, *Urginea*, *Urotropin*, *Uva Ursi*, *Valerian*, *Venesection*, *Veronal*, *Virus*, *Voice*, *Vomiting*, *Xeroderma Pigmentosum*, *Xerosis*, *Yawning*, and *Zinc*.

The long list of 941 short articles, definitions, and brief descriptions deals of necessity with various aspects of medicine and surgery, as will be evident if a glance be cast over the following subjects, taken almost at random from the Index. There are references to *Thiol*, *Thiosinamine*, *Third Nerve*, *Thirst*, *Thlipsencephalus*, *Thomsen's Disease*, *Thought-Reading*, *Thread Worms*, *Thrombus*, *Thrush*, *Thyroidotomy*, *Tic*, *Tingling*, *Tisane*, *Tobin's Tube*, *Torsion*, *Tourniquet*, *Toxicosis*, *Trachoma*, *Tricrotic*, *Trigger Finger*, *Trochlea*, *Trophoblast*, *Trousseau's Symptom*, *Tuberose*, *Tunnel-Anæmia*, *Turmeric*, *Typhomalaria*, *Uncinariasis*, *Undue Influence*, *Uniovular*, *Unwell*, *Urechysis*, *Urogosan*, *Uteralgia*, *Uvea*, *Vaccine*, *Vagabond's Disease*, *Vaporisation*, "*Varium*," *Vectis*, *Veldt Sore*, *Verga's Ventricle*, *Vernix Caseosa*, *Vesania*, *Viability*, *Vibices*, *Vis*, *Vitiligo*, *Volsella*, *Watch-Test*, *White Leg*, *Winckel's Disease*, *Wood Wool*, "*Xaxa*," *Xenomenia*, *Xiphopagus*, *Xylotherapy*, *Yeast*, *Yerba Santa*, *Yohimbin*, *Zestokausis*, *Zomotherapy*, *Zoster*, and *Zygoma*.

It may be of interest if I give here in a tabular form the number of articles—long, medium, and short—which are contained in the ten volumes which constitute this *Encyclopedia and Dictionary*:—

		Articles.			
Vol.	Scope.	Long.	Medium.	Short.	Total.
I.	Aac—Bra	75	65	834	974
II.	Bre—Ear	80	200	1,478	1,758
III.	Ear—Gum	60	102	935	1,097
IV.	Gum—Int	52	54	766	872
V.	Inu—Lum	47	69	792	908
VI.	Lum—Neph	48	82	1,098	1,228
VII.	Ner—Phy	57	75	1,190	1,322
VIII.	Phy—Rhi	58	60	919	1,037
IX.	Rhi—The	68	107	1,413	1,588
X.	Thi—Zym	64	54	941	1,059
	Totals .	609	868	10,366	11,843

The articles, therefore, which may be said to constitute the *Encyclopedia*, number 609, while the subject-headings representing the *Dictionary* amount to 10,366; contributions intermediate in size account for 868 paragraphs. The grand total of subject-headings for the ten volumes is, therefore, 11,843, an average of nearly 1200 per volume.

The *Quinquennium of Medicine and Surgery* is in active preparation, and will appear in the Spring or Summer of 1909. It will contain an account of the additions which have been made to our knowledge of Medicine, of Surgery, of Midwifery, and of the specialties during the past five years. Special emphasis will be laid upon the advances which have stood

the test of time and which may now be fairly regarded as permanent additions to the common store of medical and surgical means of diagnosis and treatment. Transient phases of opinion and unsuccessful modes of treatment (however much vaunted on their introduction) will be very shortly dealt with. The *Quinquennium*, therefore, will be something more than a record of the work of five years in medicine: it will attempt to place new methods and new views in their proper perspective; it will endeavour to distinguish between what is new and of value and what is only novel. To attain this end special subjects have been placed in the hands of those who are specially qualified to deal with them, and estimates rather than abstracts have been asked for. It is confidently anticipated that the readers of the *Encyclopedia and Dictionary* will find in the *Quinquennium* a useful supplementary volume to the former work, and a trustworthy guide to the really important additions which the past five years have made to our means of combating disease, deformity, and death.

J. W. BALLANTYNE.

24 Melville Street, Edinburgh.
December 1908.

CONTENTS

THE CHIEF ARTICLES IN VOLUME X., WITH THEIR AUTHORS

	PAGE
THYMUS GLAND. J. S. Fowler, M.D., F.R.C.P.E.	3
THYROID GLAND—	
MEDICAL. G. R. Murray, M.D., F.R.C.P.	5
SURGICAL. Frederick Page, F.R.C.S.	19
TINNITUS AURIUM. Dundas Grant, F.R.C.S.	26
TONGUE. Alexander Miles, F.R.C.S.E.	30
TONSILS. Herbert Tilley, M.D., F.R.C.S.	42
TOXICOLOGY. J. Dixon Mann, M.D., F.R.C.P.	48
TRACHEA. L. P. Gamgee, F.R.C.S.	80
TRADES, DANGEROUS. Thomas Oliver, F.R.C.P.	84
TRANSFUSION. J. M. Cotterill, *P*.R.C.S.E.	101
TREMORS. E. Farquhar Buzzard, F.R.C.P.	104
TROPICAL FEVERS. A. Crombie, M.D. Ed.	110
TUBERCULOSIS. Theodore Shennan, M.D., F.R.C.S.E.	118
TUMOURS. J. Bland-Sutton, F.R.C.S.E.	151
TREATMENT OF INOPERABLE TUMOURS. G. T. Beatson, M.D.	164
TUMOURS OF THE SKIN. George Pernet, M.R.C.S.E.	168
TYPHOID FEVER. Sydney Phillips, F.R.C.P.	176
TYPHUS FEVER. Claude B. Ker, M.D., F.R.C.P.E.	203
ULCERS. A. Miles, F.R.C.S.E.	210
UMBILICUS. William Sheen, M.D., F.R.C.S.	219
UNCONSCIOUSNESS. T. B. Hyslop, M.D., F.R.C.P.	221
UNDULANT FEVER. F. J. A. Dalton, M.R.C.S., Surgeon Captain R.N.	231
URACHUS. Alban Doran, F.R.C.S.	240
URÆMIA. Nestor Tirard, M.D., F.R.C.P.	243
URETHRA. D'Arcy Power, F.R.C.S.	251
URIC ACID. T. H. Milroy, M.D., B.Sc.	262
URINE—	
GENERAL EXAMINATION OF, ETC. A. E. Garrod, M.D., F.R.C.P.	269
BACTERIOLOGICAL EXAMINATION. J. H. Drysdale, M.D., F.R.C.P.	307

PAGE

URTICARIA. J. M. H. Macleod, M.D., M.R.C.P. 313

UTERUS—

DEVELOPMENTAL ERRORS, ETC. A. E. Giles, M.D., B.Sc., M.R.C.P., F.R.C.S.E. . 319

DISPLACEMENTS. N. T. Brewis, M.D., F.R.C.P.E. 327

INFLAMMATION OF, ETC. W. Fordyce, M.D., F.R.C.P.E. 340

TUMOURS—

BENIGN. Mrs. Scharlieb, M.D., M.S. 348

MALIGNANT. J. Bland-Sutton, F.R.C.S. 363

INSANITY ASSOCIATED WITH DISEASE OF UTERUS AND ANNEXA. Lewis Bruce, M.D., F.R.C.P.E. 376

UVULA. J. Middlemass Hunt, M.B. 378

VACCINATION. Mrs. Garrett Anderson, M.D. 380

VAGINA. T. Wilson, M.D., F.R.C.S. 401

VAGUS NERVE. Edwin Bramwell, M.B., F.R.C.P. 414

VARICELLA. J. W. Allan, M.B. 416

VEINS. D. Waterston, M.D., F.R.C.S.E. 420

VENEREAL DISEASES. D'Arcy Power, F.R.C.S. 427

VENTILATION. Leslie Mackenzie, M.A., M.D., D.Ph. . . . 433

VERRUGA PERUANA. R. W. Felkin, M.D., F.R.S.E. 441

VERTIGO. Purves Stewart, M.D., F.R.C.P. 444

VESICULÆ SEMINALES. Jordan Lloyd, F.R.C.S., M.S. 448

VICE. The late G. R. Wilson, M.D., M.R.C.P.E. 458

VISCERAL PAIN. James Mackenzie, M.D., Burnley 466

VISION, FIELDS OF. A. H. H. Sinclair, M.D., F.R.C.S.E. 480

VITAL STATISTICS. A. Newsholme, M.D., F.R.C.P. 487

VITREOUS. E. Treacher Collins, F.R.C.S. 498

VULVA. A. E. Giles, M.D., M.R.C.P. 504

WARTS. George Pernet, M.R.C.S. 510

WATER. J. C. Thresh, M.D., D.Sc., D.P.H. 512

WHOOPING-COUGH. James Carmichael, M.D., F.R.C.P.E. 526

WOUNDS. J. T. J. Morison, M.D., F.R.C.S. 533

WRIST-JOINT—

INJURIES. E. H. Bennett, M.D., F.R.C.S.I. 542

DISEASES. J. Shaw M'Laren, F.R.C.S.E. 553

WRY-NECK. Edmund Owen, F.R.C.S. 558

X-RAYS. Dawson Turner, M.D., F.R.C.P.E. 565

YAWS. F. H. A. Clayton, M.D., Surgeon, Royal Navy 585

YELLOW FEVER. Andrew Davidson, M.D., F.R.C.P.E. 588

LIST OF ILLUSTRATIONS

PAGE

Typhus Fever—
Chart 1. Showing temperature from the second day in a severe case . . . 206
,, 2. Showing temperature with slight remission in a mild case . . . 207
Undulant Fever—
Chart A. Typical of the remittent type of Fever 235
,, B. Typical of the continuously low form of Pyrexia 236
,, C. Illustrating case starting with continuously high form of Pyrexia and then taking on the remittent type 236
,, D. Illustrating in a marked manner the waves of Pyrexia 236
Uterus, Malformations of the—
Fig. 1. Diagram of the mode of development of the Female Genital Passages . . 320
,, 2. Uterus Unicornis 322
,, 3. 1*a*, Uterus Septus; 2*a*, Uterus Bicornis; 1*b*, Uterus Subseptus; 2*b*, Uterus bicornis Unicollis; 3, Uterus Unicornis; 4, Uterus Didelphys . . 323
,, 4. Uterus Didelphys 324
,, 5. Effects of Atresia on the Genital Passage 326
Uterus, Displacements of the—
Fig. 1. (*a*) Represents action of utero-sacral ligament; (*b*) intra-abdominal pressure . 328
,, 2. Position of the Organs consequent on displacement of the Pelvic Floor . 330
,, 3. Anterior Colporrhaphy (Stoltz) 331
,, 4. Bilateral Colporrhaphy 334
,, 5. Restoration of the Perineum when the Rectum has been torn and the External Sphincter Muscle divided 334
,, 6. Retroversion of the Uterus 335
Uterus, Malignant Tumours of the—
Fig. 1. Tubular Glands of the Uterus in section (magnified) 364
,, 2. Gland from the Cervical Endometrium (magnified) 364
,, 3. Microscopic characters and disposition of the Epithelium on the vaginal aspect of the neck of the Uterus 365
,, 4. Microscopic characters of Cancer beginning in the Endometrium of the Cervical Canal 365
,, 5. Cancerous Uterus in sagittal section 366
,, 6. Pelvis and its Viscera in section. From a case of cancer of the uterine cervix which invaded the bladder 367
,, 7. Cancer of the Cervix Uteri implicating the Ureter and Bladder . . 368
,, 8. Cancerous Uterus in sagittal section. A small process of cancer protruded through the uterus and infected the peritoneum 370
,, 9. Microscopic characters of Cancer of the Corporeal Endometrium . . 370
,, 10. Cancerous Uterus in sagittal section. A process of the cancer has crept into the right Fallopian tube 371
,, 11. Uterus in coronal section, occupied with a cancerous mass . . . 371
,, 12. Microscopic characters of the "Tubular" Cancer shown in Fig. 11 . . 372
,, 13. Cancer of the Uterus complicated with a Submucous Fibroid . . . 373
Visceral Pain—
Fig. 1. Distribution of Hyperæsthesia and Pain after the first attack of Angina Pectoris 470
,, 2. ,, ,, ,, ,, after repeated attacks of ,, ,, 470
,, 3. ,, ,, in a case of Renal Calculus . . . 470
, 4. ,, ,, ,, ,, Gall-stone Colic . . . 472

PAGE

Visceral Pain—*continued.*
Figs. 5 and 6. Showing area of Hyperæsthesia and Pain varying with site of Gastric Ulcer 474
Fig. 7. Site of Pain in Ulcer at pyloric orifice of stomach 474
„ 8. Distribution of superficial cutaneous Hyperæsthesia in a case of Acute Peritonitis 477
„ 9. Situation of Pain and Hyperæsthesia in Diaphragmatic Pleurisy . . 478

Vision, the Field of—
Chart 1. Normal Field of Vision 480
„ 2. „ „ for white by Bjerrum's method 481
„ 3. Peripheral limitation of the field as found in Hysteria . . . 481
„ 4. Partial Ring Scotoma from a case of Retinitis Pigmentosa . . . 482
„ 5. Detachment of Retina 482
„ 6. Central Choroiditis 482
„ 7. From a case of well-marked Tobacco Amblyopia 483
„ 8. Absolute Scotoma, which began with an attack of Retrobulbar Neuritis and became permanent 484
„ 9. Homonymous Hemianopia 484
„ 10. Showing the binocular field in Bitemporal Hemianopia . . . 485
„ 11. Bitemporal Hemianopia 485
„ 12. Scotomatous Atrophy, with peripheral restriction of the field . . . 486
„ 13. Giving the field for White and for Red as taken in a case of Glaucoma . 486

Wrist-joint Injuries—
Fig. 1. Colles' Fracture (from a cast) 546
Figs. 2 and 2*a*. Skiagrams of Colles' Fracture 547
Fig. 3. Showing position of Radius and Ulna in Colles' Fracture . . . 549
„ 4. Antero-posterior section of united Fractures 549
„ 5. Showing lines of fracture of Carpal Surface 550
Figs. 6 and 7. Section showing impaction of Upper and Lower Fragments . . 550
Fig. 8. Casts of Epiphysary Separation and Colles' Fracture 551
Figs. 9 and 10. Fractures of lower end of Radius (articular surfaces) . . 552
Fig. 11. Fracture of styloid process of Radius 552

X-Rays—
Fig. 1. Wimshurst Influence Machine 566
„ 2. Induction Coil 567
„ 3. Arrangement for altering self-induction of Coil 567
„ 4. Dipping Contact Interrupter 568
„ 5. Mackenzie Davidson Interrupter 568
„ 6. Mercury Jet Interrupter 569
„ 7. Wehnelt Interrupter, new pattern 569
„ 8. Wehnelt Spark 569
„ 9. Dawson Turner Interrupter 570
„ 10. Bichromate Battery with lifting arrangement 570
„ 11. Switchboard for supplying Coil from the Main 570
„ 12. Röntgen Ray Tube 570
„ 13. Röntgen Ray Tube, with arrangement for altering the Vacuum . . 571
„ 14. Röntgen Ray Tube with movable Cathode 571
„ 15. Tube-holder for Tube 571
„ 16. False Teeth in Œsophagus 572
„ 17. Aneurysm of ascending Aorta 573
„ 18. Congenital Dislocation, Left Hip-joint 574
„ 19. Pelvic Sinus with Probe 575
„ 20. London Hospital Lamp 578
„ 21. Dr. Bang's Lamp. Dermo pattern 579
„ 22. Dr. Strubel's Lamp 579
„ 23. Condenser Spark Lamp 579
„ 24. High Frequency Apparatus 580
„ 25. „ „ „ 581
„ 26. Autoconduction 581
„ 27. High Frequency Electrodes 582

Yellow Fever—
Charts 1–4. Temperature Curves 597
„ 5 and 6. Temperature Curves 598

ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

Thiersch's Method.—A method of skin-grafting in which long strips containing half the thickness of the skin are employed. *See* SKIN GRAFTING AND ALLIED PROCEDURES (*Methods*).

Thiersch's Operation.—An operative procedure for the cure of ectopia vesicæ (two lateral flaps) or of epispadias (four stages). *See* BLADDER, INJURIES AND DISEASES (*Malformations*); URETHRA, DISEASES OF (*Abnormalities*).

Thigh. *See* ARTIFICIAL LIMBS (*Thigh*); FRACTURES (*Femur*).

Thio- and Thi-.—In compound words *thio-* and *thi-* (Gr. θεῖον, brimstone or sulphur) means relating to or containing sulphur; e.g. *thigenol* (a soda salt of a sulphur acid used in skin diseases), *thilanin* (a substance resembling lanolin, containing 3 per cent of sulphur, and used in skin diseases), *thiocyanic acid* (sulphocyanic acid), and *thioform* (dithiosalicylates of bismuth).

Thiol.—Sulphothiolate of ammonium, used in skin diseases in the same way as ichthyol.

Thionin.—A sulphur compound (C_4H_4S), forming a dark blue solution, used as a stain in microscopic work; Louth's violet.

Thioresorcin.—A yellowish powder ($C_6H_4(SO)_2$), used as a dusting powder in place of iodoform; thioresorcinol.

Thiosinamine.—Sulphocarbamide of allyl ($C_4H_8N_2S$), prepared from mustard oil (C_3H_5NCS) by the action of strong solution of ammonia; it is crystalline, readily soluble in water, and has been used in lupus, night-sweats, and hypertrophy and stricture of the pylorus as a hypodermic injection (10 minims of a 10 per cent solution).

Thiothrix. *See* MICRO-ORGANISMS (*Bacteria, Thiothrix*).

Third Nerve. *See* OCULAR MUSCLES, AFFECTIONS OF (*Paralysis, Double Vision*); BRAIN, PHYSIOLOGY OF (*Cranial Nerves, Third*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Vascular Lesions of Crus and Pons*); BRAIN, TUMOURS OF (*Localising Symptoms, Cranial Nerves*); PHYSIOLOGY, CENTRAL NERVOUS SYSTEM (*Cranial Nerves*); SYPHILIS (*Tertiary, Eye and its Appendages, Diplopia*).

Thirst. *See* ABDOMEN, INJURIES OF (*Symptoms*); DIABETES INSIPIDUS (*Symptoms*); DIABETES MELLITUS (*Symptomatology*); HYPNOTISM (*Experimental Phenomena*); PERITONEUM, ACUTE PERITONITIS, GENERAL (*Thirst after Operation*); PNEUMONIA, CLINICAL (*Treatment*); PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Common Sensibility, Thirst*); STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology, Local Sensory Affections*).

Thiry's Fistula.—A piece of bowel is separated from the rest without dividing its mesentery, one end of it is closed and the other is stitched to the abdominal wound, while the two ends of the intestine from which it was removed are joined together; from the separated piece of intestine the secretions can be obtained. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Intestinal*).

Thlapsis Depressio.—Depression or melancholy, a "crushed" feeling (Gr. θλάω, I crush or bruise).

Thlipsencephalus.—A form of anencephalus in which the basis cranii, covered by a vascular mass, is exposed through defect of the cranial vault, and in which the degree of spina bifida is slight; the name is an indication of a belief in the pressure or mechanical theory

of origin of such monstrosities (Gr. θλῖψις, a pressure).

Thoma-Zeiss Hæmocytometer. *See* Blood (*Enumeration of Corpuscles*).

Thomas's Splint. — A long metal splint reaching from the thorax half-way down the leg.

Thompson's Fluid. — A solution, containing 1 oz. of borax, 2 fl. oz. of glycerine, and 2 fl. oz. of water; it is used for washing out the bladder in cystitis, in the proportion of half a fluid ounce of the fluid to four fluid ounces of warm water.

Thompson's Line.—A red line seen on the gums in pulmonary phthisis.

Thomsen's Disease.—Myotonia congenita. *See* Muscles, Diseases of the (*Myotonia Congenita*).

Thoracentesis.—Tapping or aspirating the thorax for the removal of fluid either for diagnosis or for treatment. *See* Pleura, Affections of, Surgical (*Exploration of the Pleura, Paracentesis Thoracis*).

Thoracic.—Relating to or belonging to the thorax or chest; *e.g.* the thoracic aorta, thoracic duct, thoracic nerves, and a thoracic aneurysm. *See* Ascites (*Causation, Chylous Ascites*); Lymphatic System, Physiology and Pathology (*Thoracic Duct*); Mediastinum (*Mediastinal Growths, Diagnosis*); Pregnancy, Intra-Uterine Diseases (*Fœtal Dropsy, Causes*).

Thoraco-.—In compound words *thoraco-* means relating to the thorax; *e.g. thoracoacromial* (relating to thorax and shoulder), *thoracocentesis* (thoracentesis), *thoracodidymus* (a double monster, the two bodies being joined by the thoraces), *thoracodynia* (pain in the chest), *thoracogastroschisis* (an open condition of both the abdominal and the thoracic cavities), *thoracopagus* (fused twins in which parts of the thoraces are united), *thoracoschisis* (fissure or an open condition of the thorax), etc.

Thoracoplasty.—Thorax resection or Estländer's Operation, consisting of resection of the ribs in order to close an empyemic cavity. *See* Pleura, Surgical Affections of (*Thoracoplasty*).

Thorax. *See* Asphyxia (*Resuscitation, Methods*); Chest, Clinical Investigation of; Chest, Deformities of; Chest, Injuries of; Chest-Wall, Affections of; Lymphatic System (*Lymphatic Glands, Thorax*); Pleura, Affections of, Surgical (*Thorax Resection*); Post-mortem Methods (*Examination of the Body Cavities*); Syringomyelia (*Thorax en Bateau*); Teratology (*Malformations of Thorax*); etc.

Thornwaldt's Disease.—A variety of naso-laryngeal stenosis due to cystic enlargement of the pharyngeal tonsil or gland; Thornwaldt's bursitis.

Thought-Reading or Thought-Transference.—The apparent reading of the thoughts of others by personal contact; it is really the appreciation of involuntary muscular movements as indications of the tendency of the thoughts of the person who is being experimented on.

Thread Worms. *See* Parasites (*Nematodes, Oxyuris Vermicularis*); Appendix Vermiformis (*Appendicitis, Causes, Thread Worm in Appendix*).

Three Days' Fever. *See* Dengue (*Synonyms*).

Thremmatology. — Experimental evolution (Gr. θρέμμα, that which is reared, or a nursling).

Thresh's Disinfector.—A disinfecting apparatus in which steam at a temperature a little above 100° C. is used; the superheated steam is got from water impregnated with calcium chloride, and the disinfection takes half an hour.

Thresh's Method.—A means of estimating the amount of oxygen in water by means of sulphuric acid, nitrite of sodium, iodide of potassium, thiosulphate of sodium, and starch solution.

Thrills. *See* Aneurysm (*Signs and Symptoms*); Chest, Clinical Investigation of (*Heart, Palpation of Chest Wall*); Heart, Affections of Myocardium and Endocardium (*Physical Examination of Heart Thrills*); Hydatid Disease (*General Symptomatology*).

Throat. *See* Children, Clinical Examination of (*Mouth and Throat*); Galvanic Cautery (*Uses, Diseases of Throat*); Hæmoptysis (*Diagnosis from Hœmorrhage from Throat*); Larynx; Neck, Region of (*Injuries, Cut Throat*); Syphilis (*Tertiary, Mouth and Throat*); etc.

Thrombin or Thrombase.—The enzyme which causes clotting of the blood; its precursor is *prothrombin*, and it is supposed that another enzyme (*thrombokinase*) is needed to act upon the prothrombin and lime salts to initiate the process. *See* Physiology, Blood and Lymph (*Blood, Coagulation*).

Thrombo-. — In compound words *thrombo-* (Gr. θρόμβος, a lump or clot) means relating to a clot; e.g. *thrombo-arteritis* (inflammation of an artery associated with thrombosis), *thrombo-phlebitis* (inflammation of a vein associated with thrombosis), etc.

Thrombokinase. *See* THROMBIN.

Thrombosis. *See* ALCOHOLISM (*Complications, Thrombosis*); APPENDIX VERMIFORMIS (*Results of Infection of Appendix*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Thrombosis in Cerebral Veins and Sinuses*); BRAIN, SURGERY OF (*Infective Thrombosis of Venous Sinuses*); CHLOROSIS (*Symptoms, Thrombosis of Veins of Leg*); GANGRENE (*Indirect Traumatic, Causes*); GASTRO-INTESTINAL DISORDERS OF INFANCY (*Chronic Diarrhœa, Complications*); GOUT (*Circulatory System*); HEMIPLEGIA (*Condition at Outset*); LEUCOCYTHÆMIA (*Symptoms*); LUNGS, VASCULAR DISORDERS OF (*Thrombosis of Pulmonary Arteries*); NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Pathology, Intra-Cranial Complications*); ORBIT, DISEASES OF (*Thrombosis of Cavernous Sinus*); OVARIES, DISEASES OF (*Complications following Ovariotomy*); PREGNANCY, AFFECTIONS AND COMPLICATIONS OF (*Cardio-Vascular Disturbances, Varicose Veins*); PUERPERIUM, PATHOLOGY (*Puerperal Venous Thrombosis*); RHEUMATISM ACUTE (*Symptoms, Phlebitis and Venous Thrombosis*); SPINAL CORD, MEDICAL (*Thrombosis of Spinal Vessels*); SYPHILIS (*Visceral, Cerebral Thrombosis*); TYPHOID FEVER (*Complications, Thrombosis*); UNCONSCIOUSNESS (*Causes*); VEINS, DISEASES OF (*Thrombosis and Phlebitis*).

Thrombus.—A blood clot formed in the heart or in a vessel on account of disease of the blood or of the vessel walls, or, simply, to slowing of the circulation. *See* THROMBOSIS and *Cross References.*

Thrush.—Parasitic stomatitis. *See* STOMATITIS (*Parasitic*); GASTRO-INTESTINAL DISORDERS OF INFANCY (*Diseases of the Œsophagus*).

Thuja.—A non-official plant (*Arbor vitæ*) belonging to the Coniferæ, containing a volatile oil and a bitter principle called *thujine* ($C_{20}H_{22}O_{12}$); it acts as an astringent and antiperiodic; externally, it has been applied to warts, and internally it is used in malaria, dropsy, rheumatism, scurvy, and gonorrhœa; the dose is ½ to 1 fl. dr. of either the tincture or the fluid extract.

Thumb. *See* AMPUTATIONS (*Upper Extremity, Hand*).

Thus Americanum. *See* FRANKINCENSE.

Thyme, Oil of.—A volatile oil obtained from *Thymus vulgaris* (thyme); it is official in the U.S. Pharmacopœia; dose, 3 minims.

Thymectomy.—Excision of the thymus gland (*q.v.*).

Thymic.—Relating to the thymus gland, *e.g.* thymic asthma. *See* ASTHMA (*Explanation of Term*); STATUS LYMPHATICUS; THYMUS GLAND (*Thymus Death*).

Thymol.—A crystalline substance derived from the oil of thyme and other volatile oils. It is almost insoluble in water, but freely in oils, alcohol, ether, and chloroform An oily liquid results when it is rubbed with menthol, camphor, chloral hydrate, or phenol. *Dose*—½-2 grs. as a pill. It is powerfully antiseptic and comparatively non-irritating, but it is too expensive for general use for surgical purposes. It has been used as a mouth-wash or gargle in stomatitis, sore throat, etc. Internally it has been tried as an intestinal antiseptic, but without any striking result. As an anthelmintic, however, especially for ankylostomiasis, it is of great value. For this purpose it should be administered in large doses (10-30 grs.), followed by a purgative. The simultaneous administration of any solvent of thymol (*e.g.* alcohol) should be avoided. Large doses of thymol may render the urine green in colour. It has been employed in the treatment of various skin diseases, including psoriasis, eczema, scabies, and ringworm. A weak solution has been recommended as a spray for burns. "Glycothymoline" is a popular proprietary preparation for catarrhal conditions of the nose, throat, vagina, etc. THYMOL IODIDE, known as *Aristol*, was introduced as a substitute for iodoform. It is very useful for a variety of purposes and gives excellent results, but it is somewhat costly.

Thymus Gland.

ANATOMY AND PHYSIOLOGY . . . 3
DISEASES OF THYMUS—
1. *Tuberculosis* 4
2. *Syphilis* 4
3. *Inflammation* 4
4. *Tumours, etc.* 4
5. *Hæmorrhages* 4
6. *Persistent Thymus* . . . 4

See also STATUS LYMPHATICUS; ACROMEGALY (*Symptoms, Respiratory System*); ADRENAL GLANDS, ADDISON'S DISEASE (*Morbid Anatomy, State of Thymus*); CHILDREN, DEVELOPMENT OF (*Glandular Organs*); LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY (*Status Lymphaticus or Lymphatism, Thymus-Tod*); MEDIASTINUM (*Tumours of Thymus*); POST-MORTEM METHODS (*Thorax*); RICKETS (*Treatment*); THYROID GLAND, MEDICAL (*Exophthalmic Goitre, Pathological Anatomy*); TUMOURS, INOPERABLE, TREATMENT OF (*Organotherapy*).

THE thymus gland is a structure of very variable dimensions in different subjects; its size appears to depend to some extent on the general nutrition, being greater in well-nourished infants than in those who are emaciated. It attains its maximum development at the end of the second year, after which it slowly diminishes in

size till puberty, when rapid fatty degeneration takes place and the organ practically disappears, becoming infiltrated with connective tissue, in which, however, remains of the gland may here and there be found. Its average length during the first two years is 2½ ins. (varying between 1¼ and 3½ ins.); its breadth is from ¾ to 1½ ins.; and its weight from a half to one ounce. It consists of two lobes of unequal size, separated by a mesial fissure, and lies in the superior and anterior mediastinal spaces, and partly in the neck. It extends down to about the fourth costal cartilage, and comes into relation anteriorly with the sternum and costal cartilages, laterally with the pleuræ and lungs, and posteriorly with the pericardium, trachea, and vessels of the neck. In specimens hardened *in situ* by formalin the deep surface of the gland is found to show a concavity for the pericardium and deep grooves for the pulmonary artery, innominate veins, and superior cava (Cunningham). The two lobes of the thymus ascend in front of the trachea to an unequal distance, usually, however, falling short of the thyroid gland, and extending outwards as far as the carotid arteries. The blood-supply is from the inferior thyroid and internal mammary arteries; the nerves of the gland are derived from the vagus and sympathetic.

The thymus is a ductless gland, and consists of a number of lobules separated from one another by connective-tissue septa arising from its sheath. In the clusters of lymphoid tissue which compose the lobules, cortical and medullary layers can be distinguished. In the latter the connective-tissue matrix is coarser and the lymphoid cells fewer; and in the medulla, also, the characteristic thymus structures—Hassall's corpuscles—are placed. These consist of concentric layers of flattened epithelial cells surrounding a granular nuclear corpuscle.

The thymus develops about the seventh week of fœtal life as a tubular diverticulum from the third visceral cleft. The walls of the diverticulum are composed of epithelium which proliferates rapidly, and as the diverticulum grows downwards it becomes expanded laterally in its lower part. Finally, the connection between the gland and the pharyngeal cleft from which it arose is severed, and the epithelial structure is invaded and almost entirely replaced by adenoid and connective tissue, the only remnant of its original constituents left in the mature gland being the Hassall's corpuscles already referred to.

Function of the Thymus.—Little is known as to the function of the thymus. In all probability it is the same as that of the lymphatic tissues elsewhere, the thymus juice containing numerous leucocytes which find their way into the general circulation through the veins of the gland. From the fact that it is usually persistent and enlarged in cases of Graves' disease, and sometimes in acromegaly and myxœdema, the idea suggests itself that it may have some function (an internal secretion?) correlated with that of other ductless glands. Svehla found that the intravenous injection of thymus extract produces (in dogs) a great fall in the blood pressure and acceleration of the heart, and, in lethal doses, dyspnœa and collapse. But in reality little is known as to the physiology of the organ.

Diseases of the Thymus.—The thymus is for the most part inaccessible to the ordinary methods of physical examination, though in young infants a little dulness may be made out over the upper part of the sternum as low as the third rib. The majority of the morbid changes to which the organ is liable are, either because of their rarity or because they are insusceptible of clinical diagnosis, of pathological interest alone. Only one condition, in fact,—hypertrophy of the thymus,—is of any practical importance; *see* Status Lymphaticus; the other diseases of the organ may be dealt with very briefly.

1. *Tuberculosis* is on the whole rare. It may occur as a primary lesion, but is more commonly part of a generalised tuberculosis. Jacobi found it thrice in children under one year, in each case as a part of a generalised miliary tuberculosis.

2. *Syphilis.*—In cases of hereditary syphilis the usual vascular lesions and increase of connective tissue are sometimes found. Small multiple abscesses are also said to occur, but there is some doubt as to whether these are really of an inflammatory nature, as the secretion of the thymus gland bears considerable resemblance to pus.

3. *Inflammation and Abscess Formation.*—Though rare, there seems no doubt that some cases of mediastinal abscess have had origin in the thymus. Chronic inflammation of the capsule may occur secondarily to pleurisy or mediastinitis. Necrotic foci were found by Jacobi in some cases of diphtheria.

4. *Tumours, etc.*—Sarcomata, lymphadenomata, carcinomata, lipomata, cysts, of uncertain origin, containing clear yellowish serum, and possibly other tumours, may develop in the thymus gland.

5. *Hæmorrhages* are not infrequently found, both in cases of "thymus-death" and apart from that condition in young infants dying of atelectasis, convulsions, whooping cough, asphyxia neonatorum, or, in short, of any disease attended by circulatory disturbance. They may in some cases be due to syphilis or to hæmophilia.

6. *Enlarged and Persistent Thymus.*—This is met with in the adult in several conditions, notably in exophthalmic goitre, of which it is a frequent accompaniment. As a lymphoid organ the thymus is also often enlarged in leukæmia.

Less constantly, but still with sufficient frequency to warrant notice, we find described persistence or enlargement in acromegaly, Hodgkin's disease, and myxœdema. Except in leukæmia and Hodgkin's disease the relation of the enlargement to the general malady is obscure. In these conditions the enlargement is clinically unimportant, but such is not the case where we have an enlarged thymus, either in children or in adults, apart from another general disease.

"Thymus-Tod."—Literally, thymus-death; sudden death due to enlargement of the thymus gland. *See* STATUS LYMPHATICUS.

Thynnus. *See* SNAKE-BITES AND POISONOUS FISHES (*Thynnus or Tunny Fish*).

Thyro- or Thyreo-.—In compound words *thyro-* (Gr. θυρέος, an oblong shield) means relating to the thyroid gland or cartilage; e.g. *thyro-arytenoid* (relating to the thyroid and arytenoid cartilages), *thyro-antitoxine* (a preparation of thyroid gland or a proteid contained in the thyroid gland), *thyro-colloid* (a proprietary preparation of sheep's thyroid), *thyrocele* (goitre), *thyroepiglottic* or *thyroepiglottidean* (belonging to the thyroid cartilage and the epiglottis), *thyroglandin* (a compound of iodo-globulin and thyroidin, used in myxœdema and obesity), *thyroglobulin* (the iodine-proteid secreted by the thyroid gland and contained in the colloid), *thyroglossal* (belonging to the thyroid gland and the tongue), *thyrohyoid* (belonging to the thyroid cartilage and the hyoid bone). *See* THYROID GLAND; LARYNX; etc.

Thyroidectomy.—Excision, in whole or part, of the thyroid gland or thyroid cartilage. *See* THYROID GLAND, SURGICAL SECTION.

Thyroid Gland, Medical Section.

ANATOMY AND PHYSIOLOGY . . . 5
PARATHYROID GLANDS 6
MYXŒDEMA 7
Clinical Features 7
Treatment 10
EXOPHTHALMIC GOITRE 10
Etiology 11
Clinical Features 11
Treatment 16
ACUTE THYROIDITIS 18
GOITRE 18
Varieties 18
Etiology 19
Symptoms 19
Treatment 19
MALIGNANT DISEASE 19
SURGICAL SECTION 19

See also ANEURYSM (*Neck, Diagnosis from Blood Cyst of Thyroid*); BONE, DISEASES OF (*Secondary Tumour from Thyroid in Bone*); CRETINISM (*Morbid Anatomy*); HEART, MYOCARDIUM AND ENDOCARDIUM (*General Pathology, Etiology, Effect of Thyroid*); INDIGESTION (*Classification, Arrest of Thyroid Gland Secretion*); INSANITY, GENERAL TREATMENT OF (*Thyroid Extract*); LARYNX, NEUROSES OF (*Paralysis of Vocal Cords*); LARYNX, NEUROSES OF (*Hypertrophy of Lingual Tonsil*); MAMMARY GLAND, DISEASES OF (*Cancer, Inoperable, Thyroid Extract*); MENTAL DEFICIENCY (*Cretinoid Cases*); NECK, REGION OF (*Enlargements of Thyroid Gland*); OBESITY (*Treatment*); PANCREAS, PHYSIOLOGY OF (*Glycosuria following Excision of Thyroid*); PHYSIOLOGY, INTERNAL SECRETIONS OR HORMONES (*Thyroid Gland*); PREGNANCY, AFFECTIONS OF (*Thyroid Enlargement*); PSORIASIS (*Treatment*); RHEUMATISM, ACUTE (*Symptoms, Exophthalmic Goitre*); TETANY (*History, Extirpation of Thyroid*); TUMOURS, INOPERABLE, TREATMENT (*Organotherapy*).

ANATOMY AND PHYSIOLOGY

ANATOMY.—The thyroid gland is composed of three parts—the two lateral lobes and the isthmus. Each lobe is about two inches long and somewhat oval in shape, the upper end being narrower and more pointed than the lower. The whole gland is of a dark reddish-brown colour, and in the adult weighs from an ounce to an ounce and a half. The gland receives an abundant blood-supply from the thyroid arteries, which anastomose freely, and end in a network of capillaries for each alveolus. The veins unite and form the superior, middle, and inferior thyroid veins. The lymphatics take origin from the lymph spaces of the connective tissue which surround each alveolus. They unite and ultimately open into the jugular lymphatic trunk, which on the left side opens into the thoracic duct, and on the right into the right lymphatic duct.

The gland is invested by a fibrous capsule, extensions of which run into the gland, and subdivide into a supporting framework for the vesicles of the gland.

The gland consists of a number of alveoli, which are rounded or polyhedral, with a diameter varying from 0·045 to 1 mm. A few of the follicles appear as a mass of cells; in the great majority, however, there is a central space filled with the colloid substance. Each alveolus is lined by a layer of epithelial cells, two kinds of which may be distinguished—the chief cells, which are cubical in shape, measuring from 8 μ to 16 μ in height by 6 μ to 8 μ in breadth, and the colloid cells, which measure from 7 μ to 9 μ in height.

The colloid substance which fills the centre of the alveolus is a glairy fluid and yellowish in colour, and in it red blood corpuscles, leucocytes, and epithelial cells may sometimes be seen. The colloid substance is secreted by the epithelial cells which line the alveolus, in the

lumen of which it is stored till required. As the gland has no duct the secretion is conveyed from the gland by the lymphatics, and possibly also by the veins. It was shown by King and by Horsley that the colloid substance could be squeezed from the centre of the acinus into the lymphatic spaces which lie outside and between the alveoli. Thence it passes directly into the lymphatic vessels which drain the gland. Hurthle has shown that the colloid substance escapes from the alveolus, either by rupture of the alveolar wall, or by passing through minute channels between the epithelial cells into the interacinous lymphatic spaces. The secretion of the gland is thus mixed with the lymph, with which it passes into the general blood-stream, and is distributed all over the body.

PHYSIOLOGY.—The thyroid gland is thus a ductless gland, the function of which is to supply an internal secretion which is poured into the blood. The important part played by this secretion in the general metabolism of the tissues is shown by the results of removal of the thyroid gland in man and lower animals. Thus in rabbits removal of the thyroid gland is followed by the gradual development of hebetude, swelling of the subcutaneous tissues, dryness of the skin, loss of hair, and subnormal temperature, all of which are symptoms of myxœdema in man. It was shown by Horsley that in the monkey especially thyroidectomy was followed by symptoms closely resembling those of myxœdema in man. In my own experience similar symptoms have also developed. Some five days after the operation a fine tremor of the limbs appears, and the animal loses all its natural liveliness, and becomes inactive and apathetic. There is progressive loss of muscular power, and the animal sits huddled up in a corner of its cage. Tonic and clonic contractions of the flexor muscles are often seen, and true epileptic fits occur in some cases. The temperature at first is irregular, but later becomes subnormal. From two to three weeks after the operation myxœdematous swelling appears in the face, the skin becomes dry, and the hair may be lost in places. It is thus evident that the symptoms closely resemble those of myxœdema in man. They develop more acutely, of course, because the gland is removed all at once instead of undergoing a progressive fibrosis as in primary myxœdema in man. In secondary myxœdema in man, however, the symptoms may develop almost as rapidly as in a case transferred to my care at the Newcastle Royal Infirmary, by my colleague Mr. A. Martin, who had removed the whole of the gland for disease. Within ten days of the operation there was swelling of the face and tongue, dryness of the skin, slow speech, with attacks of tetany, just like those which occur in the monkey.

In young animals removal of the thyroid gland is followed by arrest of development in addition to the other symptoms, so that a condition of cretinism is the ultimate result.

In man, as we shall see in considering secondary myxœdema, removal of the thyroid gland in the adult is followed by typical myxœdema. When the gland has been removed during childhood there has been an arrest of development as well, and the child has become a complete cretin.

When the importance of the thyroid gland was first demonstrated, it was thought that the results of thyroidectomy in an animal might be averted by grafting a piece of another gland into the animal. Schiff and von Eiselsberg showed by their experiments that in the cat and in the dog a piece of thyroid gland successfully transplanted was able to supply sufficient secretion to ward off the symptoms which usually follow thyroidectomy in these animals. Sir Victor Horsley then suggested that a similar method of treatment should be adopted in man for myxœdema, so as to arrest the inevitable downward progress of the disease to a fatal termination. Marked improvement followed this operation, notably so in cases recorded by Bettencourt and Serrano, by Bircher, and more recently by Dr. M'Pherson at Stirling. Unfortunately, in most of the cases thus treated it has been found that the grafted gland after a time shares the fate of the original gland and atrophies. It occurred to the writer that the supply of secretion might be continuously maintained by the internal administration of it, in the form of a glycerine extract of the sheep's thyroid gland. He selected the hypodermic method, and was soon able to show that when the supply of secretion was thus continuously maintained, the symptoms entirely disappeared in an advanced case of myxœdema. It was subsequently shown by Prof. Howitz, in Denmark, and by Dr. E. L. Fox and Dr. Hector Mackenzie, in England, that the same result was obtained more conveniently when the thyroid extract was given by the mouth.

These observations thus shortly described indicate clearly that the function of the gland is to supply an internal secretion to the blood, which plays a very important part in maintaining the normal metabolism of the tissues generally, more especially of the central nervous and cutaneous systems in the adults, and of the growing bones in the young.

THE PARATHYROID GLANDS.—Closely associated with the thyroid gland in animals are the parathyroid glands. In rabbits, cats, and dogs these glands can be removed separately from the thyroid gland. In the monkey they are embedded in the thyroid gland, and in thyroidectomy are removed along with it. We know nothing definitely yet of the results of disease of these glands in man, though it has been suggested that they may be affected in exoph-

thalmic goitre. Experimental removal of the parathyroids alone in animals has been followed by acute nervous symptoms, such as muscular tremors, clonic and tetanic contractions of voluntary muscles, loss of power, and emaciation. So that there is good evidence to show that they have some important function, and that further investigation will throw more light on their function in man.

Myxœdema

Etiology.—*Sex.*—Myxœdema is much more common in women than in men, as it occurs almost seven times as frequently in the former as in the latter.

Age. — Myxœdema rarely develops before twenty, after that it becomes more frequent each succeeding five years up to the age of forty-five, after which it gradually becomes less frequent. Thus in about one-third of all the cases the onset is between thirty-five and forty-five.

Locality.—Cases of myxœdema occur most frequently in temperate climates, and are rare in the tropics. They appear to be more common in Great Britain, especially in the north of England and south of Scotland, than in other European countries. The disease is well known in America and in Australia, but the coloured races are apparently exempt from it.

Heredity.—A certain predisposition to thyroidal disease occurs in some families, so that myxœdema may occur in one member, while some other form of thyroidal disease occurs in another. In some families a mother and daughter, two sisters, or a brother and sister have suffered from myxœdema.

Antecedents.—In a certain number of cases myxœdema has been preceded by exophthalmic goitre, and it is evident that recovery from the latter predisposes a patient to the development of the former. This can be easily understood if we remember that the development of fibrosis and atrophy of the enlarged gland, which is a natural method of recovery in exophthalmic goitre, will, if it progresses too far, of necessity lead to the development of myxœdema.

Excessive child-bearing appears to be another predisposing cause, for we find that the majority of cases are in women who have borne children.

Of the actual exciting cause of the thyroidal fibrosis, in the great majority of cases we know nothing. In one recorded case, syphilis, and in another, actinomycosis of the thyroid gland caused the myxœdema.

Symptoms.—*Onset.*—The symptoms of myxœdema develop slowly, and months may elapse from the commencement of ill-health before they become well defined. The length of the period of development depends upon the progress made by the atrophic cirrhosis of the thyroid gland, which is usually very gradual. In a few cases, especially in young adults, the symptoms have developed more rapidly, so that the nature of the disease was recognisable a few weeks, or even days, after the onset.

At the commencement a feeling of lassitude is often experienced, which gradually increases till even slight exertion becomes toilsome. This may be struggled against for a time, but sooner or later the patient is compelled to abandon active pursuits and lead a more or less sedentary existence. A feeling of chilliness on cold days is complained of at an early stage, which increases till there is a continued feeling of coldness, except in hot weather or when the temperature of the room is high. The temperature, which in the early stages may still be normal, later on falls one or two degrees lower.

The subcutaneous tissues gradually become distended by the myxœdematous swelling, which entails considerable alteration in the appearance of the face. At the first there is only an increased fulness and rounding of the features, which is most conspicuous round the eyelids and beneath the chin. The centre of each cheek is often flushed, though the skin of the rest of the face, in many cases, gradually becomes yellowish in colour. The lips are slightly thickened and the forehead transversely wrinkled. Careful examination will reveal the presence of a solid elastic œdema in the subcutaneous tissue in other parts of the body, notably above the clavicles, on the back of the hand and on the dorsum of the foot. Perspiration is diminished, so that the skin gradually becomes dry and wrinkled. The skin over the extensor aspect of the knees and elbows is specially liable to be rough and dry, even at an early stage. Subjective nervous symptoms may be present; thus both visual and auditory hallucinations may occur at a time when objective symptoms of the disease are only slightly developed.

Cutaneous System.—One of the most striking features of a fully developed case of myxœdema is the condition of the skin and subcutaneous tissues. Wherever the skin is loose, owing to the laxity of the subcutaneous tissues, the latter becomes distended by the characteristic solid œdema. Thus the face is full and rounded, and the eyelids are distended as if with the watery œdema of Bright's disease. The supraclavicular fossa, the back of the hand, and the dorsum of the foot are swollen also. All over the trunk and limbs a similar change can be observed, though here it more closely resembles an increase in the subcutaneous fat or the general œdema of renal disease. On palpation the swelling does not pit and feels more elastic than fat, and if the skin is punctured no serous fluid escapes. The appearance of the face is profoundly altered, as, in addition to the swelling of the eyelids, the cheeks may be pendulous, and the lips are thick and everted. The palpebral fissure is narrowed from falling of the upper

eyelid, which is to a certain extent counteracted by the instinctive contraction of the occipito-frontalis muscle, causing well-marked transverse wrinkling of the forehead. The nose and ears are also thickened, so that the appearance of the face is greatly altered, and it has a stolid, fixed expression, in consequence of which all advanced cases of myxœdema to a certain extent resemble each other. There may be a considerable increase in weight, and the solid œdema in some cases limits the mobility of the joints, so that there is difficulty in stooping or in bringing the hand up to the back of the neck.

The skin is dry and rough, as the secretion of the sebaceous and sweat glands ceases, and it feels cold to the touch. The superficial layers of the epidermis are frequently shed as a fine white powder. The skin of the face is yellowish in colour in some cases, in others it is white, while the centre of each cheek is usually occupied by a bright red flush, due to dilatation of the small cutaneous veins. On the extensor aspects of the joints, and on the palms of the hands and soles of the feet, the skin becomes very hard and dry, and may be cracked. On the back of the hand it is often wrinkled.

The hair is generally fine in texture and soft to the touch. On the head it is generally scanty, and in advanced cases there may be almost complete baldness, while the skin of the scalp becomes dry and scaly. Hair in other parts of the body may also be scarce or absent. The nails may be grooved and cracked, while the teeth are often carious. The mucous membrane of the mouth and nose is dry, and on the lips it is often pale and swollen as well.

The Thyroid Gland.—The thyroid gland is diminished in size in nearly all cases. In some it cannot be felt at all, and in others only the atrophied isthmus can be detected lying in front of the trachea. Rarely in slight cases it is larger than normal.

Temperature.—A sensation of chilliness may be complained of at an early stage, while in the later stages a feeling of coldness is a common symptom. This is naturally more marked in cold than in warm weather, so that in the winter much time is spent sitting close to the fire. In the early stages the temperature may be normal, but as the disease advances it gradually falls lower, and may be only from 93° F. to 95° F., or even lower before death.

Nervous System.—The functions of the nervous system are performed more slowly than in health. Thus there is marked slowness in the comprehension and also in the execution of anything new. The memory becomes defective, especially for names of people and things, and for recent events. The temper is generally placid and even. Daily duties, such as dressing and eating, are accomplished very deliberately and take up much time. Patients are often very sensitive about the alteration in their personal appearance, and consequently are apt to be shy. Sleep at night is good, and drowsiness may be felt during the day. Auditory and visual hallucinations are not uncommon, even in the early stages of the disease. Some ill-defined object may be seen moving rapidly across the floor, or some well-known figure may be actually seen. A noise like that caused by the opening of a door or actual voices may be heard. The patient is always fully conscious of the subjective nature of these phenomena.

Actual insanity occurs in some advanced cases of myxœdema, but, now that suitable treatment is usually adopted at an early stage of the disease, such cases are uncommon. The form of insanity has generally been acute or chronic mania, dementia, or melancholia.

All voluntary movements are slow. The gait is slow and awkward and the speech is deliberate. A general muscular weakness is not uncommon, and the legs may give way suddenly while the patient is standing or walking, so that she falls to the ground. Various subjective sensations may be complained of. Chilliness has already been mentioned. Headaches and neuralgic pains in various parts of the body are not infrequent. Giddiness occurs in some cases. Sensory impulses appear to be conveyed more slowly than in health, but the slowness of response may be partly due to the mental hebetude.

The special senses are often impaired. Thus the Myxœdema Committee of the Clinical Society found that in a small number of cases a general diminution of visual power and watering of the eyes were present. Hearing is more or less defective in half of the cases. I have seen with Dr. Dalgleish a woman who had been almost completely deaf, but whose hearing suddenly returned while she was being treated by thyroid extract. Taste and smell are impaired in some cases. Sexual feeling is usually diminished, and the power of reproduction is not usually retained in either sex.

Circulatory System.—In many cases, and especially in the early stages, the heart is unaffected. In advanced cases symptoms of fatty or fibroid degeneration of the cardiac muscle, such as dyspnœa, palpitation, and syncopal attacks, are not uncommon. In such cases the cardiac impulse is feeble and the heart sounds are weak. These cases require special care when treated by thyroid extract, as they are not fit for additional exercise till long after the improvement in the general condition would appear to warrant it.

The pulse is generally less frequent than in health, regular, and of low tension.

A moderate degree of anæmia is generally present; in some cases there is well-marked pallor. The red corpuscles, however, rarely fall below 3,000,000 per cubic millimetre, while

the hæmoglobin may fall to 60 or 70 per cent, or even as low as 40 or 50 per cent. Hæmorrhages are not uncommon, especially from the uterus at the menstrual period or during parturition. Bleeding may also take place from the nose, gums, throat, or bladder.

Digestive System.—Constipation is a common symptom, and dyspepsia occurs in some cases. The teeth are generally brittle and carious. The tongue is distinctly swollen in some cases. Hæmorrhoids are often present.

Urine.—The urine is usually normal in character. In some advanced cases the urine has a low specific gravity, varying from 1·008 to 1·018. In about one-fifth of the cases a small amount of albumin is present. If no renal disease is present the albuminuria disappears along with the other symptoms under treatment.

Morbid Anatomy.—*The Thyroid Gland.*—The thyroid gland is much diminished in size, and the weight is reduced in advanced cases to from fifty to seventy grains, or only about one-tenth of the normal weight. It is pale in colour and tough, and fibrous in consistence. Microscopical examination shows that there is a more or less advanced atrophy of the glandular epithelium, with general fibrosis of the whole gland. In advanced cases only a few alveoli may remain lined with epithelial cells and filled with colloid, while in some very advanced cases the whole gland is converted into fat and fibrous tissue, and not a single alveolus can be found in transverse section of one lobe of the gland. Such changes naturally lead to a gradual diminution and final arrest of the secretory activity of the gland.

Skin.—The skin is thickened as in life, and on section is somewhat translucent. The epidermal epithelium may be partly atrophied. The trabeculæ in the connective tissue of the corium are replaced by bundles of fine nucleated fibrillæ, and in some specimens the interfibrillar spaces are widened. In some cases there is endarteritis of the vessels. The sebaceous and sweat glands are partly atrophied. The hair follicles are surrounded by nucleated fibrous tissue, which in the later stages of the disease may contract, leading to compression and atrophy of the root sheath and loss of hair. There is a considerable increase of subcutaneous fat in many cases.

Nervous System.—No constant changes of importance have been found in any part of the nervous system. The hypophysis cerebri has been found to be enlarged in a few cases, the increase in size being due to hypertrophy of the anterior glandular portion.

Circulatory System.—In early cases no special changes are found in the heart or blood-vessels. In advanced cases interstitial myocarditis is found, and as a late result of this there may be fatty and fibrous degeneration of the cardiac muscle. Atheroma is commonly found in the larger arteries, and endarteritis in small vessels in all parts of the body.

Kidney.—Moderate cirrhosis of the kidney is frequently found, and with it there may be hypertrophy of the left ventricle of the heart.

Pathology.—*General Pathology.*—In considering the physiological functions of the thyroid gland we have already seen that myxœdema can be induced in lower animals by removal of the thyroid gland. In man we also know that primary myxœdema is always the result of destructive disease of the gland, and secondary myxœdema only occurs as the result of the removal of the gland by operation for goitre or malignant disease. We shall also see, in considering the treatment, that the symptoms of the disease can be entirely removed by maintaining a constant supply of the secretion in the form of thyroid extract. It is thus evident that the symptoms of the disease in man are solely and entirely due to destruction of the thyroid gland, and the consequent lack of an adequate supply of its secretion to the blood.

Chemical Pathology.—In the early stages of myxœdema there is probably an increase of the intercellular ground substance in the skin, which yields an excess of mucin. An excess of mucin was found by Dr. Charles, who examined the skin in Dr. Ord's first recorded case. In other cases, however, no excess has been found, probably, as Professor Halliburton suggests, because the patient died in the atrophic stage of the disease. The same observer has found an excess of mucin in the tendo-Achillis and in the cardiac tendons. The parotid gland in one case yielded 0·188 per cent of mucin, whereas in health it contains only a trace.

Diagnosis.—In a well-marked case of myxœdema diagnosis is easy, and can be readily made from the description of the appearance and symptoms already given. It is, however, important to bear in mind that in the early stages of the disease the appearance is much less characteristic, and the symptoms are much less obvious, so that slight cases may be easily overlooked. Languor, loss of energy, and loss of memory generally occur early, and in a middle-aged woman should always arouse a suspicion of myxœdema. At an early stage some swelling of the face, hands, legs, or feet can generally be detected when looked for, and the skin will be found to be drier than it should be. If any doubt exists as to the nature of the symptoms, a course of treatment with thyroid extract will soon settle the question. If there is myxœdema these symptoms will gradually disappear in the course of six or eight weeks' treatment; if not, no improvement will be observed.

From renal disease, myxœdema may be distinguished by the solidity of the swelling and by the absence of albuminuria, or at most the presence of only a trace of albumin in the urine, but it must be remembered that in some cases

of myxœdema there is chronic granular kidney as well.

In acromegaly there is an actual enlargement of the bony and other structures of the face and limbs, which gives quite a different aspect to the patient to the subcutaneous swelling of myxœdema.

PROGNOSIS.—Formerly the prognosis of myxœdema was a gloomy one, as the disease gradually progressed to a fatal termination. At most some temporary arrest or slight improvement during warm weather was to be expected. The duration was not uncommonly as much as five or ten years, and in a few cases twenty, or thirty. Now the prognosis is very favourable, as under treatment all the symptoms in any early case disappear and do not return as long as a sufficient amount of thyroid extract is taken. In more advanced cases also, if no incurable degeneration of an important organ has occurred, complete recovery takes place. Thus my first case, who began to be treated in 1891, more than eleven years ago, still takes one drachm of thyroid extract in the course of each week, and remains quite free from any symptoms of myxœdema.

The presence of well-marked cardiac degeneration, renal disease, or insanity naturally increases the gravity of the prognosis, though even in these cases great improvement may take place.

TREATMENT.—In the treatment of myxœdema the thyroid gland may be employed in several different forms. The raw, fresh gland of the sheep may be given finely minced and mixed with glycerine, the usual dose being from one-eighth to one-quarter of a lobe. As a rule, however, it is much better to employ one of the two official preparations, liquor thyroidei or thyroideum siccum. On the average, each lobe of the sheep's thyroid gland yields eight grains of dry thyroid powder, or 48 minims of thyroid extract. Thus, one-eighth of a lobe of the fresh gland = ♏ vj. of liquor thyroidei = gr. j. of thyroideum siccum. The liquor should be prescribed undiluted, the dose being measured out in minims, and water added at the time it is taken. It is advisable to obtain a fresh supply once a fortnight. Dry thyroid may be prescribed in either a powder, pill, or tablet.

The treatment of a case of myxœdema is conveniently divided into two stages. During the first our object is to get rid of all the symptoms, and so restore the patient to health; during the second stage we have to maintain the condition arrived at by the first. It must, of course, always be remembered that cessation of treatment will be followed by a return of the symptoms. During the first stage of the treatment of an advanced case of myxœdema it is advisable to keep the patient in the house, or even in bed if any symptoms of degeneration of the cardiac muscle are present, such as feeble or irregular pulse, dyspnœa on exertion, or weak heart sounds. In such cases only a small dose should be given at first, three minims of thyroid extract or half a grain of dry thyroid each night at bed-time being sufficient for the first week. If this dose is well borne, it may be gradually increased up to ten or twelve minims of the former or two grains of the latter preparation by the end of the second or third week. This dose may be continued steadily until all the symptoms of the disease have disappeared, when the first stage of the treatment is completed.

The second stage of necessity lasts as long as the patient lives. During this stage it is necessary to give an amount of thyroid extract each day equivalent to the daily output of secretion from a healthy thyroid gland. As a rule one drachm of liquor thyroidei each week is quite sufficient, ten minims being taken every week-day at bed-time. Thus, for example, my first case, who has been treated for more than eleven years, takes this amount regularly, and remains free from all symptoms of myxœdema. In some cases seven minims are quite sufficient for the permanent daily dose. I have not found it necessary to give more than ten minims in any case.

In slight cases it is not necessary to confine the patient to the house, and ten minims of the extract may be ordered from the commencement of the treatment. In these cases it is probable that a portion of the thyroid gland still retains its functional activity, so that when the first stage of the treatment is completed the permanent dose for the second stage may be rather smaller than in the advanced cases, where the gland is probably quite functionless. Five or seven minims of the extract each night have proved to be sufficient in some of my cases.

The most striking results of the treatment carried out in the manner just described are seen in advanced cases of myxœdema. In these, in the course of from two to six months the symptoms all disappear. The temperature gradually rises to normal, the subcutaneous swelling is gradually removed, and the face resumes its normal expression and appearance. If there has been alopecia the hair grows again. Full strength and energy are regained, and the limbs can once more be freely and actively moved. The mental hebetude is removed, the hallucinations disappear, and actual insanity has in some cases been cured. The total amount of urea in the urine is increased, the red corpuscles increase in number, and, if the menopause has not been reached, menstruation is re-established. In fact, if no incurable degenerative changes have occurred in the heart or other organ the patient is restored to health.

EXOPHTHALMIC GOITRE

HISTORY.—During the earlier part of last century several cases of what we now know to

be exophthalmic goitre were recorded without their true nature being recognised. Graves was the first to describe it as a separate disease in a clinical lecture published in 1835, and in recognition of this the name Graves' disease is often applied to it. Five years later von Basedow gave a more exact description of the symptoms, emphasising the association of the three main symptoms of exophthalmic goitre and palpitation, and in Germany the disease is often named after him. During the last sixty years several hundred papers have been written on the subject, so that the literature is now very considerable.

ETIOLOGY. — *Age.* — Exophthalmic goitre is rare in childhood, though it has been observed at the age of two and a half years. It occurs most frequently in adolescents and young adults, more cases developing during the third decade than in any other equal period of life.

Sex.—Women are far more liable to suffer from exophthalmic goitre than men, as there are at least eight times as many cases in women as in men.

Heredity.—In a few instances the disease has been observed in two and even three successive generations. Thus, in an example given by Rosenberg, the paternal grandmother, two paternal aunts, the father, and one sister of the patient all suffered from exophthalmic goitre. Less rarely two or three sisters in a family may show more or less marked symptoms of the disease. Three sisters once consulted me at the same time; one had well-marked symptoms, and the other two slight symptoms of Graves' disease. I was informed that the mother and a fourth sister both had goitre, but could not ascertain if other symptoms were present or not. It is not uncommon to find that there is a well-marked tendency among the relations of the patient to suffer from various neuroses, especially epilepsy, chorea, hysteria, and insanity.

Personal Antecedents.—Persons in all classes of life seem to suffer alike. Anæmia, quinsy, and rheumatism, in a considerable number of cases, precede the onset of the disease. In others some prolonged illness, exertion or mental strain appears to have predisposed the patient to the disease.

Exciting Cause.—Of the actual exciting cause of exophthalmic goitre in the great majority of cases we are as yet quite ignorant. In some cases, however, the symptoms have followed soon after some special event which appears to have acted as the exciting cause. This has generally been some severe fright associated with mental or physical shock, or else a prolonged mental strain or emotional disturbance. Thus one of my patients, at the age of twenty-four, fell off a bridge into the water and was nearly drowned; a goitre appeared a few months later, and when I saw her fourteen years afterwards it was still there, and though the eye symptoms had disappeared, the pulse was still 120, and there were tremors of the hands. In another of my cases, a young lady was engaged to be married when her *fiancé* developed phthisis, to which he succumbed a year later. Symptoms of exophthalmic goitre appeared soon afterwards, apparently as a result of this year of anxiety and suspense.

SYMPTOMS.—There are five leading or primary symptoms in exophthalmic goitre, with which may be associated other secondary symptoms.

The chief symptoms are enlargement of the thyroid gland, increased frequency of pulse, exophthalmos, general nervousness, and muscular tremors. Two or three of these symptoms are present in all cases, any one of which may be the first to attract the attention of the patients or the friends. In some cases the exophthalmos is first noticed, or the palpitation gives rise to alarm; in others the goitre is discovered first. In many, however, the nervousness, with irritability and restlessness, is the first obvious sign of ill-health. The several symptoms will now be considered in detail.

The Thyroid Gland.—Some enlargement of the thyroid gland is present in the great majority of cases. The swelling in the neck may be the first sign of the disease, or it may develop at the same time as the exophthalmos and palpitation, while in some cases it is not noticed until after the appearance of these symptoms. As a rule the enlargement takes place gradually during several weeks, but it has been known to come on in a few hours. The amount of enlargement is usually moderate, but readily visible to the eye; in some cases it can, however, only be detected on palpation. The whole gland is uniformly enlarged, and as the right lobe is normally larger than the left, this difference becomes more marked when the gland is hypertrophied. During the earlier stages of the disease the gland gradually enlarges, then for a variable period it remains stationary, or may show slight variations in size from week to week or even from day to day.

In many cases the enlarged gland is seen to pulsate; this movement is often communicated by the violent throbbing of the carotid arteries, though in some cases there is a genuine expansile pulsation of the whole gland. A systolic thrill can be felt in some cases on light palpation, and a systolic murmur can frequently be heard when the stethoscope is applied to the front of the neck.

In the later stages of the disease fibrosis of the enlarged gland may develop, so that it gradually becomes smaller and harder. This is really a beneficial change, and may be accompanied by subsidence of the other symptoms. In some cases, however, this process has gone on to advanced fibrosis with atrophy of the gland, and the symptoms of myxœdema have in consequence replaced those of exophthalmic goitre.

Ocular Symptoms.—Several different ocular symptoms occur either singly or in association, so that it will be convenient to consider them all together, though some might equally well be described along with the nervous symptoms.

Exophthalmos.—This when present is naturally the most conspicuous symptom of all, and consequently is all the more distressing to the patient. It generally comes on gradually, and is present in some 80 per cent of cases. The amount of exophthalmos varies considerably from a slight staring expression to so great prominence of the eyeballs that the sclerotic is clearly visible both above and below the margin of the cornea, and the eyelids cannot be closed. Sometimes the exophthalmos is more marked on one side than the other, or it may in rare cases be unilateral. In about one-fifth of the cases there is no exophthalmos. Even in the same case the prominence of the eyeball may vary in degree, from time to time, along with the other symptoms. Conjunctivitis is frequently present.

Von Graefe's Symptom.—When this is present there is defective descent of the upper eyelids when the patient looks downwards. Thus when the eyes are kept fixed on the finger during a rapid downward movement the upper lid lags behind, so that the downward rotation of the eyeballs brings the sclerotic within view between the upper lid and the margin of the cornea.

Stellwag's Sign.—In some cases there is retraction of the upper eyelid, due to increased tonic contraction of the elevator of the eyelid. This increases the width of the palpebral fissure already widened by the exophthalmos; it may, however, occur when the latter symptom is absent.

In addition to these symptoms there is sometimes weakness of convergence, so that when the eyes are fixed on an object which is gradually brought closer to them, they at first converge naturally until a point is reached at which they suddenly become parallel again, only one eye remaining fixed on the object. The actual distance at which this occurs varies in different cases, and in the same case at different times.

A general weakness of the external muscles of the eyeball is not uncommon, and there may be diplopia on looking upwards, or downwards, or to the extreme right or left. Sometimes there is actual paralysis of one or more of the muscles. Complete ophthalmoplegia externa with ptosis is rare, though I have one case in a man under observation in which, although the other symptoms of the disease have to a large extent subsided, there is almost complete immobility of the eyeballs with double ptosis.

Vision, as a rule, is unaffected, and the reactions of the pupil are normal.

Circulatory System. — An increase in the frequency of the contractions of the heart is a constant symptom of exophthalmic goitre; in fact the condition of the circulatory system closely resembles that of a man who has just taken violent exercise when out of training, or who is experiencing some powerful emotion.

This increased frequency of the pulse may come on gradually, or may at first occur only for short periods at a time, becoming constant at a later stage. Palpitation is often complained of early, and may be the first symptom noticed by the patient. The pulse-rate may vary from 90 to 100 in mild cases, though in cases of moderate severity it is usually 120. In more severe cases the pulse may be 140 or even 160. The heart is very irritable, so that with slight exertion or excitement the pulse-rate may be increased by 20 or 30 beats a minute. In some cases, even when lying in bed, there are occasional paroxysmal attacks of violent palpitation which come without any apparent reason, during which the skin is generally flushed and perspires freely. During one of these attacks I have counted as many as 200 heart-beats in a minute. The radial pulse is usually regular, but rather small and compressible.

There is generally strong pulsation in the carotid arteries, often in the abdominal aorta, and even in the retinal arteries as well.

The area and force of the cardiac impulse are both increased, and the apex beat, especially in cases of some duration, is frequently in the left nipple line or even outside it. Both heart sounds are loud, and there may be some blurring of the first.

In many cases a soft systolic murmur is audible at the apex, especially when there is dilatation of the left ventricle with secondary mitral incompetence. A soft systolic murmur is also often to be heard at the base. In several of my cases there has been a peculiarly rough, loud systolic murmur over the pulmonary area, so rough in one or two of them as to resemble pericardial friction rather than an endocardial murmur.

Nervous System.—Various nervous symptoms may occur in exophthalmic goitre, general nervousness and tremor being the most common.

General Nervousness and Mental Condition.—In nearly all cases there is a peculiar condition of nervousness. In severe cases this is shown by great restlessness, the patient fidgets about when sitting on a chair, the attitude and the position of the limbs being changed every few seconds. In other less severe cases the restlessness may be less marked, but the patient is agitated by trifling events, such as seeing a stranger, the tremor being exaggerated and the pulse-rate increased. Such patients complain that they are unduly worried by domestic cares, and are apt to be irritable upon very slight provocation with those with whom they live. Not infrequently much time and care is expended upon some trifling object, while there

is a lack of power of continuous application to any regular occupation. Though cheerful as a rule, they are liable to fits of depression with crying. In some cases these symptoms become greatly exaggerated and result in actual insanity, which usually takes the form of mania or melancholia.

Tremor. — Tremor is a common symptom which is nearly always present at one time or another during the course of exophthalmic goitre. It is most easily observed when the patient extends the arms and with the palms directed downwards and the fingers spread out. It varies greatly in degree in different cases, and in the same case at different times. When very slight, it may be felt more easily than seen by laying the fingers lightly on the back of the patient's extended hand. This symptom sometimes comes on early, but as a rule is not noticed until after the other symptoms already described have attracted attention. The whole limb is affected, but especially the flexors and extensors of the wrists, so that the tremor affects the hand as a whole, and not the fingers separately. The tremor also may affect the legs, and even the head and neck. The rhythm is rapid and regular, each complete movement occupying about one-eighth of a second. The range of movement is small, but variable. Fine voluntary movements, such as writing, may be impeded, but not prevented by the tremor in some cases. Any extended or unusual effort increases the tremor, which is least marked when the patient is lying down at rest.

Other nervous symptoms in addition to those already described have been observed. A sudden giving way of the legs, so that the patient falls to the ground, occurs in some cases. Painful nocturnal cramps of the feet and legs, and even of the hands, have been described by Dr. Hector Mackenzie. Inability to walk, while the legs can be moved when the patient lies down (astasia-abasia), has been recorded by Dr. Maude.

The Skin.—Pigmentation. — In many cases there is more or less pigmentation of the skin. This is most marked in those situations in which the skin naturally contains most pigment, and may therefore be similar in distribution to the pigmentation in the earlier stages of Addison's disease. The eyelids and the neck are common situations, as are also the axillæ, the areolæ of the nipples, and the flexor surface of the joints. The face may be uniformly coloured, or there may be brown patches on the cheeks. Pressure, such as that caused by the garter, may also determine a localised increase of pigmentation. In one of my patients all the skin of the face, neck, hands, and fore-arms was pigmented, so that she looked as if she had been working in the sun with her sleeves rolled up to the elbow. The colour of the skin may vary from a yellowish tint to brown, but does not reach the dark brown which is seen in an advanced case of Addison's disease. The mucous membrane of the mouth is not pigmented.

Perspiration.—The skin generally feels warm and moist, and the skin of the face may look rather greasy. In addition to this, profuse local or general sweats and hot flushes are not uncommon, especially in the more acute cases. They may last for an hour or so, and are sometimes repeated two or three times in the day. Profuse general perspiration often accompanies the paroxysmal attacks of palpitation already mentioned. Patients often complain of feeling hot, and seem to feel cold very little. As a result, they not infrequently go about in quite thin clothing in cold weather. They are not liable to catch cold; in fact, in some of my cases this liability appears to have diminished with the onset of exophthalmic goitre. One of my patients, who formerly suffered a good deal from chilblains, told me that they had not returned since the symptoms of exophthalmic goitre developed.

In a considerable number of cases the electrical resistance of the skin has been found to be diminished. This is probably a direct result of the moist condition of the skin, which naturally acts as a better conductor when moist than when dry, and is not peculiar to Graves' disease. Temporary œdematous swellings of the subcutaneous tissues sometimes appear in various parts of the body. Regular œdema of the lower extremities may occur, but it is to be regarded as a complication secondary to anæmia or cardiac failure.

Hair, Nails, Teeth.—Loss of hair frequently occurs in all parts of the body. The head may become completely bald, as in one of my cases in which the eyebrows and eyelashes had completely, and the pubic hair had partly, disappeared, and did not return while she was under observation. In another case the hair was also lost during the earlier part of the illness, but grew again later on when the other symptoms improved.

The nails in some cases are thin and brittle, or they may be deeply grooved. Caries of the teeth is not uncommon.

Respiratory System.—The frequency of respiration is increased in a good many cases. In some it is the result of anæmia or of cardiac weakness. It may, however, occur quite independently of either of these causes. The respirations may be as frequent as 30 or 38 in a minute. In one case which I saw with Dr. Messer of Lemington this symptom was the earliest to attract attention, and as it followed an attack of influenza it suggested the onset of some pulmonary complication such as pneumonia. The lungs, however, remained quite healthy, and other symptoms of exophthalmic goitre soon followed. Paroxysmal attacks of coughing occur in some cases, as do also attacks

of dyspnœa with cyanosis which may even prove fatal. Profuse bronchorrhœa has been observed in a few cases, and is considered by Bramwell to be analogous to the sweating and diarrhœa to which some patients are liable.

Digestive System.—In a considerable number of cases there is an increased frequency of defæcation. This must be distinguished from diarrhœa, as the motions are solid and normal in character. In these cases the bowels may act two or three times a day. No harm apparently results beyond the inconvenience which it causes. Sudden attacks of diarrhœa are not at all uncommon. As a rule there is no pain, and the motions are loose and copious. Two or three may be passed in a day, and then the attack ceases, or it may continue for two or three days and then terminate as suddenly as it began. Vomiting may be a troublesome symptom, and sometimes is difficult to control. It developed in the case already mentioned with early rapid respiration, and continued until the patient died from exhaustion. Dr. D. Drummond recently drew my attention to a great craving for food which he had observed in some cases, and since then I have seen several cases in which there was a marked increase in the appetite.

Genito-Urinary System.—The urine is normal in the majority of cases; in some, however, the quantity is increased. Intermittent albuminuria is present in some cases, and glycosuria has also frequently been observed. Menstruation is often irregular. Periods of amenorrhœa are not uncommon, while in some cases there is menorrhagia. Pregnancy may occur, and may be accompanied by improvement in the symptoms, though hæmorrhage after delivery may be excessive.

General Symptoms.—Emaciation is present in nearly all well-marked cases of Graves' disease, and is frequently in proportion to the severity of the other symptoms. The actual loss of weight is very considerable in some cases, and may amount to as much as forty pounds in a year, or even to ninety-three pounds in ten months, as in two cases mentioned by Mannheim. Varying degrees of anæmia may often be observed in exophthalmic goitre, though by no means constantly. A slight rise of temperature is not uncommon, and may be persistent, or of only temporary duration, recurring from time to time.

VARIETIES.—All the symptoms of the disease may vary greatly in intensity in different cases. In one exophthalmos may be the most striking symptom, in another the frequency of the pulse, and in another the goitre or the tremors and general nervousness, while the other symptoms are only slightly developed.

A well-marked case is easily recognised, but where one or more of the primary symptoms are slight or absent altogether, the real nature of the case may be overlooked. In the incomplete cases there may be no exophthalmos. In some of these, however, there is a slight widening of the palpebral fissure which gives rise to a startled expression. An increase in the pulse-rate is present in all cases. The presence of this symptom alone in a patient without any obvious explanation should always lead to a careful search for other symptoms of Graves' disease, for many cases of so-called "tachycardia" are really incomplete cases of exophthalmic goitre.

The enlargement of the thyroid in some cases is only slight, but in nearly all cases there is some enlargement at the time of the examination, or a clear history of it having been present at an earlier stage.

COURSE, DURATION, AND TERMINATION.—The course of exophthalmic goitre varies very considerably in different cases. There is usually a period of development during which the symptoms appear and gradually increase in severity. This period may vary from a few weeks to several months or a year. After a time the symptoms remain more or less stationary in many cases, with slight temporary variations. This stage may last only a few weeks, or may continue for many months or for several years. After this, in a favourable case, the symptoms gradually decrease in severity. In some they finally disappear altogether, while in others they become so slight that the patient feels practically well again. In these cases, however, a careful examination may still reveal a somewhat frequent pulse, slight enlargement of the thyroid gland, a faint tremor or some general nervousness of which the patient herself is fortunately unaware.

Sometimes the onset is acute, and this may be followed by an acute course and early recovery within two or three months. On the other hand, these acute cases may run a rapid downward course to a fatal termination within a similar period of time.

In the majority of cases the symptoms run a more chronic course, and may persist for years. Four or five years is a not uncommon period for the symptoms to last. In two cases which I saw twelve and thirteen years after the onset, the symptoms were still well marked. In another case, twenty years after the commencement, the pulse was 92, the thyroid gland was normal in size, and there was very slight exophthalmos with slight retraction of the upper eyelids. The patient considered that she had quite recovered from the disease, as she was not conscious of these slight traces of her former condition.

Recovery from exophthalmic goitre may be followed by the development of myxœdema. In these cases the enlarged gland is gradually reduced in size by a progressive fibrosis which is beneficial up to a certain point, and is accompanied by a subsidence of the symptoms of

Graves' disease. The fibrosis unfortunately progresses too far, so that the gland is no longer able to supply a sufficient amount of its secretion owing to the atrophy of the glandular epithelium, in consequence of which myxœdema develops.

I have only seen one example of this sequence of events in an interesting case under the care of Dr. Scott Jackson of Alnwick. The symptoms of myxœdema may begin to appear before all those of exophthalmic goitre have quite disappeared, or there may be an interval of good health between them.

In those cases in which an apparent recovery has taken place, as already described, a recrudescence of the symptoms is not uncommon. In one such case I attributed the return of the symptoms to excessive bicycling.

Death may occur from some complication, to which the patient, already exhausted by Graves' disease, can offer but feeble resistance, or from the severity of the disease itself. Cardiac failure is the actual mode of death in some cases, which may thus terminate suddenly and somewhat unexpectedly in a fatal attack of syncope. In other cases all the symptoms become acutely increased in severity, and the patient dies exhausted. Persistent vomiting or diarrhœa has contributed largely to the fatal issue in some cases. Patients with exophthalmic goitre bear operations badly. Several have died after removal of one lobe of the enlarged thyroid gland. Under these circumstances death may occur suddenly within an hour or two of the operation. One of my cases died half an hour after the operation. It is difficult to determine whether the shock of the operation itself or the anæsthetic is the chief source of danger. In some cases death occurs within a few days of the operation, from an acute exacerbation of all the symptoms and consequent exhaustion. This appears to be the result of the absorption of a large amount of thyroidal secretion, which has either escaped into the wound from the cut surface of the gland, or has been driven out of the gland into the blood-stream by pressure exerted on it during the operation, as the symptoms are the same as those of severe thyroidism produced by large doses of thyroid extract.

In operations on other organs also there is a considerable risk of death, as in a case recorded by Mr. J. D. Harris of Exeter, in which a woman aged forty-six, with exophthalmic goitre, died from cardiac failure sixty-eight hours after removal of the left breast under ether.

Diagnosis.—In a well-marked case the presence of the primary symptoms of exophthalmos, enlarged thyroid gland, and frequent pulse with probably tremors, general nervousness and emaciation, at once suggests the diagnosis. In some of the ill-developed varieties which have already been described the diagnosis is more difficult. An important feature, however, is the constant presence of a quick pulse, the presence of which without some obvious cause, such as fever or cardiac disease, should always suggest the possibility of the presence of Graves' disease, and lead to a careful search for other symptoms. In these cases the pulse is always frequent, and thus differs from that of a patient with paroxysmal attacks of tachycardia, whose pulse-rate falls to the normal between the attacks.

In a few cases there may be no obvious enlargement of the thyroid gland; in some of these cases, however, a careful examination reveals the fact that the gland is larger than in health, or there is a history of a previous enlargement which has subsided.

In the absence of exophthalmos and other earlier symptoms the diagnosis is indicated by the cardiac and nervous symptoms. Slight cases with an enlarged thyroid gland can be distinguished from cases of moderate parenchymatous goitre by the fact that the latter are not accompanied by any nervous or cardiac symptoms. It is important, however, to remember that exophthalmic goitre may develop in a patient who has previously had a simple goitre without general symptoms; in such cases the history shows that the goitre has preceded the onset of the other symptoms by a long period of months or even years.

Prognosis.—At the beginning of exophthalmic goitre prognosis is a difficult matter. It is more favourable in women than in men. The circumstances of the patient must also be taken into account, as the prognosis is certainly better in those who can lead a quiet, easy life and have proper attention, than in those who are obliged to work when not really fit to do so, and are unable to afford the many accessories which can make the illness so much more tolerable. In such a prolonged disease the patient is often lost sight of, so that it is difficult to obtain complete records of a series of cases.

Dr. W. M. Ord and Dr. Hector Mackenzie have tabulated 57 cases, including 24 observed by Dr. R. T. Williamson, in which the disease had lasted more than five years or ended fatally. Of these, 14 died, 10 recovered completely, and 11 almost completely. In 13 there was considerable, and in 4 slight improvement; 4 remained unaltered, and the remaining patient was alive, but the condition was not known.

Taking slight and severe cases together, from one-third to one-half may be expected either to recover completely or to such an extent that the remaining symptoms are scarcely noticed. About one-quarter of the cases succumb to the disease.

The onset of œdema, dyspnœa, and other signs of cardiac failure indicate that the patient is in danger, and if they continue to increase in spite of complete rest and treatment the prognosis is very grave.

Repeated vomiting and severe diarrhœa are also serious symptoms, for if they continue, the patient soon dies from exhaustion.

Pathological Anatomy.—There is general emaciation, and but little subcutaneous fat remains. The small amount of fat in the body is in striking contrast to the excess of fat which is often found in the orbit, and which is most probably the cause of the exophthalmos.

Thyroid Gland.—The thyroid gland is uniformly enlarged except in cases of long standing, in which there may be some irregularity in outline owing to the development of fibrosis in some parts of it. The superficial veins are large, and in well-developed cases the arteries are dilated and tortuous. The number of alveoli is increased owing to the enlargement of the gland, and in many of the alveoli the secretory surface is increased by folding of the wall, so that the lumen is diminished and has an irregular outline. The epithelial cells themselves are large and columnar in shape, instead of being flat or cubical as in the normal gland. The secretion seen in the alveoli is smaller in amount, is less viscid, and stains less deeply than in health.

In cases of long duration there may be more or less fibrosis.

Thymus Gland.—The thymus is generally persistent and may be increased in size.

Nervous System.—Various changes have been described in various parts of the nervous system, but none of them are constant or even frequent. Hæmorrhages have been described in the medulla oblongata by Hale White, by Greenfield, and by Grainger Stewart and Gibson. On the other hand, many observers have found no change of any kind in the medulla, and Mobius considers that, as a rule, no lesion is found in this situation.

No changes of importance have been found in either the brain or spinal cord. Some observers have described changes, such as leucocytic infiltration and degeneration of nerve-cells, in the cervical sympathetic ganglia. Similar changes have, however, been found both in healthy subjects and in other diseases, and so have no pathological importance.

The heart may be unaltered. In some cases there may be some dilatation and hypertrophy of the ventricular walls. Valvular lesions are only occasionally found.

Pathology.—The pathology of exophthalmic goitre is of much interest, and is still a matter for discussion We shall, however, endeavour to indicate what seems the most probable explanation of the phenomena of the disease in the present state of our knowledge.

We have seen that there is no constant lesion of the nervous system to be found, so that we must look elsewhere for a primary lesion. The most obviously and constantly affected organ is the thyroid gland, and this we believe to be the essential lesion of the disease. Shortly stated, we believe the symptoms to be due to the excessive formation by the thyroid gland of secretion, which may or may not be normal in character, which being carried into the general blood-stream, so acts upon the nervous and circulatory systems as to produce the familiar symptoms of the disease.

In the thyroid gland the structure suggests a great increase in secretory activity, for not only is the gland enlarged as a whole, but the plication of the alveolar wall gives an increased secretory surface, while the individual epithelial cells are also increased in size.

It is evident that these structural changes indicate increased activity, for in the monkey when one lobe of the gland is removed the other undergoes compensating hypertrophy, and similar folding of the alveolar wall associated with increased size of the epithelial cells is found.

In man, large doses of thyroid extract produce a condition known as thyroidism, in which the symptoms are remarkably like those of exophthalmic goitre. The earliest symptom is acceleration of the pulse. The pulse-rate may be increased to 100, 120, or even 130, and the patient often complains of palpitation. There may be slight tremor of the hands, and the skin becomes moist and flushed. If the large doses are continued, emaciation takes place in some cases. In exceptional cases, when such large quantities have been taken as to cause toxic symptoms, fever, diarrhœa, insomnia, restlessness, polyuria, glycosuria, albuminuria, rapid respiration, and even exophthalmos have been observed.

We have already seen that myxœdema is due to loss of thyroidal secretion, so that exophthalmic goitre being due to the opposite condition of excess of secretion, presents also a contrast in symptoms. In the former there is atrophy of the thyroid gland, with increase of weight, dry skin, absence of perspiration, subnormal temperature, placid temper, and slow pulse; in the latter, hypertrophy of the thyroid gland, loss of weight, moist skin, excessive perspiration, normal or raised temperature, nervousness, excitability, and quick pulse.

A further point of interest is that exophthalmic goitre may subside and be followed at once or after an interval by myxœdema. The progressive fibrosis of the thyroid gland leads to removal of the symptoms of exophthalmic goitre in the first place, but in its later stages causes thyroidal atrophy and myxœdema. No case has been recorded in which exophthalmic goitre has developed after myxœdema.

These facts all strongly support the theory we have stated above, as the most reasonable explanation of the pathology of the disease.

Treatment.—In exophthalmic goitre, whatever the medicinal treatment may be, the mode of life which the patient is able to adopt is a matter of great importance. We may, therefore,

in the first place, consider some general rules of living, which should, as far as possible, be followed in all active cases of Graves' disease. We will consider what is most suitable for a well-developed active case, with moderate goitre, distinct exophthalmos, nervousness, tremors, and a pulse-rate of 120 or more. At the commencement of treatment, in some cases it is a good plan to confine the patient to bed altogether for a fortnight or three weeks; in milder cases this is not necessary. Throughout any course of treatment, at least twelve out of the twenty-four hours should be spent in bed. It is advisable, therefore, for the patient to be in bed by 10 P.M., to breakfast in bed at 9 A.M., and to rise not earlier than 10 A.M. As many of the remaining twelve hours of the day as possible should be spent in the open air. During the summer months in this country eight to ten hours may be spent with advantage out of doors. Even in cold weather outdoor life is well borne, as cases of exophthalmic goitre do not feel the cold and rarely catch cold. Most of the time passed out of doors should be spent lying down on a couch or deck-chair in an open shelter or verandah, much in the same manner as in the outdoor treatment of phthisis. A short walk of a quarter of a mile each morning and afternoon may be taken at first, and the distance gradually increased, as improvement takes place, to a mile or more twice a day. Drives in an open carriage in the country may also be taken several times a week, but the patient should not drive herself. Many of those who suffer from exophthalmic goitre have previously led very active lives, and may at first find this inactivity trying, but it is remarkable how soon they appreciate its advantages. Cases of exophthalmic goitre should not be allowed to walk in a town, or attend public meetings or gatherings of any kind, as such always have a harmful effect upon them. They may, however, with advantage receive a few friends at home, but should always be relieved from any social duties or pleasures which prove in any way irksome.

Reading may be allowed, but the print must not be too small. Music also may help to relieve the monotony of the treatment.

As the course of exophthalmic goitre is often slow and protracted, this quiet life must in some cases be continued for two or three years, or even longer. As a rule, however, marked improvement takes place in the course of one or two years, so that the stringency of the rules laid down may be gradually relaxed. The early months of the year may with advantage be spent on the south coast of England; Hastings, Bournemouth, and the Isle of Wight are suitable places. The health resorts of southern Europe entail a long journey, which is not advisable, at any rate for cases in which the symptoms are active. During the summer and autumn some quiet place in the country or at the seaside should be selected.

Ordinary diet, as a rule, is all that is necessary, but it may with advantage be supplemented by one glass of milk taken at 11 A.M. and another at bed-time.

Local treatment of the goitre should always be carried out and continued for a long period. A piece of the red iodide of mercury ointment about the size of a pea should be rubbed into the skin over the goitre each night till the skin is reddened, and afterwards every other night. In some cases it is better to use the ointment of half the usual strength. On the whole, the most useful drugs are belladonna, convallaria, arsenic, the bromides, and iron if there is anæmia. Tincture of belladonna should be given in doses sufficiently large to produce slight dryness of the mouth and throat; in some of my cases, however, it has proved so disagreeable to the patient that it had to be discontinued. The digitalis group of drugs have but little effect in diminishing the pulse-rate; the most useful member is tincture of convallaria in doses of 5 to 10 minims in cases in which the palpitation is troublesome and the pulse very frequent. When the general nervousness and tremor are excessive the bromides may be given with advantage. In many cases a prolonged course of arsenic in small doses, such as 5 minims of liquor arsenicalis, twice or thrice daily after meals, has greatly improved the condition of the patient. The arsenic may be combined with any of the drugs mentioned, according to the nature of the symptoms present. The thymus gland has been used in many cases; it may be given in five-grain tablets, three to six of which may be taken daily. Opinions differ as to its utility. Dr. Hector Mackenzie has seen no good effects in twenty carefully observed cases. Favourable results have, however, been observed by others; and in one of my cases, who took three dried thymus tablets each day for nine months, the pulse-rate, which varied from 132 to 143, gradually fell to 84, the goitre disappeared entirely, the tremor became much less, and the exophthalmos was diminished. The further course of the case has not been followed, as she ceased coming to the hospital. In other cases improvement has also been observed, but not to such a marked extent.

Sodium phosphate, which may be given in doses of twenty to sixty grains two or three times a day, has been found useful by Trachewsky and by Kocher. The glycerophosphate is preferred by some, and may be given in five or ten grain doses or as the compound syrup.

Certain acute symptoms at times may develop and require special treatment. Severe attacks of palpitation, in which the heart beats 150 to 200 times a minute, should be met by complete rest in bed and the application of an ice-bag to the præcordial area. Persistent vomiting is a serious and in some cases fatal complication; it should be treated by rest in bed and rectal

feeding. Morphine is the most useful drug, and may be given by the mouth, or better by hypodermic injection. A quarter of a grain or more of morphine tartrate may be given once or twice a day with or without one-hundredth of a grain of atropine sulphate. In one of my recent cases each injection controlled the vomiting for some hours, but the patient was very nervous, and dreaded the injections so that a morphine suppository was used instead. Dr. Dreschfeld has found large doses of potassium citrate useful, but they had no result in the case just mentioned. Diarrhœa is most easily controlled by dilute sulphuric acid and laudanum.

In cases in which the symptoms are acute, and especially if rapid wasting has taken place, a course of Weir Mitchell treatment, in which isolation, rest, and over-feeding are combined with massage and electricity, proves most beneficial.

Electricity is useful in many cases. The faradic current should be employed in the following manner:—Two electrodes about 4 ins. by 2 ins. are applied to the neck, one behind and the other over the thyroid gland in front, and kept in place by means of a strap on each side. The electrodes are connected with a dry-cell faradic battery, and enough current is turned on to produce a distinct prickling sensation in the skin. The current may be applied for half an hour or an hour night and morning.

The true place of surgical treatment for exophthalmic goitre is difficult to determine. We have seen that the symptoms are due to excessive secretion by the thyroid gland, so that removal of one lobe of the gland is quite a rational treatment. A considerable number of cases who have survived the operation have been greatly benefited by it. Unfortunately there is considerable risk in the operation itself. Some patients die just after the operation, either from shock or the effect of the anæsthetic, and in others severe toxic symptoms have developed soon afterwards from absorption of an excessive amount of secretion, which has either escaped into the wound from the cut surface of the gland, or has been squeezed out of the gland during the operation. At present there are but few cases in which an operation is advisable, and even then it should only be undertaken after explaining to the patient the risks which must be incurred, and at a time when no acute symptoms are present.

[On the theory that the symptoms of Graves' disease are due to over-activity of the thyroid a number of attempts have been made to prepare a curative antithyroid serum. Möbius's serum, obtained from sheep six weeks after extirpation of the thyroid, has received a fairly extensive trial in Germany, and seems to have acted beneficially in some cases. Rodagen, made from the milk of thyroidectomised goats, has also been used, and there are now on the market several analogous "anti-thyroid" preparations. Of these it may be said that they can at least do no harm, and that they may possibly be beneficial in some cases.]

Acute Thyroiditis

Causation.—Acute inflammation of the thyroid gland is rare, and may occur as a primary disease or as the result of injury or secondary infection. Secondary thyroiditis may occur as a complication in rheumatic fever, influenza, enteric fever, and other acute infective diseases.

Symptoms.—The onset of the disease may be rapid and the attack may be ushered in by a rigor. The temperature rapidly rises to 102° or 103° F. At the same time a sensation of fulness or even actual pain may be felt in the front of the neck. The whole gland, or it may be one lobe only, then becomes swollen. In a few severe cases the swelling is large enough to press on the trachea and œsophagus, and cause difficulty in breathing and in swallowing. The inflammation may subside in a few days, and be followed by complete resolution. In a few cases, however, suppuration or gangrene of the gland occurs instead.

Treatment.—Rest in bed and a milk diet should be ordered while there is fever. The application of ice or leeches to the front of the neck or the use of belladonna fomentations gives relief to the pain. At the commencement of the attack five grains of calomel or blue pill may be given at night, followed by a saline aperient in the morning. Fifteen or twenty grains of sodium salicylate may be given three times a day. If suppuration occurs the abscess should be incised and drained.

Goitre

Goitre is a term in general use for all enlargements of the thyroid gland. As the enlargement may be due to several different causes there are several varieties of goitre.

Varieties.—A slight temporary enlargement of the gland occurs in anæmic and in pregnant women, but it is not sufficiently great to be considered as a form of goitre. A general enlargement of the gland occurs in young adults, and may attain a considerable size. This is practically a hypertrophy of the gland, and is known as simple parenchymatous goitre. The endemic form of goitre may commence in a similar manner, but later secondary changes, such as multiple cystic degeneration and fibrosis, may occur, so that very large goitres of this class are seen, composed of varying proportions of glandular tissue, fibrous tissue and cysts, fibro-cystic goitre. Adenoma of the thyroid gland is another common form of goitre. These growths are prone to become cystic, and as a rule a single-cysted goitre is due to the presence of a cystic adenoma in one lobe of the gland. Exophthalmic goitre has already been described,

and malignant goitre will be considered separately.

Etiology.—Goitre occurs much more frequently in women in this country than in men. It may occur at any time of life, but most frequently is first noticed about puberty. Goitre is hereditary in some families, though in some cases this may be due to several generations living under similar conditions. One of my cases, a young married woman, who was sent to me by Mr. A Thompson of Darlington, with a large adenoma of the thyroid, had a twin sister who also had a goitre. In certain districts, which are for the most part mountainous with deep valleys, goitre is endemic. Such districts occur in the Pennine range in England, and especially in the mountainous parts of Switzerland and the adjoining countries. The disease is also endemic in some flat countries, as on the plains of Lombardy and in the Indian Punjab. Of the many local circumstances which have been suspected of causing endemic goitre the drinking-water has proved to be the most important. The disease has developed after the introduction of a fresh water-supply, where it was not endemic before, and conversely, in a previously goitrous district change of water-supply has been followed by a steady diminution in the number of cases. What the active constituent of the water may be is not yet known, but the fact that such water is rendered harmless by boiling indicates that it is possibly a living micro-organism.

Symptoms.—When a goitre is small, a swelling in the thyroidal region, which moves upwards when the patient swallows, and a sense of fulness in the neck, may be the only signs of its presence. The character of the swelling varies according as the goitre is parenchymatous, adenomatous, cystic, or fibro-cystic. In parenchymatous goitre it is uniform in shape and consistence with the normal gland. An adenoma forms a firm rounded mass, and a cyst a smooth globular swelling in which fluctuation may be felt. In some cases, however, it is difficult to determine by palpation whether an adenoma is cystic or not. Dyspnœa is not uncommon, and is generally due to narrowing of the trachea by unilateral or bilateral pressure exerted upon it by the goitre. In other cases pressure may be caused by an extension of the goitre behind the trachea or in front between it and the sternum. Dyspnœa may, but rarely, occur as the result of pressure on the recurrent laryngeal nerves, causing paralysis of the abductors of the vocal cords. In cases of large goitre there may be sudden paroxysmal attacks of dyspnœa, especially at night. Dysphagia may occur, but it is rare. A goitre may undergo rapid enlargement as the result of hæmorrhage or of acute inflammation.

Medical Treatment.—In the early stages of endemic goitre, removal from the goitrous district, or the boiling of all drinking-water, may be followed by the disappearance of the goitre. In simple parenchymatous goitre, especially in young adults, the best results are obtained by giving thyroid extract. In some cases the goitre disappears entirely, in others it is reduced to one-half or a third of its former size, while in some cases little or no effect is produced. From one to three grains of dry thyroid powder should be given twice or thrice daily. This dose may be continued for six or eight weeks. If the goitre is then diminishing in size the treatment may be continued for another two or three months. If there is no decrease in size at the end of the first two months further continuance of the treatment is useless. This treatment may be accompanied by the inunction of red iodide of mercury ointment. Potassium iodide and iodoform have also been successfully used in the medical treatment of goitre.

Malignant Disease. — Both carcinoma and sarcoma of the thyroid gland are rare, but clinically it is often difficult to distinguish between them. Carcinoma may originate either from the alveolar epithelium of the gland or from the parathyroid gland. A sarcoma may be either round or spindle-celled.

Malignant disease may develop in a gland which has previously been healthy or in one already goitrous. In carcinoma there is generally rapid enlargement and early fixation of the goitre. The swelling is hard and irregular in shape. There may be radiating pain over the front of the neck. Paralysis of one or both vocal cords is strong, but not conclusive evidence of malignant disease. The trachea, œsophagus, and blood-vessels may be first compressed and later invaded by the growth. The lymphatic glands are only infected after the growth has extended beyond the capsule of the gland. Sarcoma is generally more uniform and softer in consistence than carcinoma and may be cystic.

The only treatment which offers any prospect of success is total removal of the whole thyroid gland as early as possible.

Thyroid Gland, Surgery of.

Introduction 19
Treatment of Goitre 21
Adenomatous Tumours 23
Exophthalmic Goitre 24
Malignant Disease 24
Tracheotomy in Thyroid Disease . 24

Until a comparatively quite recent period, operations upon the thyroid gland were looked upon with great suspicion, and were only resorted to when urgent symptoms imperatively called for surgical interference, and as a matter of necessity. This hesitation to interfere surgically with the thyroid gland was due to the

fact that death arose so frequently, either immediately from hæmorrhage during the operation, or later on from some form of blood poisoning. Now, however, that both these causes of death have been well-nigh eliminated from operative surgery, the one by the invention of pressure forceps, the other by aseptic surgery and the technique of operations on the thyroid gland brought to great perfection, the thyroid is operated upon very successfully, not only in cases of necessity, but also as a matter of expediency. No operation upon the thyroid, however apparently trifling it may seem to be, should ever, even now, be undertaken without special care, and every precaution being taken to bring it to a successful issue. Apart from the risk of hæmorrhage and sepsis, neither of which contingencies are altogether absent, there are other special dangers both during and after operations upon the thyroid gland which cannot be ignored. The trachea, pharynx, œsophagus, pleura, and the recurrent laryngeal nerve have all been injured during the operation. Death may arise during the operation from the entrance of air into veins, from suffocation, or from syncope. The first accident should not occur, and if every vein be securely tied with double ligatures before it is divided, and great care be taken not to injure the internal jugular vein, there is no danger to be apprehended from the entrance of air into veins. With every possible precaution death may arise during operation from suffocation or from syncope. Myxœdema or tetany has arisen, as a consequence, when the whole of the gland has been removed; and even after tapping a cyst or the enucleation of an adenoma, recurrent hæmorrhage has proved fatal or placed the patient's life in danger. The commonest disease to which the thyroid gland is liable is goitre, that is to say, "an enlargement not due to inflammation nor to malignant disease." Goitre is due either to hypertrophy or to the development in the substance of the gland of adenomatous growths, which may either be solid or cystic, but are generally the latter. These growths are always encapsuled. Hypertrophy of the thyroid, or parenchymatous goitre as it is perhaps more usually, though not more accurately, designated, may, though involving both lobes and reaching very large dimensions, give rise to no or to very slight inconvenience, and when this is the case there is no necessity to interfere with the condition surgically. Often, however, the patient experiences from time to time some difficulty in breathing. Sometimes this dyspnœa is very severe, comes on suddenly, and endangers life. It has proved fatal very quickly and quite unexpectedly. It may recur from time to time with little or no warning, and so make the patient's position one of great anxiety and distress without ever actually endangering life. The difficulty of breathing may also be continuous and progressive, so as slowly to destroy life by suffocation. That thyroid enlargement of any kind should cause dyspnœa is not to be wondered at when the very intimate connection between the gland and the trachea is borne in mind. The trachea is apt to be pressed upon and flattened from side to side, when both lobes are enlarged and compressed, or displaced to one or other side by enlargement of one lobe only. Whenever the trachea is encroached upon by the pressure of a goitre, it is almost invariably flattened laterally. In those very formidable and fortunately rare cases where an enlargement of the thyroid extends behind the manubrium of the sternum and presses upon the trachea, occasionally but only rarely even in such cases, the flattening is antero-posterior and not lateral. Whether the trachea be flattened or displaced, in either case the breathing is liable to be more or less interfered with. The difficulty in breathing is practically always due to direct pressure, that is to say, unless the trachea be directly pressed upon you very seldom have dyspnœa. The trachea may be displaced to a considerable extent, and little or no inconvenience in breathing ever be experienced. Much depends upon the rapidity with which the enlargement of the thyroid and consequent pressure upon the trachea takes place. The more acute the enlargement the greater is the liability for dyspnœa to arise; and the more chronic the disease the less is it likely to give rise to difficulty in breathing. Bleeding into the substance of a parenchymatous goitre occurs occasionally, and then the enlargement is sudden and great difficulty in breathing arises, so much so as to call for surgical interference to prevent the patient dying from suffocation. Inflammation seldom attacks a goitre of any kind except as a result of traumatism. When inflammation occurs, it is practically always the result of some surgical interference, such as the injection of some fluid into a parenchymatous gland or the tapping of the cyst. The inflammation may readily cause, from a rapid increase in the size of the gland, difficulty in breathing. It may also run on to suppuration. Should suppuration result, the pus must be let out as soon as ever it is detected. An abscess may burst into the trachea and suffocate the patient, or matter may find its way into the cellular tissue of the neck, a very serious condition, and one very difficult to treat with much prospect of success. There are certain periods of life and certain conditions of the body when a goitre is likely to enlarge rapidly. It is well known that a relation exists between the thyroid gland and the generative organs. Hence at or about the age of puberty an enlargement of a previously small goitre often causes difficulty in breathing. A great number of the parenchymatous goitres operated upon occur in young people at or

about the age of puberty. Particularly is this the case with boys, which accounts probably for the number of youths suffering from parenchymatous goitre, who are operated upon for the relief of dyspnœa. During pregnancy, too, and at the menstrual period, women, the subjects of parenchymatous goitre, not infrequently regularly suffer from some difficulty in breathing, owing to an increase in size of the goitre. All goitrous subjects are apt to experience dyspnœa whenever they suffer from catarrh, and often at no other time. Of the medical treatment of goitre it is unnecessary for me to speak. It is the parenchymatous variety which is in any way amenable to medical treatment. Adenoid disease is a surgical affection, and no benefit is likely to be derived from its medical treatment. Various surgical methods have, from time to time, been resorted to for both varieties of goitre. Setons and injections of various kinds have been employed. Ligature of the thyroid arteries, division or removal of the isthmus are still occasionally practised, but extirpation of a part of the gland in parenchymatous enucleation of the growth from the substance of the gland when the disease is due to adenoma are, however, the recognised operations of the present time. No one would now, I suppose, for one moment entertain the idea of treating goitre by means of a seton, though not so very long ago this method was recommended and practised. Sir Felix Semon recommends the injection of from twenty to thirty drops of tincture of iodine into a parenchymatous goitre every third day, and he has given precise directions as to how this treatment should be carried out. The results, however, are not by any means encouraging. Suppuration and death have ensued far too frequently to make this treatment popular. Other fluids, such as tincture of iron, have been injected into a goitre with the view of a cure, but the principle of injection has little to recommend it, and should not be resorted to. Permanent cures are quite exceptional from this method, and the risk is very considerable. Ligature of the thyroid arteries has lately been revived as a cure for certain cases of parenchymatous goitre. At best this is an uncertain means by which to attempt to cure any form of goitre. The supply of blood to the gland is so remarkably free, that unless both the superior and inferior thyroid arteries be tied, a cure can hardly be hoped for. It is not a particularly difficult thing to ligature the superior thyroid vessels, but to do so will not interfere permanently with the size of an enlarged thyroid. If in addition the inferior thyroids be tied, the operation has become a severe and difficult procedure. The inferior thyroids are deeply situated behind the gland, and there is a considerable risk of wounding veins in the attempt to reach and tie them. The operation necessary to tie the four thyroid arteries, or three of them, which has been recommended, is quite as serious a procedure as removal of half the gland, and is not nearly so likely to permanently cure the patient. Simple division of the isthmus has been resorted to in cases of parenchymatous goitre for the purpose of relieving urgent dyspnœa. Temporary relief has usually followed this comparatively simple operation, but with the healing of the wound the difficulty in breathing has recurred. This operation and that of excision of the isthmus have both been resorted to in the belief that the difficulty in breathing was due to pressure of the isthmus on the trachea, but the belief is an erroneous one. If it were true, immediate relief would follow either division or excision of the isthmus, but though relief has in most cases followed both procedures it has not been immediate. It is just in those cases where immediate relief from urgent dyspnœa is imperative that the isthmus will be divided, and as the relief does not follow at once there is not much to be said for the operation. The relief which follows the division or excision of the isthmus for urgent dyspnœa is due, not to the relief from pressure of the isthmus on the trachea, but to drainage from the cut surfaces, causing reduction in the size of the lobes generally, and thus relieving the lateral pressure which is the cause of the difficulty in breathing.

The pressure upon the trachea is not from before backwards, but is almost invariably exerted laterally. This has been demonstrated over and over again, is admitted, and there is no dispute about it. The dyspnœa of goitre is often of a spasmodic nature, suggestive of spasm of the glottis from irritation of the recurrent laryngeal nerve, but it is exceedingly doubtful whether in goitre this ever is the explanation of the difficulty. In malignant disease, where the structures round about the growth are infiltrated, spasm of the glottis from implication of the recurrent laryngeal nerve is by no means uncommon, and indeed is one of the consequences to be looked for. The character of the dyspnœa in malignant disease is quite different from that in goitre. In goitre the difficulty is very generally almost always tracheal, and there is seldom any difficulty in speaking or aphonia present. In malignant disease the difficulty is often laryngeal, so much so, indeed, that aphonia or dysphonia, in a case of disease of the thyroid, should always make us suspicious of malignancy. Whenever it becomes necessary to interfere surgically with a parenchymatous goitre, the proper operation to be performed is thyroidectomy. It is undesirable to remove the whole of the gland on account of the liability of myxœdema ensuing, but the greater part of both lobes and the isthmus, or one lobe with or without the isthmus, may be excised with very little risk of such a result arising.

Myxœdema has, however, followed—and sometimes rapidly—the removal of one lobe only. It may be necessary to operate under urgent circumstances during an attack of dyspnœa; but in the majority of cases the operation is performed during an interval when the danger of suffocation during the operation is very much diminished. The risk of death occurring from suffocation during removal of part of the thyroid is undoubtedly very much increased by the administration of a general anæsthetic. It is best, therefore, particularly in those cases where the breathing is already, at the time of operation, embarrassed, and even in those cases where it is known dyspnœa is likely to arise, not to place the patient under a general anæsthetic. Some local means of blunting sensibility may be resorted to, such as the injection of cocaine into the line of the skin incision shortly before operating. The operation is by no means a particularly painful one, and no hesitation need be felt in performing it without any kind of general anæsthetic, should it be desirable or necessary to do so. The administration of a general anæsthetic has caused death during thyroidectomy on numerous occasions, even when no particular danger was apprehended. Death too has arisen, from the anæsthetic, even before the operation has been commenced, sufficiently often to emphasise the danger of its administration. Thyroidectomy may be a comparatively easy operation, and, on the contrary, it may turn out to be a most difficult and tedious one, taxing the patience and skill of the surgeon to the utmost. The object of the operation is to relieve dyspnœa, due to direct and nearly always lateral pressure of the enlarged gland upon the trachea, and as this object can be accomplished by removal of one lobe, and at most of part of the other, it is seldom necessary to remove more; and, at any rate, the whole of the gland should not be taken away. If only a small part be left, the risk of myxœdema arising is very much diminished. In performing the operation the most important principle is to gain free access to the parts involved. Some differences of opinion exist among surgeons as to the best skin incision. All agree that it must be a very free one, but upon the direction of the incision there is not quite the same unanimity. Erichsen recommends "an incision in the middle line from the upper border of the tumour to the sternal notch." Kocher, who is certainly the greatest authority upon the surgical treatment of goitre, recommends "a transverse curved incision as the best." Its advantages are, he says, "It falls along the line of cleavage of the skin and gives rise later to an almost imperceptible scar. A transverse curved incision with the concavity dissected upwards is carried across the most prominent part of the swelling, and is prolonged farther upwards and backwards over the sterno-mastoid upon the side on which the disease is most marked. The incision is placed on a higher or lower level, as may be required; in goitre situated low down, quite at the lower part of the neck." Berry, in his exhaustive and excellent treatise on the diseases of the thyroid gland, advocates an oblique incision parallel to the inner margin of the sterno-mastoid. He says, "This is the incision that should be adopted in most cases of extirpation, and in those generally in which the operation is likely to be difficult or dangerous. It gives the operator plenty of room, and enables him to reach the upper horn of the gland without undue difficulty. The lower end of the incision should, in almost all cases, be carried down to the upper border of the sternum if it be intended to operate upon both lobes of the thyroid, the lower part of the incision being carried well across the middle line at a somewhat higher level than the top of the sternum." Attention should also be drawn to an angular incision which Kocher recommends, "for the sake of those who have less experience." It is commenced over the prominence of the sterno-mastoid at the level of the thyroid cartilage, and extends almost transversely in the direction of the skin creases as far as the middle line of the neck, and from thence vertically downwards to the sternal region. In deeply situated goitres it is prolonged on to the manubrium sterni. Whichever of these incisions is adopted, and there is something to be said in favour of each, according to the size and position of the tumour, the subsequent stages of the operation are much the same. The platysma and fascia are to be divided freely to the same extent as the skin. Any veins in the way must be carefully secured between two catgut ligatures, and divided. If now, by drawing to the outer side the sterno-laryngeal muscles, sufficiently free access can be obtained to the gland, the operation may be completed without dividing any muscles. When this can be done it is an advantage, but unfortunately it is seldom possible. Should there be any difficulty, either owing to the size of the goitre or to its fixation, it is better to divide these muscles and turn them down at once. It may be requisite also to divide, or to partially divide, the sterno-mastoid muscles. It is much better to divide all the muscles than to be cramped by want of room. The capsule of the gland must be thoroughly exposed. It is not to be opened, the object being to remove the gland covered by its capsule. There may be some difficulty in recognising the capsule. It is a very delicate structure. Immediately beneath it large distended thin-walled veins, readily torn, will be seen coursing over the surface of the gland. The next step is to free the gland, covered by its capsule, from surrounding structures. This must be done with great gentleness, care being taken not to tear the capsule, nor any veins,

and not to bruise the gland by any rough handling of it. The gland is best separated from its surroundings by means of the forefinger. Then the vessels supplying the gland are to be secured. The superior thyroid arteries with their accompanying veins are usually easily secured. The artery enters the apex of the lobe on its inner border and upon its anterior surface. It is much smaller than the inferior thyroid, and should be tied with a double silk ligature. Sufficient tissue must be left between the two ligatures to render that on the distal side of the tumour secure from slipping when the intervening portion is cut through. Some veins are usually found passing from the middle zone of the lobe to the internal jugular. Under normal conditions of the gland these veins are small and insignificant; but when the thyroid is enlarged from goitre they are often large, and should be carefully sought for, isolated, and tied with double ligatures of silk, the distal ligature being applied as far from the junction with the internal jugular as possible. In securing these veins great care must be taken not to injure the internal jugular vein, which, distended with blood, and sometimes considerably displaced, has from time to time been punctured. The group of veins lying on the trachea, included under the name of inferior thyroid, which empty themselves into the innominate, must be secured and divided between two ligatures. Sometimes a thyroidea ima artery is found also lying on the trachea, passing from the isthmus generally to the arch of the aorta, and may also require to be tied. It should now be possible to dislocate the gland towards the middle line for the purpose of finding and ligaturing the inferior thyroid artery. The inferior thyroid is a much more difficult artery to secure than the superior. It is a much larger vessel, deeply situated and distributed upon the posterior surface of the gland. It is not, as a rule, accompanied by a vein, but occasionally a middle thyroid vein may leave the gland close to the spot at which the inferior thyroid artery or some one or other of its divisions enters the gland. It is best to tie the inferior thyroid artery after it has divided into branches as near as possible to the tumour, and any veins may be secured in the same ligature. The inferior laryngeal nerve has a close but not a constant relation to the inferior thyroid artery, usually lying superficially to the vessel, but sometimes passing beneath it, and occasionally being found among its branches. Great care must be taken not to injure the nerve at this stage of the operation. The isthmus has now to be dealt with. Along its borders run branches of the superior and inferior thyroid arteries, inosculating with those from the opposite lobe. Upon the surface of the isthmus some veins may generally be seen. These vessels should all be ligatured, after which the isthmus may be divided with scissors. In some cases it has been found convenient to divide the isthmus before tying the inferior thyroid artery, more particularly in those where the isthmus is bulky. The only connection now to be dealt with is that between the trachea and the otherwise free lobe which is to be removed. In separating this often tough connection there is often danger of dividing the recurrent laryngeal nerve. During the performance of the somewhat prolonged, it may be, operation of thyroidectomy, every precaution and care must be taken to keep the wound aseptic. No bleeding vessel, however small, artery or vein, should be left unligatured. All clots must be removed by gentle sponging. If it has been found necessary to divide muscles they must be as accurately as possible sutured with catgut. The skin wound should be closed with horsehair sutures, and it is best to drain the large and somewhat irregular wound by means either of a tube or piece of gauze. Healing should take place very quickly without constitutional symptoms.

Adenomatous Tumours.—Adenomata occur commonly in the thyroid gland. They vary considerably in size and number, and one or more may exist in one or both lobes. Occasionally an adenoma is found developed in the substance of the isthmus. They are usually cystic, and then may grow to considerable dimensions. Bland Sutton, in his work on tumours simple and malignant, gives a drawing of one successfully enucleated by T. Burns, which reached as low as the umbilicus. Whether solid or cystic they are always encapsuled, and should, except under very exceptional circumstances, be removed by enucleation. It may be necessary to remove the lobe in which a number of cysts are grouped together, on account of the serious bleeding which would attend an attempt to enucleate them one by one, but enucleation should always be preferred when practicable. Whenever an adenoma, solid or cystic, gives rise to dyspnœa, it should always be removed. Though causing no inconvenience, if they should be considered unsightly, if they be increasing in size, or in order to prevent their becoming the seat of inflammation or of the extravasation of blood into them, which is not so very uncommon an occurrence, particularly in the cystic variety of the disease, they should either be enucleated or left alone. A cystic goitre should not be tapped. To enucleate an adenoma, whether cystic or solid, the capsule of the thyroid gland must be exposed much in the same way as when a part of a lobe is about to be excised. In enucleation the capsule of the thyroid and the thyroid tissue lying over the adenoma have to be divided. Before doing this, however, any veins on the surface of the thyroid, in the line of incision, should be tied with double silk ligature and divided. Then the superjacent

thyroid tissue must be cut through till the capsule of the tumour is reached. By means of the forefinger the whole surface of the capsule of the growth must be carefully, but as quickly as possible, freed from the thyroid tissue in which it is embedded. This is the stage of the operation at which, not infrequently, very free and sometimes alarming bleeding takes place. As soon as the growth is enucleated every bleeding vessel must, as rapidly as possible, be secured and tied with silk. The interior of the cavity left in the thyroid by the enucleation of the growth must be quite dry and free from clots before it is closed. It is better to drain the wound by means of a little gauze. Union should take place by first intention, and the scar should be almost imperceptible.

GRAVES' DISEASE.—The pathology of Graves' disease is not at present thoroughly understood. The condition has been most successfully dealt with by physicians. Although it is probable no case has been actually wholly cured, the condition of the patients suffering from Graves' disease has been much improved by medical treatment, and the progress of the disease has undoubtedly, in numerous instances, been permanently arrested. The surgical treatment of Graves' disease has not been nearly so successful. According to the views entertained by the surgeon as to the pathology of the affection, various operations have been performed. The superior cervical ganglion has been removed, the sympathetic nerve has been divided, and both sympathetics have been dissected out from the neck and removed, together with their ganglia, on the supposition that the condition might be due to disease of the sympathetic nerves. No sufficiently satisfactory results have followed any of these operations to warrant their repetition. Kocher has tied the thyroid arteries for cure of Graves' disease a number of times, but again the permanent results have not been satisfactory. No case has been actually cured by either of these procedures. A portion of the thyroid, generally one lobe, has been removed a good many times on the theory that Graves' disease was due to functional over-activity of the thyroid gland. Some of these patients are reported to have been completely cured, others to have benefited, and a large proportion to have died during the operation, or a very few hours after. Stars, who has published some statistics on the results of cases of Graves' disease treated by excision of a part of the thyroid, says, "Death after these operations is not due to hæmorrhage, nor to any want of antiseptic precautions. In all the cases reported there has been a sudden rise of temperature to 105°, 106°, 107°, a very rapid pulse (180 to 200), extreme nervous excitement and restlessness, with great anxiety and distress, profuse sweating, and finally collapse and death from heart failure." There is considerable difference of opinion among surgeons who have experience of this method of treating Graves' disease as to its efficacy, and on the whole the balance of opinion is against it, or any other surgical interference.

MALIGNANT DISEASE OF THE THYROID, whether carcinoma or sarcoma, is fortunately a very rare disease. It is seldom met with in patients younger than forty years, and there are no means by which with any certainty a sarcoma can be distinguished from a carcinoma. Malignant disease of the thyroid is a most unsatisfactory condition to treat surgically, the mortality arising directly from operation being very high and immunity from a recurrence being very short. The nature of the disease can seldom be diagnosed early enough for the patient to be operated upon with much prospect of success. Clinically, malignant disease is met with in two stages. In the earlier the growth is confined within the capsule of the gland, and there are no signs by which it can, in this stage, be distinguished from a non-malignant disease. Later on, when the growth has extended beyond the boundaries of the capsule and is infiltrating neighbouring structures, the growth is apt to become irregular in outline, and it will be fixed, grow fast, and give rise to a considerable amount of pain. The trachea is often invaded by a prolongation of growth into it, the carotid sheaths become infiltrated and fixed to the growth. The recurrent laryngeal nerve, too, is pressed upon and is apt to become embedded in the rapidly increasing growth, and in consequence speech becomes difficult; the voice is altered in character, and spasm of the glottis may give rise to alarming attacks of difficulty in breathing, any one of which may destroy the patient. Sometimes, too, there is difficulty in swallowing. These symptoms make, at this stage of the disease, diagnosis easy, but unfortunately it is quite hopeless to interfere with a view of curing the patient by a removal of the disease. It is only while the growth is confined within the capsule that there is any chance of removing it successfully, and even then the prospect is a very forlorn one. Sometimes malignant disease of the thyroid runs a very acute course. Berry mentions two cases which terminated fatally—one in three, the other in four months after the first appearance of the growth. They were both males, aged 60 and 25 years respectively.

TRACHEOTOMY IN THYROID DISEASE.—In certain cases of thyroid disease where death is imminent from difficulty in breathing, tracheotomy may be the only means of affording the immediate relief which is necessary in order that life may be prolonged. The necessity for tracheotomy is most likely to arise in cases of malignant disease of the gland. Should the case be one where the urgent symptoms are due to pressure upon and displacement of the trachea by a simple growth or parenchymatous goitre, the proper treatment

is either enucleation or excision. Tracheotomy should never be resorted to if it can be avoided. If for any good and sufficient reason it should be found impracticable, even in cases of non-malignant disease, to remove a swelling pressing injuriously upon the trachea, then tracheotomy might have to be resorted to as the only expedient possible. The difficulties and risks of the operation may be found to be very great indeed. The veins of the neck are often, in malignant disease, for which the operation is most likely to be called for, large and distended with blood. There may be great difficulty in reaching the trachea, either because it is embedded in the growth, or displaced and perhaps altered in shape, or it may be both embedded and displaced to a considerable extent, rendering the operation one of enormous difficulty. The trachea may be displaced to the outside of the carotid artery. The high operation should be performed; usually no other is practicable, and it may be found necessary to cut through growth to get into the trachea. Having opened the trachea, it will usually be found that the ordinary tracheotomy tube is not long enough to reach sufficiently low to relieve the difficulty in breathing, and that a special tube must be used for the relief of dyspnœa in malignant diseases of the thyroid. The relief gained has usually been only very temporary, and life has been prolonged for but a short time.

Thyroid Gland, Therapeutics of.—The administration of some preparation of the thyroid gland is now universally recognised to be the specific treatment for myxœdema and cretinism. When the treatment was first introduced attempts were made to transplant the gland of one of the lower animals to the body of the patient; but the effects of such procedures, although beneficial at first, diminished, and finally ceased as the gland became absorbed by the tissues of the host. Next, thyroid feeding was carried out by the administration by the mouth of the sheep's gland itself, either raw, lightly cooked, or minced up and spread on bread. Excellent results were obtained, but the method is obviously inconvenient. A glycerin extract made by soaking the gland in an equal quantity of glycerin for twenty-four hours and then squeezing out the active ingredients will keep for a considerable period. It is given in doses of from 3 to 15 minims. The *Liquor Thyroidei* of the B.P. is made in a similar manner with the addition of carbolic acid (·5 per cent). *Thyroideum Siccum*, the dry powdered thyroid of the sheep, is the form now most commonly employed. It is given in doses of from 1 to 15 grs. or more often in cachet or in the convenient form of the tablets of thyroid extract (1½ and 5 grs. in each). At the commencement of treatment a small dose is prescribed, and this is gradually increased until the proper dose for the particular patient is determined. In myxœdema and cretinism the administration must be kept up permanently. *Thyroglandin* is a dry thyroid extract given in pills of 1 gr. or tablets of 2 grs. *Iodothyrine*, prepared from the pancreatised gland by extraction with petroleum ether, solution in soda, and precipitation with sulphuric acid, is made up in the form of tablets, each containing 5 grs. "In myxœdema" the dosage may be in the form of minute amounts daily, or as full quantities for a few days in each month. In some cases of obesity the use of thyroid has given good results, but care must be exercised in treating such cases with this drug. It has been recommended in large doses in certain forms of mania, melancholia, and dementia. Good results have been obtained from heroic doses of thyroid in the prevention and relief of early symptoms of eclampsia. It has been tried at various times in exophthalmic goitre, but the general opinion now is that it is positively contra-indicated in this disease. Cases of psoriasis and chronic eczema have been temporarily benefited by the administration of thyroid. Its precise value in various other diseases, in the treatment of which it is being given more or less experimentally, has not yet been definitely decided Overdoses cause headache, feeble rapid action of the heart, lowering of the blood-pressure, nausea, diarrhœa, restlessness, and rarely delirium. The action of the parathyroid glands is not yet fully understood, and preparations from them have not been introduced into general use. *Anti-thyroid Serum* (Moebius) is obtained from the serum of animals which have undergone thyroidectomy. It is given in doses of from 8-75 minims by the mouth or hypodermically in cases of exophthalmic goitre, but the results are inconclusive. The milk of thyroidectomised animals has also been employed in this disease. *Rodagen* is a powder consisting of the dried milk of thyroidectomised goats, with milk sugar added as a preservative. It is given in Graves' disease in doses of 5-10 grammes daily. *Thyroidectine* is prepared from the dried blood of thyroidectomised animals; it is given in doses of 5 grs. or more in cachet. None of these anti-thyroid preparations can be said to have given conclusively good results in the treatment of exophthalmic goitre.

Thyroidism. *See* THYROID GLAND, MEDICAL (*Exophthalmic Goitre, Pathological Anatomy, Effect of Thyroid Extract*).

Thyroiditis. *See* THYROID GLAND, MEDICAL (*Thyroiditis, Acute*).

Thyroidotomy.—Incision of the thyroid gland.

Thyroprivus.—A morbid state following upon loss or removal of the thyroid gland.

Thyrotomy.—the splitting or incision of the thyroid cartilage of the larynx. *See* LARYNX, MALIGNANT DISEASE (*Treatment*); LARYNX, AFFECTIONS OF CARTILAGES (*Stenosis, Treatment*).

Tibia.—The larger of the two bones of the leg, the smaller being the fibula; the vessels and nerves associated therewith are named *tibial*, and muscles connected with the bones are called *tibialis* (anticus and posticus). *See* ANEURYSM (*Lower Limb, Tibial*); ANKLE-JOINT. REGION OF, INJURIES (*Displacement of Tibialis Tendon*); ARTERIES, LIGATURE OF (*Anterior and Posterior Tibial*); DEFORMITIES (*Bow-Legs*); FRACTURES (*Bones of Leg*); KNEE-JOINT, DISEASES OF (Tibio-Fibular Joint); KNEE-JOINT, INJURIES OF (*Fracture of Tibia*).

Tibio-.—In compound words tibio- means relating to the tibial bone, *e.g.* tibio-calcanean (belonging to the tibia and the os calcis).

Tic.—A twitching or convulsive movement of certain muscles, occurring generally as a habit; painless tic. General convulsive tic may be mistaken for chorea (*see* CHOREA, *Diagnosis*). *See also* NEUROSES, OCCUPATION.

Tic Douloureux.—Neuralgia of the fifth nerve in one or more of its branches; also, twitching of muscles supplied by the seventh nerve. *See* ALCOHOL (*Indications in Disease*); HERPES (*Herpes Ophthalmicus, Diagnosis*); HYPNOTISM (*Treatment of tics in Degenerate Children*).

Tickling. *See* PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Common Sensibility, Tickling and Tingling*).

Ticks.—Varieties of *acarus*; *tick fever* is also called Texas Fever and ixodiasis of Uganda and other places. *See* PARASITES (*Sporozoa, Texas Fever*); SKIN DISEASES OF THE TROPICS (*Ixodiasis*).

Tidal Air.—The air which enters and leaves the chest during ordinary respiration, averaging 300 c.cms. *See* PHYSIOLOGY, RESPIRATION (*Amount of Air Respired*); RESPIRATION (*Rhythm and Rate*).

Tidy's Process.—A process by which the organic matter present in water is estimated; the oxygen process; a permanganate solution is added to the water along with a little dilute sulphuric acid, a change in colour indicates the presence of organic matter, and the calculation of the oxygen consumed is done by means of iodide of potassium, thiosulphate of sodium, and starch solution.

Tight-lacing. *See* LIVER (*Perihepatitis, Causation, Traumatic*; MENSTRUATION AND ITS DISORDERS (*Menorrhagia, Causes, General*).

Tightness, Feeling of. *See* INDIGESTION (*Symptoms*); STOMACH AND DUODENUM (*General Symptomatology, Local*).

Tigretier.—A form of religious mania with delusions of demoniacal possession met with among Abyssinian women; it is probably a severe form of hysteria.

Tin. *See* TOXICOLOGY (*Inorganic Poisons, Irritants*).

Tincæ, Os. — The os uteri externum; literally, the mouth of a tench, from the sensation given by it to the finger.

Tinctura or Tincture.—A solution of the active principle of a drug in alcohol, made by maceration or percolation or simple solution, *e.g.* tinctura opii, tinctura arnicæ, and tinctura quininæ respectively; most tinctures are given in doses of ½ to 1 fl. dr. or of 5 to 15 minims, but the tincture of iodine has a dose of 2 to 5 minims. *See* PRESCRIBING.

Tinea.—Ringworm (*Lat. tinea*, a worm or moth), or, more generally, a spreading skin disease, due most often to a parasitic fungus. *See* NAILS, AFFECTIONS OF (*Parasitic, Tinea unguis*); PSORIASIS (*Diagnosis, Tinea Circinata*); SKIN DISEASES OF THE TROPICS (*Craw-craw, Dhobie Itch, Tinea Imbricata*); SKIN, PARASITES (*Tinea Tonsurans, Tinea Circinata, Tinea Barbæ, Tinea Unguium, Tinea Versicolor, Tinea Erythrasma*); SYCOSIS (*Diagnosis*).

Tingling.—A stinging sensation, often called "pins and needles," felt after pressure on a nerve or in various nervous diseases. *See* BRAIN, INFLAMMATIONS OF (*Progressive Softening, Clinical Features*).

Tinkling, Metallic.—A chinking sound heard on auscultation, especially in cases of pneumothorax. *See* PLEURA, DISEASES OF (*Pneumothorax, Physical Signs*).

Tinnitus Aurium.

Varieties 27
Diagnosis 27
Treatment 28

See also AUDITORY NERVE AND LABYRINTH (*Nerve Deafness*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Hyperæmia, Symptoms*); CHLOROSIS (*Symptoms*); EAR, EXAMINATION OF (*Symptomatology*).

THIS symptom, described by patients as buzzing, singing, or ringing in the ears or head, is especially common in diseases of the ear, giving rise to defective hearing, but it may be associated with perfectly normal hearing, and in rare instances with abnormally heightened auditory acuity.

The noises are in general termed "subjective," inasmuch as there is no objective cause for them

outside the individual, and, accurately speaking, a distinction should theoretically be drawn between entotic or periotic and purely subjective noises which arise from irritation of the auditory nerve or centres by disturbances which would not in themselves produce sound, or, in other words, by stimuli which are not sonorous. Such would be the sensations of sound arising from fluxional or inflammatory disturbances of nutrition in the labyrinth, auditory nerve, or auditory centres. On the other hand, the pulsations in the arteries of the ear, the muscle sounds, and the crepitations and cracklings accompanying middle ear catarrh are in themselves sonorous, and the sound perceived by the patient is the result of sonorous vibrations in the structures of the ear, and therefore sometimes differentiated as entotic. The distinction, though interesting, cannot always be drawn, and it is of theoretical rather than practical value. With regard, however, to entotic noises, they become audible when an obstructed condition is present which shuts in the sound, and when clinically there is increased bone-conduction for the tuning-fork.

Noises in the head are sometimes classified according to their characters and according to patients' descriptions. These are unlimited in number—chirping, hissing, rustling of leaves, boiling of kettles, rushing of water, rumbling of carts, ringing of bells, escape of gas, blowing-off steam, "telegraph wires," Æolian harp, clicking, pumping, musical notes, voices singing, voices talking, sometimes intelligibly, sometimes not.

These noises may be divided into two groups, the "elaborate" and the "simple," the former including such sounds as distinct melodies or voices uttering intelligible words. These may be pure "auditory hallucinations," arising in the brain itself, or they may be "auditory illusions," as when there are simple sound sensations, such as humming or hissing, excited by disease of the ear, but interpreted by the diseased brain as music or speech.

Simple sounds may take various forms, but they may be divided into two great groups, pulsating and continuous.

Pulsating sounds may be taken to be arterial in origin, and we have to endeavour to decide whether the arterial congestion giving rise to them is in the external or middle ear, or in the labyrinth; in the former case they are checked by compression of the common carotid artery in the neck by the well-known method; when produced in the labyrinth they are checked by the less well-known method of compressing the vertebral arteries in the suboccipital triangle; for this purpose the observer presses a thumb and middle finger into the depressions behind and slightly below the mastoid process, formed by the recti and oblique muscles, while counter-pressure is made with the other hand on the patient's forehead. It will be remembered that the labyrinth is supplied by the internal auditory artery, which is a branch of the basilar, and that the basilar is formed by the junction of the two vertebral arteries. Such sounds are sometimes audible to the auscultator; and when they are heard through the bones of the skull and accompanied by giddiness, optic neuritis, and other evidences of intracranial tumour, there is great probability that the tumour is an aneurysm.

Continuous simple sounds may be divided, in the first place, into high-pitched and low-pitched. The continuous tones of a low-pitched character, humming and rushing, are generally venous in origin, and may arise either from passive congestion (as from pressure of a bronchocele or other growth on the jugular vein, or disease of the right side of the heart); others, again, are the muscle sounds, namely, the contraction of the tensor tympani, and these latter are believed to produce the sea-shell noise of which some patients are conscious as resembling the sound heard when a sea-shell is held to the ear. This includes also the humming noises which occur in the subjects of anæmia. A curious "clicking" sound, audible often to the observer as well as the patient, is sometimes produced by "clonic" spasms of the tensor palati and tensor tympani. High-pitched tones are usually due to some stimulation of the labyrinth, as when from increased tension of the tympanic apparatus more or less intermittent pressure is exercised on it by the stapes driven into the fenestra ovalis. It also occurs in disease of the labyrinth itself, whether idiopathic, congestive, inflammatory, or degenerative, or resulting from injuries to the head.

Diagnosis of the Cause of Tinnitus Aurium.—In any given case it is more important to be able to diagnose the cause, and this depends as a rule upon other factors rather than the character of the sound.

It is, in the first instance, necessary to determine whether it depends on some disease of the structures of the ear or upon disturbance in the central nervous system due to local or constitutional conditions. We must decide, in the first place, whether there is an associated abnormality of hearing-power. If the hearing-power is diminished we are entitled to assume that the tinnitus and the dulness of hearing arise from the same cause, and we must decide the form of disease which gives rise to the defect of hearing-power, whether situated in the auditory centres, the internal, external, or middle ear, and it is upon this that the choice of treatment to be adopted depends. The methods of making these distinctions have been set forth in preceding articles, notably in the writer's (Dundas Grant) chapter on disease of the auditory nerve and labyrinth, vol. i. p. 329 of this work.

Given that there is a defect of hearing and the noise is simple, the tinnitus is due to disease of the organ of hearing. Should the simple noise be pulsating in character, and if it can be checked by compression of the carotid artery in the neck, it is due to arterial congestion of the external or middle ear, but if by compression of the vertebral arteries in the suboccipital triangles it is then due to arterial congestion of the internal ear, which it is to be remembered is often of nervous or vaso-motor origin. The tinnitus produced by quinine and salicylates is probably of this nature. It may arise, however, from any cause which increases the arterial tension, notably, more particularly chronic cirrhotic disease of the kidney and vascular disturbance associated with the menopause. Should the pulsating murmur be readily heard on auscultation of the cranium or ear, search must be made for the other signs of intracranial aneurysm.

Should the noise be not pulsating, a high-pitched hissing or singing sound is probably due to increased tension of the tympanic structures, leading to pressure on the labyrinth, or, on the other hand, it may arise from disease of the labyrinth itself. In the former case the tuning-fork tests, probable alleviation on inflation, and the examination of the tympanum will enable us to make a diagnosis. In the latter case the diminution of bone conduction will, among other tuning-fork tests, enable us to decide that the disease is situated in the internal ear. A low-pitched, dull, rumbling, buzzing, or rushing noise, made worse by lying down, the ingestion of food or stimulants, or constipation, indicates venous congestion. On the other hand, if it is relieved by lying down, by taking food or stimulants, and accompanied by pallor, it is due to anæmia. It may be noted that the inhalation of nitrite of amyl increases tinnitus when congestive, but diminishes it when anæmic. If, along with dulness of hearing, the tinnitus is of an elaborated character, such as melodies, or, still more, voices of people talking, the patient may be said to be suffering from auditory illusions, the elaborated character being due to the misinterpretation of the simple sounds induced by the aural disease.

Instead of being defective the hearing-power in some cases is abnormally acute, owing to increased excitability of the auditory nerve or centres, usually associated with neurasthenic conditions. This peculiarity is often overlooked, and the sounds of which the patient is conscious are usually those produced by the circulation and other movements in the structures surrounding the auditory organs.

There remain a certain number of cases in which the tinnitus is unaccompanied by any abnormality of hearing (tinnitus nervosa), and is therefore not attributable to disease of the ear itself, but to some disturbing agent irritating the central auditory nervous structures, and which may be in the form of gross lesions such as new growths, or of the more minute changes in the brain which give rise to mental disease. The tinnitus may be either simple or elaborate in character, the latter being the auditory hallucinations usually associated with insanity. The "Æolian harp" sounds have been found to depend on the irritation of the tympanic membrane by one of the hairs of the external meatus.

Treatment of Tinnitus Aurium.—It has been shown that in most cases this symptom is due to disease of the auditory apparatus, and the treatment to be selected is in the first instance that suitable for whatever auditory disease may be present. The external auditory meatus will have to be freed from masses of cerumen or desquamated epidermis. An indrawn membrane and narrowed Eustachian tube indicate the necessity for restoring the ventilation of the tympanum by inflation or dilatation of the tube, and the freeing of the naso-pharyngeal and nasal passages. In acute inflammatory conditions with arterial congestion of the middle or external ear, depletion by means of leeches on the tragus or incisions into the turbinated bodies may be called for, while derivation to the intestines and the administration of such a sedative as bromide of potassium in full doses is carried out. If the various tests indicate that the exciting cause is situated in the internal ear, we vary our treatment according as it is congestive or anæmic in character. In the former case we should in general administer bromide of potassium, and deplete through the intestines by means of calomel or salines or hydrobromic acid. Pilocarpine is also useful in these cases, having apparently the power of diminishing congestion of the internal ear. If the tinnitus is due to the administration of such drugs as quinine and salicylates, they must be at once withheld. Attention should be particularly paid to the condition of the kidneys and heart, and improvement will sometimes follow the limitation of the diet to milk and fish, with the avoidance of animal food, at all events in any great quantity. Temporary relief may often be obtained by compression of the vertebrals in the suboccipital region by means of the fingers or of two conical pads placed in the hollows already described, and kept in position by a strap going round the forehead. In tinnitus due to venous congestion any conditions leading to pressure on the veins or difficulty in the return of blood to the right side of the heart should be removed. The excision of enlarged glands in the neck, the reduction of a bronchocele, and the regulation of the cardiac circulation by means of digitalis or strophanthus must be kept in view. In case of auditory illusion, which, as before said, is a misinterpretation of the simple sounds produced by aural or central disturbance,

it is most important to attend to the local condition, as successful treatment of this may greatly help in combating the symptoms of insanity which may be present. If the noises are of an elaborate character and hearing is normal in every respect, treatment is more appropriately relegated to the alienist than to the aurist.

If the hearing is abnormally acute (hyperakusis oxyakoia) we have to counteract the neurasthenia, and to select specially those remedies which, while tending to relieve the neurasthenic condition, diminish the acuity of the auditory nerve. Of the latter bromide of potassium is a good type, but quinine is also possessed of such properties that small doses of this remedy have, in the author's experience, appeared at the same time to diminish the sensibility to sounds produced in the surroundings of the ears, and at the same time to give a slight fillip to the nervous system. In any given case it is difficult to say beforehand how it is going to act, but the writer believes that when the patient finds the sound of his own voice unpleasantly loud (apart from abnormal dilatation of the Eustachian tube), and when the condition of the internal ear is one of anæmia rather than congestion, the quinine is most appropriate; it may be given in doses of ½ to 2 grs., and it is wise to combine with it 10 or 15 drops of dilute hydrobromic acid.

Tinnitus nervosa has been found in some cases to yield to the continuous galvanic current. The positive pole is applied to the ear by means of a moistened pad, and a current of from two to four milliampères is allowed to run for about ten minutes at a time, sudden interruptions being carefully avoided. Mental and bodily rest are often indispensable.

Tirol. *See* THERAPEUTICS, HEALTH RESORTS (*Germany and Austria*).

Tisane.—A decoction acting as a medicine, *e.g. tisane de Feltz* (decoction of sarsaparilla with isinglass and sulphuret of antimony).

Tissue. *See* PHYSIOLOGY, THE TISSUES (*Epithelium, Connective Tissue, Muscle*, etc.).

Titubation.—A staggering gait (Lat. *titubo*, I stagger), due sometimes to disease of the nervous system, *e.g.* the cerebellum.

Toast Water. *See* INVALID FEEDING (*Toast Water*).

Tobacco. *See* TABACI FOLIA; *see also* AMBLYOPIA (*Toxic, Tobacco*); ANGINA PECTORIS (*Etiology, Poisons, Tobacco*); APPETITE (*Loss*); COLOUR VISION (*Acquired Colour Blindness*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Etiology, Exciting Causes, Chemical*); RETINA AND OPTIC NERVE (*Central Retinal Affection, Toxic Influence*); TOXICOLOGY (*Alkaloids and Vegetable Poisons, Tobacco*).

Tobi Hi.—Pemphigus contagiosus. *See* SKIN DISEASES OF THE TROPICS.

Tobins Tube.—An inlet for air used in the ventilation of rooms and public buildings. *See* VENTILATION AND WARMING (*Inlets and Outlets*).

Tocodynamometer.—An instrument for measuring the expulsive action of the uterus in labour.

Tocology.—Obstetrics or midwifery (Gr. τόκος, birth; λόγος, discourse).

Toddalia.—Lopez root, the root of *Toddalia aculeata*, used as a bitter in Eastern countries for dyspepsia and dysentery; it is official in the Indian and Colonial Addendum to the B. P. of 1898; there are two preparations, the *Infusum Toddaliæ* (dose, 1 to 2 fl. oz.), and the *Liquor Toddaliæ Concentratus* (dose, ½ to 1 fl. dr.).

Toes. *See* AINHUM; AMPUTATIONS (*Toes*); BRAIN, PHYSIOLOGY OF (*Functions of Cerebral Cortex, Motor Areas*); BRONCHI, BRONCHIECTASIS (*Clinical Phenomena, Clubbing of Toes*); DEFORMITIES (*Hallux Varus, Hallux Rigidus, Hammer-Toe, Toes, Deformities of*, etc.); FRACTURES (*Metatarsal Phalanges*); HEART, CONGENITAL MALFORMATIONS OF (*Clubbing of Toes*); TYPHOID FEVER (*Complications and Sequelæ, Tender Toes*).

Toilet.—The washing or cleansing of a wound or of a body cavity during an operation, *e.g.* the peritoneal toilet in abdominal sections.

Toisson's Solution.—A diluting solution for use in connection with hæmatocytometric estimations; it contains methyl violet, chloride and sulphate of sodium, neutral glycerine, and distilled water.

Tokelau.—A skin disease of the Pacific Islands and Oceania; tinea imbricata or Bowditch Island Ringworm. *See* SKIN DISEASES OF THE TROPICS (*Tinea Imbricata*).

Tolu, Balsam of. *See* BALSAMUM TOLUTANUM.

Toluene or Toluol.—Methylbenzene (C_7H_8, or, rather, $C_6H_5CH_3$), a hydrocarbon obtained from coal-tar and used as a parasiticide; from it is prepared *toluidin* ($C_7H_7NH_2$), a homologue of aniline.

Toluidin-Blue.—The double salt of dimethyltoluthionin and zinc chloride, which has been used in place of methylene blue in gonorrhœal conjunctivitis.

Toluylene-diamine. *See* HÆMOGLOBINURIA (*Toxic*); INTESTINES, DISEASES OF

(*Ulcers, Causes of*); JAUNDICE (*Pathology, Hæmatogenous*); LIVER, DISEASES OF (*Hypertrophic Biliary Cirrhosis, Etiology, Experiments*).

Tolypyrin.—The homologue of antipyrin, tolyldimethylpyrazolon ($C_{12}H_{14}N_2O$), given in the same kind of cases and in similar doses.

Tomatoes. *See* INVALID FEEDING (*Vegetables*); INVALID FEEDING (*Cooking in Diabetes, Stuffed Tomatoes*).

Tomes' Fibres.—Dentinal fibrils, processes prolonged from the tooth pulp into the dentinal tubules. *See* TEETH.

Tonga.—The fluid extract of the root of *Raphidophora vitiensis* and the bark of *Premna taitensis*, obtained in the Fiji Islands; it has been used in neuralgia, dose ½ to 1 fl. dr. of the fluid extract; *tongaline* is a proprietary medicine said to contain salicylate of sodium, of colchicin, and of pilocarpin, extract of Cimicifuga, and tonga bark, and recommended in gout.

Tongue.

DEVELOPMENT 30
ANATOMY 30
INJURIES 31
1. *Wounds* 31
2. *Scalds* 31
INFLAMMATORY AFFECTIONS—
I. PARENCHYMATOUS GLOSSITIS . . 31
II. ACUTE SUPERFICIAL GLOSSITIS . 32
III. CHRONIC SUPERFICIAL GLOSSITIS . 32
IV. TUBERCULOUS DISEASE . . . 33
V. SYPHILITIC AFFECTIONS . . . 34
TUMOURS—
I. CARCINOMA 36
II. SARCOMA 40
III. INNOCENT TUMOURS . . . 40
IV. CYSTS 40
V. THYRO-GLOSSAL CYSTS AND TUMOURS 40
MALFORMATIONS 41
NERVOUS AFFECTIONS 42

See also ACHONDROPLASIA (*Clinical Features, Protrusion of Tongue*); ACTINOMYCOSIS (*Tongue*); ANÆSTHESIA, CHLOROFORM (*Falling back of Tongue*); CHILDREN, CLINICAL EXAMINATION OF (*Mouth*); CRETINISM (*Diagnosis*); GLOSSO-PHARYNGEAL NERVE; HÆMOPTYSIS (*Hæmorrhage from Tongue*); HEMIPLEGIA (*Symptoms*); HERPES (*Herpes of Maxillary Nerves*); KIDNEY, SURGICAL AFFECTIONS OF (*Suppurative Pyelonephritis, Parrot Tongue*); LEPROSY (*Lepra Tuberosa, Symptoms*); PARALYSIS (*Cerebral Diplegia, State of Tongue*); SCARLET FEVER (*Symptomatology, Tongue*); SPRUE (*Clinical Features, Tenderness of Tongue*); SYPHILIS (*Secondary Symptoms, Tongue*); SYPHILIS (*Tertiary, Mouth and Throat*); TYPHOID FEVER (*Symptoms, Tremor of Tongue*).

DEVELOPMENT.—The tongue is developed in relation to the mandibular or first branchial arch and the adjacent cleft. According to His the mesoblast becomes thickened and leads to fusion of the arches and grooves in the front of the neck, whereby the "meso-branchial area" is formed. From the portion of the meso-branchial area which lies behind the lower jaw a projection grows towards the mouth, and constitutes the *tuberculum impar*, which by extending forward ultimately becomes that portion of the tongue which lies in front of the circumvallate papillæ. The base of the tongue and the epiglottis are derived from a second prominence which develops behind and below the tuberculum impar. The lateral portions of this prominence grow forward in a forked manner and enclose the tuberculum, with which they ultimately become fused. The line of fusion is consequently V-shaped, and its position is marked in the fully-developed tongue by the circumvallate papillæ. At the angle of this "lingual V" is a small pit or depression, rarely more than one cm. in depth—the *foramen cæcum*—which represents the upper end of a peculiar strand of embryonic tissue which connects the tongue with the thyroid gland. This strand of tissue is known as the *thyro-glossal tract*, and it extends from the foramen cæcum through the substance of the tongue in the raphé between the génio-hyo-glossi muscles, passing in front of the hyoid bone and thyro-hyoid ligament to the pyramidal lobe of the thyroid gland.

ANATOMY.—The tongue is essentially a muscular organ, and the fibres of which it is composed interlace in a highly complicated manner. Posteriorly it is attached to the hyoid bone; anteriorly, through the medium of the genio-glossus muscle, to the lower jaw. It is covered with stratified epithelium which in some respects resembles that of the skin, and in others corresponds to the covering of a mucous membrane. On the base of the tongue, behind the circumvallate papillæ, is a mass of lymphadenoid tissue —the *lingual tonsil*.

The tongue receives its vascular supply chiefly through the lingual artery and its branches. Small twigs from the facial and ascending pharyngeal arteries also enter the tongue. The blood is returned by a freely communicating plexus of veins into the internal jugular, external jugular, and anterior jugular veins.

The study of the *lymphatic system* of the tongue is of great surgical importance in relation to the question of glandular infection in cancer. The lymphatic circulation in the organ is particularly free, and the communications with the cervical glands very numerous. As any or all of the groups of glands beneath the jaw and in the neck may be infected in cancer, it is necessary to refer briefly to their arrangement. The *submental* glands, two or three in

number, lie in the connective tissue beneath the chin, and receive lymph from the tip of the tongue and the region of the frenum. The efferent channels lead to the submaxillary glands. The *submaxillary glands* occupy the digastric triangle. Some are adherent to the sheath of the submaxillary salivary gland, and some lie in its substance. These glands drain the whole of the front half of the tongue, and are very frequently involved in cases of cancer. The *cervical glands*, which lie along the line of the internal jugular vein and carotid arteries, ultimately receive nearly all the lymph from the tongue, and are consequently those most commonly infected by malignant disease. The large gland which lies at the bifurcation of the common carotid is usually one of the first and most palpably enlarged glands. The *parotid* and *mastoid* lymphatic glands may also be implicated in cancer, but this is comparatively rare.

Nerves of the Tongue.—The muscles of the tongue are supplied by the *hypoglossal* nerve. The *lingual* nerve is the nerve of common sensation and of taste to the anterior two-thirds of the tongue. The *chorda tympani* branch of the facial is also a nerve of taste. The *glosso-pharyngeal* supplies the posterior third of the tongue with sensation and taste. Its communication with Jacobson's nerve may explain the pain in the ear which so frequently accompanies cancer of the base of the tongue. The *superior laryngeal nerve* sends fibres to the base of the tongue and epiglottis, hence the frequency with which disease in this position gives rise to reflex cough, hiccough, and vomiting. This connection may also explain how spasm of the glottis and pharynx may be caused by sudden depression of the base of the tongue. On the other hand, the excitation of the respiratory movements when the tongue is forcibly drawn upon may be due to stimulation of the respiratory centre through this nerve (Laborde); obstinate hiccough, too, has been relieved by traction on the tongue (Lépine).

Salivary Glands.—The salivary glands—particularly the sublingual and submaxillary glands—lie in close relationship to the tongue, and often share in its diseases. (*See* "SALIVARY GLANDS," vol. ix.)

INJURIES OF THE TONGUE

Lacerated wounds of the tongue most frequently result from a bite, as, for example, when a child falls and strikes the chin, the tongue at the moment being protruded between the teeth. During an epileptic fit also the patient may bite his tongue. *Punctured* wounds are less common. They may be produced by a pipe-stem, a displaced tooth, or a sharp fragment of a fractured jaw being driven into the tongue. Bullets occasionally lodge in the tongue. Clean-cut *incised* wounds are very rare.

When the wound merely implicates the anterior portion of the tongue the bleeding is seldom serious except in hæmophylics. Wounds of the posterior part of the tongue, on the other hand, particularly when they penetrate deeply into the substance of the organ, may give rise to free hæmorrhage. If septic complications ensue secondary hæmorrhage may prove troublesome and even dangerous on account of the difficulty of securing the bleeding vessel amongst the soft, disintegrated tissue. This risk is greatest when a foreign body, *e.g.* a bullet or a piece of pipe-stem, remains embedded in the tongue.

Treatment.—Primary hæmorrhage should be arrested at once, the patient being anæsthetised for the purpose if necessary. In wounds near the tip of the tongue, after the clots have been removed, the bleeding can usually be controlled by bringing the edges together with stitches. When the wound is situated far back and passes deeply into the substance of the tongue the bleeding point must be secured by a ligature, and to enable this to be done it may be necessary to enlarge the wound. Ligature of the lingual artery in its continuity is seldom called for in cases of primary hæmorrhage, but may be necessary for secondary hæmorrhage on account of the friability of the tissues rendering it difficult to apply forceps or a ligature to the bleeding point.

Septic complications seldom arise if an antiseptic mouth wash, such as weak Condy's fluid or boracic acid, is frequently used. Punctured wounds should be carefully examined lest a foreign body be left embedded. In some cases such an object as a bullet, a needle, or a portion of a pipe-stem, has remained embedded in the substance of the tongue for many months, and has given rise to a firm, indolent swelling closely simulating a solid tumour. Serious hæmorrhage may accompany the attempt to remove the foreign body in such cases.

Scalds of the tongue frequently occur in children from sucking the spout of a tea-pot or kettle. The pharynx is usually scalded at the same time. The tongue becomes swollen and painful, and if the posterior part is implicated respiration may be interfered with. The injury to the tongue seldom calls for more active treatment than the administration of ice, and the use of an anodyne and astringent mouth wash.

INFLAMMATORY AFFECTIONS OF THE TONGUE

Inflammation may affect the whole thickness of the tongue—*parenchymatous glossitis*; or only its covering epithelium—*superficial glossitis.*

PARENCHYMATOUS GLOSSITIS

Acute Parenchymatous Glossitis.—This condition, in which the whole substance of the tongue becomes acutely inflamed and œdematous, is

comparatively rare. From the records of reported cases it would appear that young adult males suffer more frequently than others. From the fact that the condition occurs oftener during the winter months, it has been assumed that exposure to cold and damp is at least a predisposing factor, and the history of the patient usually bears this out. The infection may follow an acute infectious disease, such as erysipelas or scarlet fever. The determining cause is doubtless an organismal infection by one or other of the numerous forms of bacteria which exist in the mouth. Different forms of the disease—the streptococcal and the staphylococcal for example—are recognised according to the nature of the more active organisms present.

In the *streptococcal variety* the whole tongue becomes swollen and œdematous, and the inflammation tends to spread to the tissues of the neck, setting up that form of acute cellulitis known as "angina Ludovici." The inflammation does not usually go on to suppuration. In the *staphylococcal* variety, on the other hand, the inflammation tends to remain localised to the tongue or even to one part of it, and is liable to end in the formation of a circumscribed abscess.

Clinical Features. — The most prominent symptoms are severe pain and general swelling of the tongue, and these usually come on suddenly. Mastication, swallowing, and speech are seriously interfered with, and there is profuse salivation. In the course of twenty-four or forty-eight hours the tongue may completely fill the mouth and even protrude beyond the teeth. When the swelling is extreme respiration may be so impeded as to necessitate tracheotomy. The submaxillary, salivary, and lymphatic glands are enlarged and tender. As a rule resolution takes place in three or four days, but in the staphylococcal form of the disease a localised abscess usually situated between the genio-hyo-glossi muscles may form.

Treatment.—A sharp purgative should be administered in the first instance, and the mouth should be washed out frequently with an antiseptic lotion. Leeches may be applied to the submaxillary region with a view to diminishing the congestion. When the swelling is interfering with respiration or deglutition several longitudinal incisions should be made into the substance of the tongue. In this way the œdema and congestion are usually rapidly relieved.

Acute Parenchymatous Hemi-glossitis.—In a considerable number of cases the acute inflammation is confined to one side of the tongue, and affects particularly the anterior part. In the centre of the inflamed area a firm nodule is usually to be felt deep in the substance of the organ. The clinical features are the same in kind as those of the condition last described, but the general constitutional effects are less severe, and there is less interference with swallowing and breathing. The inflammation usually subsides without going on to suppuration. The treatment is carried out on the same lines as for acute glossitis affecting the whole tongue.

Mercurial Glossitis.—An acute form of inflammation of the tongue was formerly common amongst those who worked in mercury, and in patients undergoing a mercurial course of treatment for syphilis. Owing to the care now taken in factories where mercury is used, and the greater discrimination employed in prescribing this drug medicinally, this disease is very rarely met with. It differs clinically from ordinary acute parenchymatous glossitis chiefly in that there is much more profuse salivation and extreme fœtor of the breath. It is accompanied also by sponginess of the gums, looseness of the teeth, and other signs of mercurial stomatitis. The *treatment* consists in preventing the further ingestion of mercury, and in employing an antiseptic and astringent mouth wash, such as Condy's fluid, alum, or chlorate of potash.

Gangrene of a portion of the tongue—usually the anterior portion—may result from any form of acute glossitis, or from the spread of cancrum oris from the cheek or gums. It has also followed crushing or laceration of the organ by forceps used to pull out the tongue during anæsthesia. In addition to the use of ordinary mouth washes, frequent washing with peroxide of hydrogen (ten volumes strength) has been found very useful in keeping the mouth sweet. As soon as a line of demarcation forms the slough should be clipped away with scissors.

Glosso-anthrax. — In a few rare cases the tongue has been directly inoculated with anthrax. In addition to the ordinary signs of acute glossitis the characteristic black slough and ring of pustules are present. The condition is almost invariably fatal.

Actinomycosis may also be mentioned as one of the very rare infectious diseases met with in the tongue.

SUPERFICIAL GLOSSITIS

ACUTE SUPERFICIAL GLOSSITIS.—Various forms of inflammation originating in the superficial epithelium of the tongue are met with. As a rule the whole of the buccal mucous membrane is implicated, and the inflammation of the tongue is therefore only part of a general stomatitis (*see* "STOMATITIS," vol. ix. p. 398).

CHRONIC SUPERFICIAL GLOSSITIS.—The term *wandering rash* has been applied by Barker to a comparatively rare affection of the tongue usually met with in children. It consists in the appearance on the dorsum of the tongue of several smooth, red patches, which soon spread and form oval or circular rings with a more or

less yellow margin. The fusion and intersecting of these variegated rings give to the tongue a peculiar appearance, which, on account of its resemblance to the markings on a map, has led to the term "geographical tongue" being applied to it. There are seldom any subjective symptoms. The exact nature of the disease is as yet undetermined. It runs a very slow course, and is but little amenable to any form of treatment, local or constitutional.

Dyspeptic Tongue.—In certain cases of dyspepsia, particularly of gouty origin, the tongue becomes red, raw, and excoriated, and the superficial layers are the seat of a chronic inflammation. The epidermis is destroyed in patches, and the bare surfaces are tender when touched, or when hot or irritating food comes in contact with them. The relief of the dyspepsia is speedily followed by recovery of the tongue condition.

Leukoplakia or Leukokeratosis.—By far the most important form of chronic superficial glossitis is that known as leukoplakia or leukokeratosis. Much of its clinical importance lies in the fact that it is frequently followed by the development of epithelioma in the tongue. The disease consists in an overgrowth of the horny layer of the epithelium covering the organ, with disappearance of the normal papillæ, whereby smooth, bluish white patches are formed on the dorsum of the tongue. The submucous tissue is infiltrated with leucocytes. The white patches, which are best seen when the tongue is dried, have the appearance of being covered by a thin pellicle, which is soft and pliable and firmly adherent to the surface.

The disease is almost invariably met with in males, and may occur at any age between twenty and sixty. Gout, rheumatism, and syphilis appear to be predisposing factors, and any form of irritation—for example, the chewing or smoking of tobacco, the drinking of raw spirits, the friction of a rough tooth or an ill-fitting tooth-plate — tends to produce the condition.

The milder forms give rise to no discomfort, but when the condition is advanced the patient complains of dryness and hardness of the tongue, with impairment of the sense of taste, and in some cases persistent thirst. If the hygiene of the mouth be neglected cracks and fissures may develop, and the patient has then pain on taking hot or spiced food.

Unfortunately treatment is of little avail in most cases, and the condition, even if it disappears for a time, is very liable to recur. Eventually an epitheliomatous tumour may develop in relation to one of the patches or cracks. The tumour may take the form of a nodule in the substance of the tongue, or of a wart or ulcer on its surface.

All sources of irritation, such as chewing tobacco or smoking, should be removed, and an antiseptic mouth wash used regularly. Alkaline washes, such as a solution of bicarbonate of soda, chlorate of potash, or borax, are particularly useful. Butlin highly recommends the application of ointments, for example, vaseline, lanoline, or cold cream, with eucalyptus, borax, or some other mild antiseptic added. The tongue is first dried, and then covered with the ointment, which the patient rubs into the part by rolling the tongue against the roof of the mouth. This should be repeated night and morning. The application of salicylic or lactic acid may be useful in removing the excess of epithelium. Caustics are to be rigorously avoided, as they only cause irritation, and so tend to favour the development of epithelioma. Constitutional treatment has little or no effect on the leukoplakia, even if the patient is syphilitic. Patients suffering from leukoplakia should be kept under observation for long periods, with a view to the early recognition of malignant disease should it supervene.

The "*smoker's patch*" is the name applied to a small, oval area on the dorsum of the tongue, from which the papillæ have been removed as a result of excessive smoking. The patch is slightly raised, smooth, and red, and may be covered with a yellowish brown or yellowish white crust. It causes no pain or discomfort unless the crust is removed, and the raw sensitive surface exposed. If the patient persists in smoking the condition may spread all over the tongue and mucous membrane of the cheeks, and may assume the characters of leukoplakia. The treatment consists in stopping smoking, painting the patches with chromic acid, tannic acid, or alum, and using an alkaline mouth wash.

As a result of chronic superficial glossitis the whole tongue may become perfectly smooth from loss of its papillæ ("*smooth tongue*"), or cracks and fissures of various kinds may appear on its surface. *Eruptions*, comparable to those met with on the skin, in herpes, lichen, eczema, or psoriasis, are also met with on the tongue.

Tuberculous Disease.—The tongue is very rarely the primary seat of tuberculosis. In the great majority of cases in which it is affected the patient suffers from advanced pulmonary or laryngeal phthisis, and the tongue is infected by bacilli from the sputum entering some crack or fissure produced—for example, by a ragged tooth. Less frequently the disease spreads by direct continuity of tissue from a patch of lupus on the face or nose.

Clinically the condition may be met with either as a tubercle nodule in the substance of the tongue, or as an ulcer.

In the *nodular* form a lump, seldom larger than a hazel nut, appears on the side or tip of the tongue. At first this swelling has a thin covering of epithelium, and is firm and painless. In course of time, however, caseation takes place, the nodule softens, the epithelium gives

way, and tuberculous débris escapes, leaving an open sore. This sore may take the form of a short, narrow, deep fissure, with thickened, overhanging edges, which ultimately caseate and break down and leave a tuberculous ulcer. Less frequently the epithelium around the fissure is raised into folds, and gives to the nodule a papillomatous appearance.

Before the nodule breaks it is liable to be mistaken for a syphilitic gumma, while after the fissure has formed it may simulate a cancerous ulcer.

The treatment of these forms of tuberculous disease is to excise a V-shaped segment of the tongue, cutting wide of the disease. The edges of the wound are then united with sutures, and as a rule primary union occurs if the mouth be kept sweet by the frequent use of an antiseptic wash.

The *ulcerative form* is that most frequently met with clinically. It is usually merely a further stage of the nodular form, the ulcer having been produced by the caseation and breaking down of a nodule in the tongue.

The surface of the ulcer is uneven, pale, and flabby, and is covered with a yellowish grey layer of coagulated mucus, with here and there feeble granulations showing through. The edges are sharply defined, sinuous in outline, and as a rule show little or no induration. Around the ulcer there may be seen several small miliary tubercle nodules, and the tissues of the tongue are usually swollen. The ulcers vary greatly in depth, in some cases being quite superficial, in others eating deeply into the muscular substance of the tongue. The tip of the tongue may be completely eaten away, so that it looks as if it had been cut across with a knife.

At first there may be comparatively little pain, but as the disease advances the pain becomes severe, and there is profuse salivation. The submaxillary glands are usually, but not always, enlarged.

The diagnosis is, as a rule, rendered easy by the presence of other evidence of tuberculous infection When no such evidence is forthcoming, it is extremely difficult to distinguish between tuberculous and tertiary syphilitic and cancerous ulcers. A microscopic examination of a portion cut out from the edge of the ulcer may be of some help, particularly in regard to malignant disease. The effects of treatment by iodide of potassium and mercury are not to be relied upon for diagnostic purposes.

The prognosis is on the whole very unfavourable, in view of the frequency with which the patient suffers from other advanced tuberculous lesions. The ulcer may, under suitable treatment, improve, or even heal, but it is very liable to break down again.

Treatment.—Primary ulcers should be treated in the same way as tuberculous nodules—by excising a V-shaped portion of the tongue. Even in cases where other tuberculous lesions are present the operation is often justifiable for the relief of pain and to enable the patient to take nourishment. The wound in the tongue must be accurately sutured, and every possible effort made to secure primary union, otherwise the raw surface may become reinfected from the sputum. When operation is impracticable it is only possible to palliate the symptoms by attending to the hygiene of the mouth, and removing all sources of irritation. Pain may be relieved by the application of ointment containing cocaine or morphia.

Syphilitic Affections of the Tongue.—Syphilitic affections of the tongue are exceedingly common, and the disease may show itself in so many forms that, as Butlin says, "there is hardly any affection of the tongue in which the possibility of syphilis should not be taken into account in making the diagnosis."

The *primary sore* of syphilis is very seldom met with on the tongue in this country. The most common sources of infection are tobacco pipes, the blow-pipes of glass blowers, and similar implements, and spoons, forks, or drinking cups which have been used by syphilitic persons. Direct infection may, of course, occur from kissing. The sore presents the same general characters on the tongue as on other parts of the body. The submaxillary lymphatic glands are markedly enlarged on one or on both sides. The generalised secondary manifestations of syphilis usually appear early when infection takes place on the tongue.

The local *secondary* manifestation most frequently met with on the tongue is the *mucous patch*. This is very common, particularly in the later periods of the secondary stage. Mucous patches may appear on any part of the tongue, but are most common on its edges, where they form smooth, irregular, greyish white areas of a rounded or oval outline.

If the thin white layer be removed a smooth red surface is exposed. When situated on the under surface of the tongue the patches frequently assume a condylomatous or warty appearance. If they are irritated by the teeth or by the passage of food over them, they may become cracked, or even deeply ulcerated. As the secretion from mucous patches is highly contagious the patient should always be warned of the danger of using spoons, cups, pipes, or other articles, in common with those with whom he lives or works. Kissing must also be avoided.

If left untreated the surface of a mucous patch may break down and leave a tortuous or stellate fissure or ulcer which tends slowly to increase in width and depth. The edges are punched out and undermined, the base raw, sloughy, and devoid of granulations, and the sore is exceedingly tender. After healing, these sores leave puckered scars of a silvery or leaden hue.

In addition to the ordinary constitutional treatment by mercury, the mucous patch may be dusted with a powder composed of equal parts of calomel, boracic acid, and starch, or painted with silver nitrate. Butlin strongly recommends painting the affected part with a 10 per cent solution of chromic acid every two or three days. Irritating food, alcohol, and smoking should be avoided.

The *tertiary* manifestations of syphilis in the tongue are "tertiary plaques" or "sclerosing glossitis," gummata, and ulcers.

Sclerosing Glossitis.—This term was applied by Fournier to a condition in which, as a result of cellular hyperplasia, patches of the tongue become infiltrated with leucocytes, and smooth plaques or tuberous masses are formed on the surface of the organ. These patches appear chiefly on the dorsum, and at first only involve the more superficial strata of the tongue. They tend to be oval in outline, are elevated beyond the level of the mucous membrane, and present a dull red mammillated or lobulated appearance comparable to the surface of a cirrhotic liver. They are firm and elastic, and are insensitive. The neighbouring lymphatic glands are not enlarged. If untreated they tend to break down and form superficial ulcers which heal readily. The process of healing, however, is attended with excessive cicatricial contraction, with the result that deep clefts and furrows are formed, and the tongue is permanently disfigured. In its typical form the disease is very characteristic, and is not likely to be mistaken for any other affection of the tongue. The presence of other evidence of syphilis and the speedy improvement which follows antisyphilitic treatment prevent it being confused with epithelioma.

Gummata may appear on the tongue at any time during the tertiary stage of syphilis. They are usually situated on the dorsum, and more frequently towards the centre than at the edges. As they seldom implicate the floor of the mouth or the base of the tongue, the tongue can usually be freely protruded. It is necessary to distinguish between a gumma which has not broken down on the surface and one which has ulcerated. An unbroken gumma forms an indolent, firm swelling in the substance of the tongue, devoid of pain, and seldom accompanied by enlargement of the adjacent lymphatic glands. After breaking down it forms a deep excavated ulcer, which is often extremely painful and tender, and the glands are usually enlarged.

Two forms of gummata are met with—the superficial and the deep or parenchymatous.

Superficial gummata are almost always multiple, and appear as small, hard nodules in the mucous and submucous tissue, projecting on the dorsum of the tongue. They vary in size from a pin's head to a pea and are quite painless. The mucous membrane over the swellings may retain its papillæ or may be quite smooth. These gummata tend to break down early, forming extensive superficial ulcers.

The *deep or parenchymatous* gummata may be single or multiple. They may appear on any part of the tongue, but are most common on the dorsum. They are met with both in the middle and at the margins of the organ. As a result of congenital syphilis they are occasionally met with in children. The swellings vary in size from a hazel nut to a walnut, and form hard lumps, usually deeply placed in the substance of the tongue. The overlying mucous membrane is of normal colour, but has usually lost its papillæ. Sooner or later, if untreated, the gumma approaches the surface, and after softening breaks down, leaving a deep ragged ulcer, with a foul sloughy surface and firm raised edges. The edges, however, are seldom indurated and everted like those of a cancerous ulcer.

The differential diagnosis of these gummatous swellings is often very difficult. They have to be distinguished, on the one hand, from innocent tumours, such as fatty or fibrous tumours, and on the other from carcinoma. The main points characteristic of innocent tumours are that they are seldom multiple, they are well defined, and movable in the substance of the tongue. Carcinoma is almost invariably single, is usually situated near the margin of the tongue, and generally occurs in patients over forty years of age.

Syphilitic Ulcers and Fissures.—These are almost invariably the result of the breaking down of a gumma. The ulcers are characterised by having a ragged and sloughy base, on the surface of which is a yellowish grey slough, aptly compared in appearance to a piece of wash-leather. The edges are steep, ragged, and often undermined, but they want the typically indurated and everted character of those of a cancerous ulcer. The surrounding parts of the tongue are thickened and firm. The neighbouring glands are not necessarily enlarged. The ulcer is extremely painful when irritated by food, hot fluids, or spirits. If untreated the sore may remain indolent for months, and show no sign either of spreading or of healing, but it may at any time take on the characters of a malignant ulcer.

Syphilitic fissures are met with as long, narrow, deep clefts, or as stellate or sinuous cracks in the substance of the tongue. After the healing of these ulcers and fissures permanent clefts and tumours remain in the tongue.

Treatment.—The tertiary manifestations of syphilis in the tongue are treated on the same general lines as other tertiary lesions. Iodide of potassium, with or without mercury, is given in large doses, beginning with 10 or 15 grains three times in the day, and increasing to 25 or 30 grains. Locally an antiseptic mouth wash,

such as chlorate of potash, Condy's fluid, or black wash diluted with lime water, is used. Mercurial ointment may be applied with benefit. If the pain is severe a powder containing iodoform, borax, and a small quantity of morphia may be insufflated. Orthoform is also useful in relieving the pain. Before any of these local applications are used the sore must be thoroughly cleaned and dried. Irritating foods and fluids should be avoided, and any irregularities in the teeth must be attended to. When the diagnosis is doubtful and speedy improvement does not follow these measures, the question of excision of the tongue must be considered.

Tumours of the Tongue

The tongue is very frequently the seat of malignant tumours. Innocent tumours and cysts are comparatively rare.

Carcinoma

As carcinoma is not only the most common, but also the most serious form of tumour met with in the tongue, and as it is often difficult to diagnose it from certain of the lingual affections which have just been described, it is convenient to consider it first. The tumour is nearly always of the squamous-celled or epitheliomatous variety. In the latest edition of their work on *Diseases of the Tongue*, Butlin and Spencer state that "glandular carcinoma has occurred in the submaxillary gland, but from the mucous glands on the surface of the tongue no instance seems to have been seen." Since this work was published the present writer has reported, in conjunction with Dr. T. Shennan, a case in which the disease began in the mucous glands.

Epithelioma.—Statistics vary as to the relative frequency with which the sexes suffer, but they appear to indicate that about six males suffer to one female. So far as the writer's experience goes these figures exaggerate the frequency with which epithelioma of the tongue occurs in women. The age at which the disease appears is usually from forty to sixty, but it has been met with as early as twenty in patients of both sexes. In these young subjects the growth of the tumour is rapid, the glands are early and extensively invaded, and the tendency to recurrence after operation is great.

The actual *cause* of the disease is still uncertain, but there is good ground to believe that any long-continued irritation of the tongue, such as is produced, for example, by the rubbing of a jagged tooth or an ill-fitting tooth-plate, is a potent predisposing factor. The evil influence of tobacco smoke appears to have been exaggerated, but there is no doubt that the friction of the rough end of a clay pipe against the tongue is conducive to the development of cancer. Chronic superficial glossitis (leukoplakia), syphilitic fissures and scars, and chronic ulcers, are frequently present for some time before an epithelioma appears, and they seem to play some part in its causation. Butlin expresses himself strongly to the effect that the repeated application of strong caustics to chronic inflammatory conditions of the tongue is a most important, and by no means uncommon, determining cause of cancer.

As regards the *development* of the tumour, it may first appear in the form of an indolent excoriation, ulcer, or fissure, as a warty growth—particularly in association with a patch of leukoplakia—or less frequently as a distinct hard nodule beneath the surface of the tongue. In whichever of these ways the growth makes its appearance it passes through a stage—the so-called *precancerous stage*—in which inflammatory changes, such as infiltration with leucocytes, superficial ulceration, and certain changes in the epithelium of the nature of hypertrophy, or the formation of warty projections, alone are present. These conditions gradually give place to those characteristic of the epitheliomatous tumour, and ulceration soon begins. When the ulcer has formed, its base and the surrounding parts become characteristically hard and indurated, and the adjacent lymphatic glands soon become infected.

Clinical Features.—The tumour is usually situated on the anterior half of the tongue, and is more frequently on the edge than on the dorsum or under aspect. The clinical appearances vary widely. Sometimes the surface of the tumour is covered with a warty growth, sometimes it is excavated, forming a deep ulcer with raised nodular edges. In other cases the ulcer is smooth and its edges even and rounded. Extreme hardness of the edges and of the base of the ulcer is always a characteristic feature. Another characteristic feature is the fixation of the tongue, which prevents it being protruded beyond the teeth. This is especially marked when a growth starting on the edge of the tongue spreads towards the floor of the mouth and invades the lower jaw. There is constant gnawing pain in the tongue, and severe spasmodic pain shooting towards the ear and along the branches of the fifth nerve. In the more advanced stages of the disease there is profuse salivation, and the saliva may be tinged with blood. Owing to the fixation of the tongue the patient has difficulty in swallowing the saliva, and it continuously dribbles from the mouth. The speech is thick and indistinct, owing to the difficulty in moving the tongue. The breath is usually extremely fœtid.

Enlargement and hardness of the adjacent lymphatic glands are always early signs in cancer of the tongue. The particular group of glands first to become infected depends largely on the position of the primary growth. When the tumour is near the tip of the tongue, or in relation to the frenum and adjacent part of the

floor of the mouth, the submental glands are the first to become enlarged. Later the submaxillary group follows suit. When the tumour is further back on the tongue the submaxillary glands are first implicated, and later those along the line of the carotid artery and internal jugular vein. Owing to the close proximity of the sublingual and submaxillary salivary glands, and the fact that lymphatic glands frequently lie inside their capsules, these glands are often infected. The enlarged glands on the neck are very liable to become cystic, or to suppurate and burst to the surface, leaving unhealthy sinuses or malignant ulcers.

Metastasis to the liver, lungs, or other internal viscera may take place, but is rare. The patient usually dies in from twelve to eighteen months from repeated small hæmorrhages, or from marasmus, septic absorption, or a low form of pneumonia.

Glandular Carcinoma.—As the writer's case of carcinoma of the tongue originating in the mucous glands appears to be the only example hitherto recorded, it may be briefly described. The patient was a sailor, æt. 59, who, save for three attacks of yellow fever, had always enjoyed good health. He denied having had syphilis. About five years before coming under observation he noticed on the dorsum of his tongue a small white patch, which was hard, but not painful. For two years it slowly increased in size, and later began to cause pain. For a year before seeking advice he had had gradually increasing difficulty in moving his tongue in mastication, deglutition, and articulation, until eventually it became almost fixed. During the same period the pain had greatly increased, till it was constant and severe, shooting into the ears and through the head. He had lost a stone and a half in weight in about two years.

On examining the mouth it was found to be filled up almost completely by the tongue, which was thickly coated with a greyish brown fur. There was a large ulcerated area on the right side of the tongue covered by a grey slough. The jaws remained slightly apart, but were almost fixed, and the tongue could not be protruded even to the level of the teeth. Articulation was very indistinct, and he could swallow only with difficulty. There were several large, hard glands behind the angle of the jaw on the right side, and another in the line of the vessels. On the left side only one small gland could be detected.

The tongue was excised by Whitehead's method, but recurrence took place, and the patient died seven months after the removal of the tongue. On post-mortem examination it was found that there were numerous glandular swellings on both sides of the neck, some of which had undergone cystic degeneration. In addition metastatic tumours were found in the lungs, the suprarenal glands, and the pancreas.

Differential Diagnosis.—Cancer of the tongue has to be diagnosed from syphilitic and tuberculous affections, warty growths, and simple ulcers and fissures; and in connection with the differential diagnosis it has to be borne in mind that any of these conditions may take on malignant characters and develop into an epithelioma. In many cases it is impossible from the clinical evidence to arrive at a definite diagnosis, and it becomes necessary to remove a portion of the growth for microscopic examination. Should any doubt still remain, the condition should be treated as if it were malignant.

Treatment.—From the point of view of treatment cases of cancer of the tongue may be divided into two classes: (1) those in which there is a reasonable prospect that the disease may be completely eradicated by free removal of the primary growth and of the infected lymphatics, and (2) those in which the disease has extended so widely that complete removal cannot be hoped for.

In the first class the operation should always be recommended as the only line of treatment which offers any hope of permanent cure. The treatment of early cases by the repeated application of caustics cannot be too strongly condemned. Not only does it fail to arrest the growth of the tumour, but the irritation caused by the agent applied actually hastens its spread.

Even in cases where recurrence takes place the operation is usually justified, as it prolongs the patient's life by six or eight months, and by removing the foul ulcerated mass from the mouth adds greatly to his comfort. Further, the recurrence usually takes place in the glands, and is not attended with great suffering. Metastasis to distant organs is rare in cancer of the tongue, and the possibility of its occurrence seldom influences the question of operative interference.

In the second class—where there is little hope of being able to extirpate the disease—the surgeon often has great difficulty in deciding as to whether or not the attempt should be made. Speaking generally, it may be said that if the local disease in the mouth can be completely removed, this should be done, even if the affected glands cannot all be excised. The patient's suffering is almost entirely due to the ulcerated tumour in the mouth causing agonising pain, profuse salivation, and extreme fœtor of the breath, and even if the operation does nothing more than free him of this, and so render his last days bearable, it is amply justified.

If, however, the disease is situated near the base of the tongue, and has spread extensively in the floor of the mouth, on to the tonsils, the palate, or the lower jaw, or towards the opening of the larynx, and at the same time there is

widespread infection of glands, the operation is seldom advisable. Not only is it attended with serious immediate risk, as the patient is usually very much run down, but rapid local recurrence with persistence of the symptoms renders it futile.

In such cases *palliative treatment* alone is available. The mouth must be kept as sweet as possible by the frequent use of an antiseptic mouth wash, such as Condy's fluid, sanitas, izal, or peroxide of hydrogen. The pain may be relieved to some extent by local applications, such as cocaine or orthoform, but as a rule the free administration of morphia is called for. When feasible, division of the lingual nerve may be performed, but the benefit is seldom great. Hæmorrhage, if slight, may be arrested by local styptics, such as adrenaline or turpentine; if more copious, by ligature of the external carotid in the neck. Tracheotomy may be called for when the respiration is impeded. When the taking of food by the mouth causes much pain, recourse must be had to rectal feeding.

Operative Treatment.—Numerous operations have been devised for the removal of the tongue, and in selecting that most suitable to a given case, consideration must be had to the site and extent of the tumour, and the degree of implication of the lymphatic glands.

The chief *immediate risks* of the operation are shock, hæmorrhage, and interference with respiration by the entrance of blood into the air passages.

In patients who are not already exhausted by the disease, who are not suffering from albuminuria, or any other general affection, and who have not been addicted to alcohol, the shock is seldom severe. By adopting the usual measures it can be reduced to a minimum (*see* "SHOCK").

The prevention of hæmorrhage is one of the most important points in the operation. Some surgeons advocate a preliminary ligature of one or both lingual arteries in the neck as a routine measure. Others only carry this out when an incision has to be made in the neck in any case for the removal of infected glands. In a recent series of cases the writer has found the application of a *temporary ligature* to the external carotid, just below the origin of the lingual, of great service in diminishing the loss of blood. This measure is particularly useful in cases where extensive operations are called for, as the hæmorrhage from the facial and its branches, as well as from the lingual, is controlled. The carotid is exposed in the removal of the infected glands, and a broad catgut or kangaroo-tendon ligature applied as the first, that is the double, twist of a "surgeon's knot." As this loop is tightened the wall of the artery is infolded by the lateral pressure of the finger-nail, so that the lumen is occluded without risk of rupturing the tunica intima.

The subsequent removal of the ligature is facilitated by the simple manœuvre of including in its grasp a piece of stout silk laid parallel with the artery. The ends of the ligature and of the included piece of silk are brought out through the wound. After the operation on the tongue is completed the knot on the ligature is undone by pulling upon the two ends of the silk. This obviates the necessity of handling the wound in the neck after the fingers have been soiled in the mouth.

The risk of blood entering the air passages and causing asphyxia can usually be reduced to a minimum if the patient's head be so placed that the blood tends to flow out of the mouth, and if the back of the pharynx be frequently sponged during the operation. This sponging should be done very gently and no oftener than is necessary, as rough handling of the back of the pharynx appears to add to the shock.

When it is necessary to encroach on the base of the tongue, and when the tumour fills the mouth, it may be advisable to perform a preliminary laryngotomy or tracheotomy. The back of the pharynx can then be firmly plugged while the anæsthetic is administered through the tracheal tube.

The most important of the *remote risks* of the operation is septic infection. It is impossible to lay too much stress upon the importance of thoroughly purifying the mouth before the operation. For several days the mouth should be washed out every two or three hours with an antiseptic wash; the teeth should be cleansed and the ulcer on the tongue purified and dusted with iodoform or some other antiseptic powder. After the operation a special nurse should be detailed to attend to the purification of the mouth and the feeding of the patient. By constant douching and by careful feeding the mouth may be kept perfectly sweet, and the risk of septic complications reduced to a minimum.

Watson-Cheyne recommends the preliminary injection of antistreptococcus serum in cases where there is reason to anticipate septic complications.

Treatment of Infected Glands.—Until within recent years it was generally accepted that operative interference was contra-indicated when there was any considerable infection of the submaxillary and cervical glands, and particularly when the enlarged glands had become fixed. It has been shown, however, that much can be done in the way of removing these glands even when they have formed adhesions to the carotid sheath and its contents. As a rule, it is only the jugular vein which is seriously implicated in the adhesions, and a considerable portion of this vessel may with safety be removed along with the glands if necessary. Cheyne has deliberately removed a portion of the vagus nerve along with a cancer-

ous gland with no further ill effect than a temporary acceleration of the pulse. The writer has had a similar experience

It is only necessary to remove glands which are enlarged, and the attempt need not be made to clear out the lymphatic vessels between the primary tumour and the infected glands. When there are no glands palpably enlarged Cheyne recommends that the surgeon should rest content in the first instance with the removal of the primary disease in the mouth, and be prepared to deal with any glandular enlargement which may appear later. Butlin and Jacobson, on the other hand, urge the advisability of removing the glands in every case as a matter of routine. "The operation should be performed as soon as the patient has recovered sufficiently from the operation within the mouth to bear it" (Butlin)

Methods of Operating.—It is only necessary to describe three of the numerous methods of excising the tongue—Whitehead's, Syme's, and Kocher's.

Whitehead's Operation.—This method is specially useful in cases of cancer limited to one side of the anterior portion of the tongue, but it may be employed even when the whole tongue is implicated The mouth is opened widely with a gag, and a loop of stout silk is passed through the substance of the tongue on each side of the middle line, to enable the organ to be pulled well out of the mouth. This loop should be introduced as far back in the tongue as possible, so that, when traction is put upon it, the base of the tongue will be brought forward. The mucous membrane is divided with scissors along the middle line, first on the dorsal and then on the under aspect of the tongue, and the muscular substance is torn through The mucous membrane and muscles of the floor of the mouth are next snipped through with scissors, the vessels being caught with forceps as they are divided. The anterior pillar of the fauces is next divided, and the tongue can then be pulled well out of the mouth by means of the silk sutures passed through it. The operation is completed by cutting transversely across the affected half of the tongue well beyond the tumour The mucous membrane should first be divided with a knife or scissors and the muscular fibres then torn through The lingual artery can usually be recognised and clamped before being cut If it bleeds it is easily secured.

The operation for removal of the whole tongue is carried out on the same lines as for removal of one half, save that the organ is not split along the middle line

When the disease invades the floor of the mouth the sublingual gland should be removed along with the tongue.

In some cases there is difficulty in withdrawing the tongue from the mouth on account of its fixation in the region of the anterior pillar of the fauces, and in order to gain access it may be necessary to split the cheek from the angle of the mouth back to the anterior edge of the masseter. Langenbeck advised in addition division of the jaw just behind the molar teeth in such cases When the tumour is adherent to the jaw it is necessary to remove a portion of the bone

Syme's Operation.—This operation is suited to cases in which the whole tongue with the structures in the floor of the mouth must be removed. It consists in carrying an incision in the middle line through the lower lip, across the chin, and down to the hyoid bone. One of the central incisors is extracted and the lower jaw divided The writer has found Gigli's saw very useful for this purpose The tongue is then separated from the jaw by dividing the muscles inserted into the genial tubercles. The two halves of the jaw are then forcibly retracted in order to expose the floor of the mouth, and to give free access to the tongue. The further steps of the operation are similar to those of Whitehead's operation, the tongue being removed by cutting with scissors wide of the disease in every direction After removal of the diseased tissues the jaw is wired and the soft parts brought together with stitches The stump of the tongue must be secured with a strong silk suture to prevent it falling back and obstructing the air passages.

Kocher's operation is also suited to cases in which the whole tongue, right down to the hyoid bone, has to be removed A preliminary tracheotomy is performed A curved incision is made from just below the symphysis menti down to the hyoid bone, and then onwards along the line of the digastric muscle and anterior edge of the sterno-mastoid as far as the lobule of the ear. The integument is reflected up over the jaw, and the submaxillary and anterior triangles clear of lymphatic glands. The mylohyoid muscle and mucous membrane are then divided, and the cavity of the mouth thus opened The anterior pillars of the fauces are next divided through the mouth, and the tongue can then be pulled out through the wound in the neck The tongue and other diseased tissues in the floor of the mouth are cut out with scissors, the blood-vessels being secured as they are cut

After-Treatment.—After removal of the diseased parts the hæmorrhage is arrested, and the whole of the raw surface may be painted over with a solution of chloride of zinc, forty grains to the ounce. A few catgut stitches may be inserted into the mucous membrane, where possible, to diminish the extent of the raw surface Care must be taken not to attempt too much in this direction lest the discharge be retained and septic absorption result. It is best to trust to frequent douching and sponging with antiseptic lotions to keep the mouth dry

and sweet. For this purpose the writer has found nothing better than Condy's fluid. The patient should be fed as soon as possible after the operation. When there is pain or difficulty in swallowing rectal feeding may be employed for a day or two, but the sooner the patient gets food in the ordinary way the better. The food should be administered through a rubber tube attached to the spout of a feeding-cup, and passed well back into the pharynx. After each diet the mouth must be thoroughly douched to remove any fragments of putrescible material.

The purification of the mouth is greatly facilitated if the patient can sit up in bed. This posture is also advantageous in warding off septic lung complications, by preventing the discharges gravitating towards the air passages. As soon as the effects of the anæsthetic have passed off, therefore, he should be propped up in bed with pillows. The patient is usually able to be on the sofa in five or six days, and is practically well by the end of three weeks.

Sarcoma

The tongue is very seldom the seat of sarcoma. When this form of tumour occurs it is usually of the lympho-sarcomatous type, and corresponds to similar growths met with in the tonsils. The small, round-celled form also occurs. The tumour may begin either in the muscular substance of the tongue or in the submucous tissue. Both forms tend eventually to fungate through the mucous membrane. The diagnosis is always difficult, and is seldom certain before removal and examination of the tumour. In all suspicious cases full removal of the tumour and the portion of the tongue in which it grows is to be recommended. Some tumours show little tendency to recur after removal, while others are extremely malignant.

Innocent Tumours

Innocent tumours of the tongue are rare. The only forms that call for mention are lipoma, fibroma, angioma, and papilloma.

Lipomata usually occur in males beyond the age of sixty. They may be single or multiple, localised or diffuse. They are of slow growth, soft, or semifluctuating, and present the same lobulated character as do fatty tumours in other situations. In many cases a diagnosis can only be made after exposing the growth by an incision. The treatment consists in shelling out the tumour.

Fibromata are met with at all ages, but are most frequently seen in adults. They usually occur on the dorsum of the tongue, and are not unfrequently multiple. They may become pedunculated, particularly when they originate in the submucous tissue. As the growth is made up largely of cellular tissue, it is soft to the touch and simulates a lipoma, or even a cystic swelling. It only causes inconvenience by its size, filling the mouth and interfering with speaking and eating. The treatment consists in removing the growth.

Angiomata.—Several varieties of angiomata are met with on the tongue, the most common being the capillary and venous nævi. As a rule the tumour is small, affecting only a part of one side of the tongue and giving rise to no trouble. In rarer cases it implicates a considerable area of the tongue, and by its size interferes with speaking and eating. It is also liable to be injured by the teeth, and so give rise to hæmorrhage and septic complications.

Small growths which are causing no trouble may safely be left alone. If situated on the edge of the tongue, and liable to cause bleeding by being bitten, a small wedge-shaped portion of the tongue, including the nævus, should be excised. Extensive nævi may call for excision of the portion of the tongue implicated. As the operation is likely to be attended with copious bleeding, preliminary ligature of the linguals or temporary occlusion of the external carotids should be employed.

Papillomata, or warty growths, are not uncommon on the tongue. They are frequently associated with a patch of leukoplakia, and their chief importance lies in the fact that they are liable to become epitheliomatous. In all cases the growth should be removed by carrying an elliptic incision around its base. The wound on the tongue is closed with a few catgut stitches.

Among the rarest forms of tumour of the tongue may be mentioned fibromyoma, rhabdomyoma, osteoma, and chondroma.

Cysts

Dermoid cysts are occasionally met with underneath the tongue. They lie on the upper surface of the mylohyoid, between the genio-hyo-glossi muscles. Histologically they present the same characters as in other situations. They may be noticed soon after birth, or may only attract attention in adult life. The tumour usually forms a swelling under the chin, varying in size from a pigeon's to a turkey's egg. When it bulges towards the mouth it may simulate a retention cyst of the salivary glands, from which, however, it differs in that it is more deeply placed, pits on pressure, and presents a yellowish instead of a purplish appearance.

The *treatment* consists in excising the tumour, preferably from the outside.

Hydatid and *Cysticercus* cysts are very rarely met with in the tongue.

Thyro-glossal Cysts and Tumours

It is convenient here to refer to certain cysts and solid tumours which may develop in connection with the embryonic tract, which passes from the isthmus of the thyroid gland to the

foramen cæcum at the base of the tongue—the thyro-glossal tract. The solid tumours have the same structure as the thyroid gland, and they may grow to a considerable size. In some cases the thyroid tissue appears to be physiologically active, as the removal of the growth has been followed by symptoms of myxœdema. The cysts are characterised by being lined with ciliated epithelium, and having thyroid tissue in their walls. These tumours may be met with at any age; in some cases they are congenital. They are usually situated at the base of the tongue, but have been met with in the region of the hyoid bone. As seen from the mouth they are of a bluish brown or dark red colour. As the walls of the cysts are very vascular they are liable to bleed frequently. The blood may fill the cyst, causing it to enlarge suddenly, or may escape into the mouth. The frequent variations in size of the swelling and the repeated attacks of bleeding help to distinguish thyro-glossal cysts from other swellings at the base of the tongue. The growth may attain sufficient dimensions to interfere with speech or swallowing, in which case it should be removed. When not causing inconvenience it is best left alone.

Malformations of the Tongue

Complete or partial *absence* of the tongue, due to arrested development of the tuberculum impar, is extremely rare.

Occasionally the fore part of the tongue is *bifid*. The split may merely amount to a notch at the tip of the tongue, or it may extend for a considerable distance into the substance of the organ. This deformity may be associated with other congenital malformations of the face and jaws. The function of the organ is not seriously interfered with, and the operation of paring and uniting the edges of the cleft is only called for to remove the disfigurement.

Congenital tongue-tie or *ankyloglossia* is a condition in which the tip of the tongue is bound down to the floor of the mouth by an abnormally short and narrow frenum, or by folds of mucous membrane on each side of the frenum. This deformity is extremely rare. It is very common, however, for parents to blame a supposed "tongue-tie" when a child is slow to begin to speak, or when he speaks indistinctly or stammers, and surgeons are frequently requested to divide the frenum under such circumstances. In the vast majority of cases an examination shows that there is nothing wrong with the frenum, and that the child is not tongue-tied. This fact is emphasised because hæmorrhage, septic complications, deforming cicatricial contractions, and other untoward effects have followed the needless division of the frenum.

In the rare cases of true tongue-tie the tongue cannot be protruded between the teeth. The condition usually improves as the child grows older. If it persists and interferes with the functions of the tongue it is then necessary to divide the shortened structures close to the symphysis, and towards the floor of the mouth, and to prevent the cut surfaces from reuniting by frequently packing with gauze, or by gently stretching the parts with the tip of the finger.

Excessive length of the frenum is occasionally met with, and in young children may allow of the tongue falling back into the throat and causing sudden suffocative attacks, which may prove fatal — so-called "tongue-swallowing." In some cases the frenum is so long and slack that the patient is able voluntarily to fold the tongue back behind the soft palate.

Macroglossia is the name applied to a variety of conditions in which the tongue becomes unduly large. The typical form—*lymphangiomatous macroglossia*—is due to a dilatation of the lymphatic spaces of the tongue. It is often congenital, and may affect the whole or only a part of the tongue. The enlargement may be progressive from the first, or it may remain stationary for years, and then begin to increase somewhat suddenly—it may be after an injury or as the result of some inflammatory condition.

At first the tongue is slightly swollen and is very sensitive to contact with hot or irritating food. Blisters are liable to form on the surface and burst, giving exit to a clear serous fluid. Recurrent attacks of acute glossitis, accompanied by hæmorrhage into the substance of the tongue, and followed by a deposit of fibrous tissue, lead to permanent enlargement and hardening of the organ. The tongue may eventually become too large for the mouth and remain permanently protruded. The patient has then difficulty in taking food and in breathing. The protruded portion becomes dry and cracked, swallowing and speech are interfered with, and there is great pain and tenderness. The course of the disease is very chronic and may last for years. In long-standing cases the lower jaw may become altered in shape, and the lower lip everted and thickened.

The *treatment* consists in removing a wedge-shaped portion of the tongue, including as much as possible of the lymphangiomatous tissue. The operation should be done as soon as the condition is recognised. It is not attended with serious hæmorrhage.

In cretins, in certain imbeciles, and in the subjects of general hemihypertrophy, the tongue is frequently hypertrophied in whole or in part —*muscular macroglossia*.

Atrophy of the tongue is rare as a congenital condition. *Hemiatrophy* occurs in various diseases of the central nervous system, as well as after injuries and diseases implicating the hypoglossal nerve.

Nervous Affections of the Tongue

Neuralgia of the tongue is seldom confined to the distribution of the lingual nerve. As a rule the pain radiates along other branches of the trigeminal. The pain in the tongue is usually unilateral, and may be associated with the appearance of an herpetic eruption or of marked exfoliation of the epidermis. The condition usually yields to medical treatment, but in inveterate cases it is sometimes necessary to stretch or resect the nerve.

Butlin describes a condition closely allied to neuralgia, in which *pain in the region of the foliate papilla* is the most prominent symptom. The patient is usually a middle-aged woman, decidedly neurotic, and often with a gouty or rheumatic tendency. She complains of severe burning or aching pain in the region of the foliate papilla, which is situated on the edge of the tongue just in front of the fold of mucous membrane which passes from the anterior pillar of the fauces to the tongue. The pain is usually worst at night, and may last for months or even for years. It is seldom possible to discover any definite cause for the pain. The surgical importance of the condition lies in the fact that the foliate papilla is prominent and red, and is very liable to be mistaken on superficial examination for an early ulcerating cancer. An examination of the opposite side of the tongue, however, will reveal an exactly similar swelling which is not painful.

The first and most essential step in treatment is to assure the patient that the affection is not cancerous. If all doubt can be removed from her mind on this point, improvement usually takes place rapidly. Potassium bromide and anti-gouty and anti-rheumatic remedies may be prescribed, and if the pain is severe cocaine may be applied locally. Caustics and other irritating applications are to be sedulously avoided, as they are calculated to produce ulceration which may assume malignant characters.

Spasm of the tongue frequently occurs after injuries of the head implicating the centre or trunk of the hypoglossal nerve. It may also appear as a reflex condition in inflammatory affections of the teeth and gums, or as a manifestation of some general disease of the central nervous system.

Paralysis of the tongue—unilateral or bilateral—may be due to injury or disease of the nerve centres of the hypoglossal, less frequently to injury or pressure on the nerve trunk.

Tongue-Tie. *See* Tongue (*Malformations of*).

Tonic.—A medicine, regimen, or method of treatment which gives to the patient a feeling of greater health and robustness, which increases the *tone* of the whole body or of a special organ such as the stomach or nervous system; many tonics act by stimulating the digestion, others by improving the blood; *tonic* is also used as a term opposed to clonic, indicating continuous tension, *e.g.* of a muscle in a state of contraction, in tonic spasm. *See* Pharmacology; Physiology, Neuro-Muscular Apparatus (*Muscular Contractions*).

Tonka Bean.—The bean of *Dipterix odorata*, containing *Coumarin* ($C_9H_6O_2$), used as an antispasmodic in whooping cough (dose, 5 to 8 minims of the liquid extract).

Tonometer.—An instrument for measuring the tension of the eyeball. *See* Glaucoma (*Symptoms, Increased Tension*).

Tonsillitis.—Inflammation of the tonsils. *See* Tonsils, Diseases of (*Tonsillitis, Acute, Chronic*).

Tonsillolith.—A tonsillar concretion. *See* Tonsils, Diseases of (*Chronic Inflammation, Morbid Anatomy*).

Tonsillotome.—An instrument for removing the tonsil or for cutting off a part of it (*tonsillotomy*), a tonsilsector. *See* Tonsils, Diseases of (*Chronic Tonsillitis*); Nose, Post-Nasal Adenoid Growths (*Operative Treatment*).

Tonsils, Diseases of.

Introduction 42
Tonsillitis 43
Acute 43
Chronic—Enlarged Tonsils . . 45
Chronic Tonsillar Abscesses . . 47

See also Anæsthesia, Ether (*In Enlarged Tonsils*); Breath (*Offensive Odour*); Cautery (*Uses*); Diphtheria (*Diagnosis*); Galvanic Cautery (*Diseases, Hypertrophic Tonsils*); Larynx (*Hypertrophy of Lingual Tonsil*); Mumps (*Symptoms*); Nose, Post-Nasal Adenoids (*Treatment*); Oxygen (*Hydrogen Peroxide in Tonsillitis*); Palate (*Cleft Palate, Effects*); Pharynx, Examination of (*Tonsils*); Physiology, Food and Digestion (*Alimentary Canal, Pharynx and Tonsils*); Rheumatism in Children (*Symptoms, Tonsillitis*); Tongue (*Anatomy, Lingual Tonsil*); Undulant Fever (*Diagnosis, Tonsillitis*).

General Considerations. — Concerning the function of the tonsils we have little real knowledge. The free passage of leucocytes from their interior to the surface is an established fact (Stohr), and possibly such cells exercise a protective function by destroying septic organisms which gain access by the mouth. The frequent absence of tonsils both in children and adults shows that any protective powers which they may exert when present cannot be of great importance to the organism.

On the other hand their absorptive power, coupled with the peculiar structure of the gland,

often render these lymphoid masses a positive source of danger to the individual. Recent researches have amply proved that the tubercle bacillus may gain access to the cervical glands and the general system through the tonsils, while there is good ground for believing that the poison of rheumatic fever, scarlatina, and diphtheria may first invade the organism through the portals of the tonsil. Furthermore, it has been shown that septic infection, terminating in pulmonary gangrene, certain cases of endocarditis, acute phlegmon of the pharynx, septic thrombo-phlebitis, and acute suppurative osteomyelitis, may arise from a primary infection of the tonsils by pathogenic organisms.

Even in conditions of health the tonsillar crypts are often filled with evil-smelling contents consisting of disintegrating epithelium cells, fibrinous débris, and various septic organisms, amongst which staphylococci, streptococci, pneumococci, and leptothrix are abundant.

Under the combined influences of certain external causes and a lowered condition of the general health, such organisms may rapidly develop, causing a local inflammation accompanied by constitutional effects.

The part played by the "supratonsillar fossa" in many forms of recurrent tonsillar inflammation has been investigated by Paterson. This vestigial space of varying size and shape represents a portion of the second branchial cleft, and is situated between the anterior and the posterior faucial pillars, at the upper end of the tonsil. It communicates with the mouth by an orifice between the aforementioned pillars and also with the uppermost lacunæ of the tonsils. Like the latter it is often filled with septic matter which may find difficulty in discharging into the mouth, and lacunar or peritonsillar inflammation may result, or the retained material may calcify and ultimately form a tonsillar calculus. A strabismus hook will be found an excellent instrument for examining this cavity.

Tonsillitis or inflammation of the tonsils may be acute or chronic. Acute tonsillitis may be classified in the following manner:[1]—

I. Toxic tonsillitis, without any visible exudate.
  (*a*) Rheumatic.
  (*b*) Catarrhal or influenzal.
  (*c*) Tonsillitis occurring in infective diseases, *e.g.* scarlet fever, measles, syphilis, etc.
II. Infectious tonsillitis, with exudate.
  (*a*) Lacunar tonsillitis.
  (*b*) Diphtheritic.
III. Parenchymatous tonsillitis (quinsy), } both of which
IV. Peritonsillitis, } may end in suppuration.
V. Phlegmonous or gangrenous tonsillitis (in septic pharyngitis).
VI. Acute ulcerative tonsillitis.

[1] The above classification is modified from that of Sandner (*Medical Summary*, Dec. 1900).

Acute Tonsillitis

Etiology.—Lacunar, parenchymatous, and peritonsillar inflammations, which are the more frequent forms of tonsillitis, have so many features in common that they may be discussed together. Such inflammations most frequently occur during adolescence, and not rarely appear in epidemic form during the spring and fall of the year.

Whilst cold acts as an exciting cause of tonsillitis, the rheumatic diathesis has, during recent years, been almost universally regarded as the most important predisposing cause of the throat affection. The frequent association of the diseases in the same individual, their common etiological factors, and their amenability to the same remedies, lend support to this view. On the other hand the sudden onset, clinical course, infectious nature of and constitutional symptoms associated with acute tonsillitis, seem to point to a general toxæmia of which the tonsil is the source.

Other predisposing causes of acute tonsillitis are the presence of chronically enlarged tonsils, the gouty diathesis, and conditions of ill-health.

Acute lacunar tonsillitis is undoubtedly contagious, the infective material being conveyed by milk, water, and air. Defective sanitary conditions will often explain the frequent occurrence of tonsillitis in a household. In young children acute inflammation of the fauces should awake suspicions of the onset of one of the exanthems, while secondary syphilis must not be forgotten in the adult.

Traumatism, septic matter arising from carious teeth, and intranasal operations not aseptically performed, are local causes of acute tonsillitis.

Recurrent attacks are often due to the accumulation of septic matter in the supratonsillar fossa (*vide supra*), the outlet of which has been unduly narrowed by previous attacks of inflammation.

Morbid Anatomy and Pathology.—In the toxic forms of tonsillitis, *e.g.* rheumatism, influenza, and scarlatina, there is a general hyperæmia of the fauces in which the tonsils participate. In scarlatina the parts may be œdematous and of a livid red colour.

In lacunar tonsillitis, although the inflammation is mainly limited to the lacunæ, the tonsils are often much swollen and hyperæmic, while the crypts are filled with a yellowish creamy exudate composed of epithelial scales, leucocytes, fibrinous débris, and various septic organisms, amongst which staphylococci and streptococci usually predominate, while diplococci, tetracocci, and pneumococci are present in less numbers. Hence, while a mixed infection is the rule, yet

occasionally an acute tonsillitis may be due to a pure monococcal infection.

When streptococci predominate the constitutional symptoms are liable to be more severe. If the Klebs-Loeffler bacillus be present the case should be considered diphtheritic, even though clinically identical with lacunar tonsillitis.

Parenchymatous tonsillitis is characterised by inflammation of the lymphoid and deeper tissues of the gland; the latter is often much swollen, and eventually suppuration ("quinsy") may result.

Peritonsillitis signifies an inflammation which is supposed to commence in the connective tissue bed in which the tonsil lies. Probably the infection arises from one of the neighbouring lacunæ, or even more often from the supratonsillar fossa, in which region the peritonsillar abscess most frequently points.

In acute ulcerative tonsillitis a well-defined ulcer (or ulcers) with slightly raised edges of varying size, and covered by a greyish slough, occupies more or less of the surface of an uninflamed tonsil. Staphylococci, streptococci, and a rod-shaped bacillus staining with methyl-blue, have been found in connection with the ulcers.

Symptoms.—The onset of lacunar tonsillitis is usually ushered in by feelings of general malaise, sore throat, and fever. Sometimes the latter takes the form of a rigor. With the rapid increase of the inflammation, the quickened pulse, pyrexia varying from 101° to 103°, foul tongue, constipated bowels, febrile urine, and general prostration, all indicate the severity of the toxæmia, which may be further manifested by a fine erythematous rash which occasionally appears on the body. To the same cause must be attributed certain forms of endocarditis which may appear during an attack of tonsillitis not necessarily of rheumatic origin. Such general symptoms are more marked in lacunar than in peri- or parenchymatous tonsillitis, in which affections the pain in swallowing and other local symptoms are more pronounced. In these forms the dysphagia may be so severe that the patient, rather than swallow even his saliva, will allow it to dribble from the mouth.

When the tonsils are much swollen the voice is thick and guttural, and the breathing may be noisy, especially at night.

Deafness, earache, and tinnitus indicate an extension of the inflammation to the pharyngeal orifices of the Eustachian tubes and nasopharyngeal mucosa.

Examination of the throat in lacunar tonsillitis will reveal the crypts filled with a yellowish exudation which may often be wiped away without difficulty. Both tonsils and the surrounding faucial tissues may be much inflamed, although the inflammation may at first be limited to one gland.

In peritonsillitis and parenchymatous inflammation, the tonsil, with the neighbouring tissues and submaxillary glands, may be so inflamed that any movements of the head, especially opening the mouth, are very painful, and the general discomfort of the patient is greatly increased.

At first one tonsil is usually inflamed and much swollen, but not uncommonly the subsidence of the inflammation is followed by acute symptoms in the other gland.

In these forms of tonsillitis suppuration often occurs some four to five days from the onset of the disease, the abscess in peritonsillitis tending to point in the region of the supratonsillar fossa. In such a case the soft palate is much swollen above and in front of the tonsil, and should the pus not be evacuated by the surgeon, it will eventually burst into the mouth and thus afford rapid relief to all the symptoms.

In septic or "hospital sore throat" grey membranous patches may be found on the tonsils (or fauces), and in the former case are due to suppuration of the lymphoid follicles. Lacunar inflammation may be associated with these appearances. The general symptoms of malaise, pyrexia, etc., indicate the toxic nature of the disease.

Acute ulcerative tonsillitis is met with in weakly, anæmic individuals, and gives rise to few constitutional symptoms. The neighbouring lymphatic glands are not enlarged.

Prognosis.—This is almost invariably good. During the early days of tonsillitis in children the possibility of a developing exanthem must be borne in mind. Apart from the intensely septic and gangrenous forms which may be followed by visceral complications, tonsillitis is rarely a fatal disease.

Diagnosis.—Lacunar tonsillitis is the only form of inflammation which may give rise to difficulties of diagnosis, for in some cases it may be impossible from clinical appearances and symptoms to say whether the disease be diphtheria or lacunar tonsillitis. This will be especially the case when both maladies are prevalent in the some locality. For example, in a certain case of diphtheria the membrane may be limited to the tonsils and even to the orifices of the lacunæ, there may be no albumin in the urine, the general asthenia may not be marked, but in the course of a few weeks paralytic sequelæ develop, and meanwhile the possibility of disseminating the disease has been unrestrained. On the other hand, in a case of lacunar tonsillitis the exudate from one crypt may fuse with that of its neighbour, it may not be easy to remove it, albumin may be present in the urine, especially if the temperature be high, and the submaxillary glands may be enlarged, while the patient may be in a low, asthenic condition. Therefore in all doubtful cases the patient should be isolated while a bacteriological investigation is being made. If the Klebs-Loeffler bacillus be found the case

must be regarded as diphtheria and treated as such. If it be a case of lacunar tonsillitis the isolation will have minimised the risk of other members of the household becoming infected.

Acute ulcerative tonsillitis would be distinguished from a chancre of the tonsil by the combined absence of stony hardness of the tonsil and enlarged cervical glands; from tertiary ulceration by the history and effect of antisyphilitic treatment.

Treatment.—In all forms of tonsillitis it is well to commence treatment with an efficient aperient. There are few more suitable for this purpose than calomel followed by a saline draught. In lacunar tonsillitis with high temperature, salicylate of soda in twelve-grain doses every four to six hours, until its physiological effects are produced, will usually give good results in relieving pain and lowering temperature. It may be combined with a diaphoretic, *e.g.* acetate of ammonia. Fifteen grains of benzoate of soda every two hours is said to give excellent results after some six doses have been taken.

Drachm doses of the ammoniated tincture of guaiacum, given in hot milk every two hours until purging is produced, is especially useful in lacunar and parenchymatous tonsillitis. It may also be prescribed in five-grain doses of the powder.

Drop doses of aconite tincture given every hour is a favourite remedy for acute pharyngeal inflammations in children.

With regard to local remedies, considerable relief may be afforded in lacunar tonsillitis by spraying the inflamed tonsils with a hot alkaline solution flavoured with listerine; in other cases by painting the tonsil with equal parts of guaiacol and olive oil. Others recommend the sucking of ice-pills, or the frequent application by means of a brush of menthol dissolved in liquid paraffin (gr. xv. ad ʒj.). Swallowing of food is rendered easier by a preliminary painting of the tonsil with cocaine (gr. xv. ad ʒj.), while during the act of deglutition pain is greatly minimised by firm pressure externally just in front of the tragus of the ear. Gargles are generally useless because of the pain they cause, but sedative pastilles, *e.g.* borax and chlorate of potash, menthol, cocaine and borax, are sometimes useful. Sips of sweetened lemon-juice will enable the patient to cleanse the throat of the sticky exudation so abundant in many cases of tonsillitis. Hot external applications occasionally give relief, but a cold Leiter's coil applied early in the disease will do much to alleviate subsequent suffering by reducing the inflammation.

In parenchymatous or peritonsillar inflammation the tonsil should not be punctured unless it be fairly certain that pus is present, but when that is the case the operation should not be delayed for a moment.

The puncture should be made towards the upper and outer side of the tonsil over the region of the supratonsillar fossa. A partially sheathed bistoury should be passed in for at least half an inch, and then withdrawn downwards and inwards so as to enlarge the aperture of entry. If pus does not follow the withdrawal of the knife a pair of Lister's sinus forceps should be passed into the puncture and opened in one or two directions, for in this way the abscess may often be discovered and relieved.

After an acute attack of tonsillitis the patient should be put on a tonic plan of treatment, and should seek change in the country or seaside. When he has quite recovered his health the tonsils should be removed if they are enlarged.

An acutely inflamed tonsil should never be removed unless it is giving rise to serious difficulty of respiration.

I. Chronic Inflammation of the Tonsils—Enlarged Tonsils

Etiology. — While in many instances the morbid enlargement of the tonsils during the early years of life seems to occur without any definite reason, so that one is driven to ascribe such hypertrophy to some congenital influence, yet it is no less a well-established observation that in other cases the enlargement is due either to repeated attacks of inflammation in the tonsil, or dates from an attack of one of the acute specific diseases. In yet other instances it may be one of the numerous manifestations of the strumous diathesis. Enlarged tonsils usually make their appearance before the age of puberty and tend to shrink after that period of life has been passed.

Morbid Anatomy and Pathology. — Three varieties of chronically inflamed tonsils are most frequently met with.

1. Chronic lacunar tonsillitis, in which the tonsils are generally somewhat enlarged and in which the lacunæ are filled with yellow, evil-smelling, cheesy accumulations consisting of epithelium scales, the débris of inflammatory exudation, and a variety of septic organisms. Such accumulations may often be squeezed out of the lacunæ or from the supratonsillar fossa. In certain cases such caseous plugs may calcify and produce tonsillar calculi, or "tonsilloliths." In rarer instances the lacunar inflammation may lead to the production of epithelial outgrowths, or "papillomata" of the tonsil. Should the mouth of a lacuna become blocked, a chronic tonsillar abscess, or the formation of a cyst, may result.

The surface of a tonsil subject to chronic lacunar inflammation is often scarred and presents a trabeculated appearance; in long-standing cases of the disease the lymphoid tissue of the follicles may have almost entirely disappeared, having given place to epithelial proliferation of the lacunæ and general fibrotic overgrowth.

2. Fibrotic, in which the enlarged tonsil is mainly composed of fibrous overgrowth.

3. Lymphoid.—This is particularly common in children. In it there is excessive development of the lymphoid tissue, and such growths are frequently accompanied by adenoid vegetations of the nasopharynx.

Hypertrophied tonsils vary in size and shape. They may be so moderately enlarged that unless acutely inflamed they give rise to little trouble: on the other hand they may reach such a size as to almost meet in the middle line and cause great difficulty in breathing.

Sometimes they are more or less pedunculated, or while projecting very little from the fauces they may be enlarged from before backwards in an irregular lymphoid mass, which below passes imperceptibly into the lingual tonsil.

Symptoms.—In children respiratory difficulties and some alteration of the voice are common symptoms. The patient breathes noisily at night, and during the day is liable to choking attacks when taking food. The obstruction to free nasal respiration engenders mouth breathing, followed by a lack of development of the facial bones which is indicated by the long, narrow face and laterally contracted nostrils, together with the high arched palate, projecting incisor teeth, and retracted upper lip. The obstructed respiration also tends to the production of the pigeon-breast deformity, especially in rickety subjects. The voice is usually thick and guttural.

Temporary attacks of deafness or other ear symptoms are not uncommon, and in a small number of cases certain reflex phenomena, such as cough, vomiting, epigastric pains, etc., have immediately disappeared on removal of enlarged tonsils.

Enlarged cervical glands are frequently present, and no doubt are due to absorption of septic matter from the lacunæ and tonsillar surfaces (*vide supra* "General Considerations"). Probably the listlessness, mental apathy, and unhealthy aspect of many patients with enlarged tonsils, are in part due to absorption of septic matter into the system from the tonsils, as well as to imperfect oxygenation of the blood arising from deficient respiratory movements.

It must not be forgotten that many of the above symptoms are often aggravated or partly caused by the co-existence of adenoids or hypertrophic conditions of the nasal and naso-pharyngeal mucosa.

Diagnosis.—There can rarely be any difficulty in this, but it may be as well to point out that sudden unilateral enlargement of a tonsil in an adult should arouse suspicions of malignant disease.

Treatment.—If the tonsils be so enlarged that they produce any of the aforementioned symptoms, it is the duty of the surgeon to advise their removal, and to discountenance all ineffectual and temporising methods, such as the application of astringent paints, caustics, change of air, etc. This is the more imperative when we remember—

(*a*) That the operation of tonsillotomy is, with certain reservations, practically free from risk.

(*b*) That a great improvement in the general health almost always results.

(*c*) That evil results are almost sure to follow if enlarged tonsils be allowed to remain untreated.

Hypertrophied tonsils may be removed by the following methods:—

1. By means of the guillotine, *e.g.* Mackenzie's or Reiner's (*see* "Morcellement" and "Enucleation," p. 47).

2. By "morcellement," when the shape of the tonsils or other reasons render the first method impossible or inadvisable.

3. Enucleation for reasons similar to those last mentioned.

4. Galvanic cautery or "ignipuncture."

5. Wire ecraseur.

The removal of tonsils by means of a bistoury is only mentioned in order to condemn it as slow, painful, often incomplete, and more likely to be followed by hæmorrhage than any other method.

1. *Guillotine.* — Except in young, nervous children, tonsillotomy should be performed without a general anæsthetic. In other cases the painting of the tonsillar surfaces with a 10 per cent solution of cocaine is all that is necessary. When the guillotine is used it will greatly assist the surgeon if an assistant, standing behind the patient, pushes the tonsil inwards by external pressure applied behind the angle of the jaw. As a general rule, the slight hæmorrhage which follows the operation ceases spontaneously in the course of a few moments.

The patient should be kept quiet for twenty-four to forty-eight hours, and food should be cold and of a soft consistence.

In the case of young children in whom a general anæsthetic is advisable, the writer prefers that the head should hang backwards over the end of the table immediately the operation commences, and that if adenoids be present, their removal should immediately follow that of the tonsils.

Hæmorrhage after Tonsillotomy.—Apart from cases of hæmophilia, profuse hæmorrhage following tonsillotomy is uncommon, but the surgeon must always be prepared to meet it. It is more likely to occur in the fibrotic tonsils met with after twenty years of age than in young children, and its source is usually some branch of the ascending pharyngeal artery. In rare cases a rapid and fatal hæmorrhage has resulted from the wounding of an abnormally placed internal carotid artery. The hæmorrhage may immedi-

ately follow the tonsillotomy, or come on a few hours after the operation, especially under the influence of exertion or excitement.

Treatment of Hæmorrhage after Tonsillotomy.—If the hæmorrhage be only a free capillary oozing the patient should be induced to sit up and sip some iced water or suck pellets of ice. Gargling with hazeline and water (3 to 1) is often a useful remedy, as also is the application of styptic colloid to the bleeding surface.

A pad of cotton wool dipped in a creamy solution of tannic acid ʒiij., gallic acid ʒij., and water ℥j., and applied to the tonsil, will rarely fail to check hæmorrhage, especially if counter-pressure be at the same time applied externally and maintained for three to five minutes.

If the arterial jet can be seen, an endeavour should be made to seize the bleeding point by means of suitable forceps, and either twist or apply a ligature to it.

The hæmorrhage nearly always ceases if the patient becomes faint, and it would only be when every other method had failed that one would have to consider the advisability of tying the external carotid artery.

In all cases the patient should remain quiet for forty-eight hours after the operation, and only take cold, soft food.

2. "*Morcellement.*"—This method is suitable for long flat tonsils which cannot be made to project through the ring of a guillotine. By means of "punch-forceps," as introduced by the writer, such tonsils may be safely, quickly, and effectually removed at one sitting. The method is also convenient for removing tonsils in adults, or in any case where hæmorrhage may be feared.

3. "*Enucleation*" has been advised for large tonsils which on account of their shape are not suitable for removal by the guillotine. The anterior pillar is dissected off the upper part of the tonsil, and the index finger inserted above the gland, which is peeled downwards out of its bed. The method gives excellent results, but is not often necessary if "morcellement" be adopted. For enucleation a general anæsthesia will be necessary, a practical point in which it compares unfavourably with "morcellement."

4. *Galvano-puncture.*—This method has been advocated for the gradual destruction of tonsils in adults where tonsillotomy might be followed by hæmorrhage. At each sitting three or four punctures are made into the substance of the tonsil, and repeated in the course of a few days until the tonsil has shrunk sufficiently. The method is long, somewhat painful and tedious, and with "morcellement" to fall back upon the writer considers the use of the cautery rarely necessary.

5. *Wire Ecraseur.*—The wire loop has been used both hot and cold for removal of tonsils in adults, because of the freedom from hæmorrhage. The pain after removal is often exceedingly severe, especially when some minutes have been occupied in tightening up the snare. Otherwise the matter is safe and efficient.

Chronic Tonsillar Abscess

The etiology of the condition has already been referred to. The abscess may be situated in the tonsillar substance or in the supratonsillar fossa. In the latter case it often finds an exit into the mouth, and the patient complains of a frequently recurring unpleasant taste. In some cases it is possible to cause the pus to flow by pressing the tonsil from below upwards. Such abscesses produce aching pains in the side of the throat and various forms of throat discomfort. Sometimes the abscess contents caseate or calcify, and a tonsillolith or calculus results, which if hidden in the supratonsillar fossa may easily escape notice.

Such abscesses should be opened by guillotine, bistoury, or punch-forceps, and their cavities made to freely communicate with the mouth, when they will at once granulate up and evince no tendency to recur.

Tonus, Muscle.—The tense condition of resting muscle between its points of origin and insertion, due partly to passive elasticity and partly to a continuous contraction kept up by the action of the nervous system. *See* Paralysis (*Spastic Paralysis, Muscle Tonus*); Physiology, Tissues (*Muscle, Physical Characters*); Spasm (*Muscle Tonus*).

Tooth. *See* Teeth; Cocaine (*Uses in Toothache*, etc.).

Topæsthesia. — Local sensibility to touch (Gr. τόπος, a place; αἴσθησις, perception by the senses).

Topalgia.—Pain in a limited area which does not correspond to the distribution of any particular nerve.

Tophaceous.—Of the nature of *tophi*. *See* Tophus.

Tophus.—A "chalk stone," such as occurs in gout about the joints of the hands and feet and in the cartilages of the ear (Lat. *tophus* or *tofus*, tufa or stone); it consists chiefly of urate of sodium. *See* Gout (*Morbid Anatomy, Chalky Deposits*).

Topical.—Relating to a place, local (Gr. τόπος, a place or locality); so a topical remedy (*topicum*) is one whose action is local, and topical diagnosis is based on phenomena due, for instance, to causes acting on a limited area of the brain or spinal cord.

Topo. — In compound words *topo* (Gr. τόπος, a place) has the meaning of local or localised, e.g. *topoalgia* (localised pain), *topographic anatomy* (descriptive or surface anatomy), *topo-*

neurosis (a local neurosis), and *topophobia* (morbid fear of places).

Torcular Herophili.—The point of meeting (literally, the press or cellar of Herophilus) of the great venous sinuses on the inner aspect of the occipital bone.

Tormina.—Griping or colicky pains in the intestines, or in the uterus after delivery (*post-partum tormina*), or of the bladder in strangury (*tormina urinæ*).

Torpor.—Sluggishness, deficient sensation, mental lack of response; *torpefactio universalis* is torpidity of the whole body. *See* Insanity, Nature and Symptoms (*Melancholic Stupor*, etc.).

Torquay. *See* Therapeutics, Health Resorts (*English*).

Torricellian Vacuum. *See* Physiology, Blood and Lymph (*Collection of the Gases of the Blood*).

Torsion.—Twisting or rotation, *e.g.* the twisting of an artery to stop hæmorrhage, the twisted character of the umbilical cord, the occasional twisting of the pedicle of a tumour (such as an ovarian cyst or uterine fibroid) or of the spermatic cord. *See* Hæmorrhage (*Local Treatment*); Scrotum and Testicle, Diseases of (*Torsion or Axial Rotation of Spermatic Cord*); etc.

Torticollis. *See* Wry-Neck; Hysteria (*Contractures*); Rheumatism in Children (*Arthritis*).

Torula. A name given to different varieties of fungi (*blastomycetes*), some of which (*e.g.* the *Torula cerevisiæ*) cause alcoholic fermentation. *See* Micro-organisms (*Blastomycetes or Yeasts*).

Torus.—An elevation, prominence, or protuberance; also, the *tuber cinercum* of the brain.

Total Aphasia. *See* Aphasia (*Clinical Features, Total*).

Total Necrosis.—A phase in the structural degeneration of the red blood corpuscles occurring in normal blood as a result of long exposure to the air (three to four hours), and in pathological blood in which the globulicidal powers of the plasma are increased (*e.g.* severe anæmias); corrugations appear in the stroma of the erythrocyte, amœboid movements are seen, decoloration follows, the corpuscle becomes a poikilocyte, and finally fragmentation (schistocytosis) occurs.

Touch. *See* Brain, Physiology of (*Sensory Centres, Touch*); Children, Development of (*Special Senses, Sensibility to Touch*); Mind, Education of the (*Education of the Special Senses*); Physiology, Cerebrum (*Touch Centre*).

Touch Corpuscles. *See* Physiology, Neuro-Muscular Mechanism (*Tactile Sense, Corpuscles*); Skin, Anatomy and Physiology (*Nerves, Touch, Cells of Merkel*, etc.).

Tourette's Disease. *See* Gilles de la Tourette's Disease.

Tourniquet.—An instrument for arresting hæmorrhage by compressing the whole limb or one particular artery in it; it is usually a screw and a pad held in position by a circular strap, but a handkerchief and a piece of wood or an indiarubber band (Esmarch's tourniquet) may be employed in emergencies. *See* Hæmorrhage (*Local Treatment*).

Tow.—Flax or hemp refuse; used to absorb discharges, as in cancer cases, *e.g.* cancer of the cervix uteri; oakum or marine lint.

Toxæmia.—Blood poisoning or the presence of toxic substances in the blood. *See* Headache (*Causes, Toxæmia*); Heart, Myocardium and Endocardium (*General Pathology, Exciting Causes, Chemical*); Pregnancy, Physiology (*General Changes, Toxæmia of Pregnancy*); Scurvy in Adults (*Etiology*).

Toxalbumins.—Poisonous proteid substances due to chemical changes in micro-organisms, and causing some at least of the phenomena of diseases ascribed to microbes. *See* Immunity (*Toxic Filtrates of Bacterial Cultures*).

Toxalbumoses. *See* Toxicology (*Animal Foods causing Poisoning*).

Toxic.—Belonging to or due to poisons, *e.g.* toxic amblyopia in which tobacco is the poison. *See* Angina Pectoris (*Varieties, Toxic*); Hæmoglobinuria (*Toxic*); Retina and Optic Nerve (*Retinitis of Toxic Origin*); Senile Insanity (*Pathogenesis*).

Toxicity.—The toxic quality of anything, *e.g.* urine, blood, etc., poisonousness.

Toxicogenic.—Poison-producing. *See* Micro-organisms (*Toxicogenic Bacteria*).

Toxicology.

Definition 49
Diagnosis of Poisoning . . . 50
Symptoms of Corrosive and Irritant Poisoning 50
General Treatment 51
Post-Mortem Appearances . . . 51
Inorganic Poisons 52

A. *Corrosives*

1. *Mineral Acids—Sulphuric Acid, Nitric Acid, Hydrochloric Acid, Acetic and Tartaric Acid, Oxalic Acid, Potassium Binoxalate, Carbolic Acid* . . 52
2. *Alkalies—Potash, Soda, Ammonia* . 54

B. *Irritants*

Potassium Nitrate 54
Potassium Chlorate 54
Barium Chloride 55
Magnesium Sulphate 55
Arsenic 55
Acute 55
Chronic 56
Tests 56
Antimony 58
Antimony Chloride 58
Tests 58
Mercury 58
Acute 58
Chronic 59
Lead 59
Acute 59
Chronic 59
Copper 61
Acute 61
Chronic 61
Silver 61
Acute 61
Chronic 62
Zinc 62
Tin 62
Bismuth 62
Iron 62
Chromium 62

C. *Non-Metallic Elements*

Phosphorus—Acute and Chronic . . 62
Iodine, Iodoform, Potassium Iodide . . 63
Bromine 63
Chlorine 63
Boric Acid 63

D. *Gaseous Poisons*

Sulphuretted Hydrogen 63
Carbon Dioxide 63
Carbon Monoxide—Acute and Chronic . 64

E. *Cyanogen Compounds*

Hydrocyanic Acid 65
Potassium Cyanide 65
Oil of Bitter Almonds 65

Organic Poisons

Alcohol 65
Chloral Hydrate 66
Chloroform 66
Sulphonal 66
Carbon Bisulphide—Acute and Chronic . 67
Petroleum and Paraffin Oil . . . 67
Benzene, Nitro-Benzene 67
Aniline and other Coal-Tar Derivatives . 67
Creasote 68

Alkaloids and Vegetable Poisons

Strychnine 68
Nux Vomica 69
Cocculus Indicus 69
Opium and Morphine 69
Tests 70
Belladonna 71
Henbane and Stramonium . . . 71
Indian Hemp 71
Gelsemium 71
Cocaine—Acute and Chronic . . . 72
Tobacco—Acute and Chronic . . . 72
Spotted Hemlock 72
Foxglove—Digitalis 73
Colchicum 73
Hellebore—Veratrine 73
Monkshood—Aconitine 74
Pilocarpine 74
Physostigmine or Eserine . . . 74
Santonin 74

Abortifacients . . 74

Food-Stuffs, Poisoning by

Vegetable 75
Fungi, Lathyrism, etc. . . . 76
Animal Foods— 77
Meat, etc. 77

See also under the various poisons (*e.g.* ANTIMONY, ARSENIC, etc.); ABORTION; MEDICINE, FORENSIC (*Abortion*); MORPHINOMANIA AND ALLIED DRUG HABITS; PHARMACOLOGY; PRESCRIBING; and TRADES, DANGEROUS.

TOXICOLOGY, the science which treats of poisons, comprises a description of the nature and constitution of poisons, the symptoms and post-mortem appearances produced by them, the treatment of poisoning, and the physiological and chemical tests by means of which poisons may be recognised. To the medical practitioner the most important of these are the symptoms, treatment, and post-mortem appearances; consequently, in this epitome they will receive the chief consideration.

It is difficult to accurately define the word poison, inasmuch as almost any substance, that is not absolutely inert, may produce injurious effects if taken in excessive amount, when it would come under the definition of a poison; yet such substances are included in the preparation of ordinary food, as, for example, nutmeg. A practical definition of a poison may be thus formulated: a poison is a substance which, either by being absorbed into the living organism, or by its chemical action on the tissues, injures health and destroys life. Among the conditions which have to be taken into account when estimating the injurious effects which may be produced by poisons are: age, idiosyncrasy, habit, and state of health, together with the physical condition and the mode of administration of the poison. The susceptibility of infants and children to opium and the exceptional intolerance of adults towards certain drugs—such as arsenic, quinine, or mercury—are well known. The habitual use of opium produces an unwonted tolerance towards it, as does also the presence of diseases such as delirium tremens and tetanus. The physical condition and the

modes of administration of poisons tend to modify their action. Arsenic in the gaseous form, as arsenietted hydrogen, is much more virulent than ordinary white arsenic. Strychnine injected subcutaneously acts more rapidly than when taken by the mouth. When a poison is swallowed its action tends to be materially modified by the presence or absence of food in the stomach: if the stomach is empty, a corrosive poison expends its full power on the coats of the viscus; whilst if it contains a mass of solid food some of the corrosive action of the poison is expended on the food. Absorption takes place more slowly in the full than in the empty stomach and intestines; hence the action of a fatal dose of strychnine may, for this reason, be delayed or accelerated.

The Diagnosis of Poisoning.—An important indication is afforded by the sudden occurrence of acute symptoms in a person previously in his usual health. It is to be remembered, however, that a pathological condition may declare itself abruptly and give rise to the suspicion of poisoning: rupture of a previously unsuspected gastric ulcer, or immediate intestinal obstruction, are diseases of this type. Again, it is to be noted that the early symptoms produced by many poisons may simulate those of disease: arsenic produces symptoms which have been mistaken for those of cholera or of gastro-intestinal catarrh. Another circumstance indicative of poisoning is the occurrence of unusual symptoms directly after eating and drinking. Here, again, care must be exercised not to mistake the effects due to changes in the food itself, whereby toxic products have been formed, for those of an added poison. Even wholesome food, which is difficult of digestion when eaten hastily by a person who has fasted longer than usual, may give rise to vomiting and pain which closely resemble the symptoms produced by an irritant poison. If, after partaking of a common meal, a number of people are simultaneously attacked with peculiar symptoms, there is ground for suspecting the presence of poison in the food, which may be due either to noxious substances which have been incorporated with it, or to the food itself being tainted.

When a medical man is in attendance on a case and suspects poisoning, he should note in writing all that he observes, and should take possession of any suspicious substances in order that they may be chemically examined. In addition, specimens of urine, fæces, and vomit should be submitted to analysis. In cases of doubt the patient may be safeguarded by securing the presence of trained nurses, day and night, with instructions to prepare all food, to administer it and the medicine with their own hands, and not to leave the patient when on duty.

Symptoms of Corrosive and Irritant Poisoning.—A *corrosive poison* is one that destroys tissue by direct chemical action; when administered in a dilute form a corrosive may act as an irritant. In the act of swallowing a corrosive poison an *immediate* and violent pain is produced, which extends from the mouth down the œsophagus to the stomach, from whence it radiates over the abdomen. Uncontrollable retching and vomiting occur within a few minutes, the vomited matter consisting, at first, of the contents of the stomach, and subsequently of shreds of mucous membrane, coagulated mucus, and blood, the colour of the blood being often altered by the chemical action of the poison. There is intense thirst, with difficulty or impossibility of swallowing, each attempt to swallow causing increased vomiting. The patient is in a condition of extreme collapse; the surface is pale, cold, and clammy; the features are pinched, and the eyes present a terrified look. The voice is hoarse, almost inaudible; the mouth is filled with shreds of membrane and ropy mucus; the lips are swollen, and, along with the chin, probably show signs of the local action of the poison. The mouth and tongue exhibit indications of corrosion, the colour of the surface varying with the nature of the corrosion. The abdomen is usually distended. The breathing is laboured and noisy; attempts to clear the air-passages give rise to a distressing, hoarse, futile cough. The pulse is thread-like, of low tension, and often very rapid. The bowels are confined. The urine is diminished in amount, partly owing to the rejection of fluid by the persistent vomiting, and partly to the extremely low arterial tension. The mind usually remains clear to the end; death—which in some instances is preceded by convulsions—takes place in a condition of extreme collapse within twenty-four to thirty-six hours.

An *irritant poison* is one which by its specific action sets up inflammation in the digestive tract. A pure irritant does not produce corrosion, though some substances classed as irritants may act as corrosives. When a substance that acts solely as an irritant is swallowed, the symptoms do not come on in the act of swallowing, nor immediately after, as is the case with corrosives; an interval of from half an hour to an hour or more elapses. Taking a metallic irritant as a type, there may or may not be an astringent metallic taste perceived when the poison is swallowed. After a short interval a hot, burning sensation, with a feeling of constriction, is felt in the throat; the burning sensation is felt in the stomach, and it rapidly develops into severe pain, which spreads over the abdomen. Violent and persistent vomiting and purging occur, the discharged matter often becoming blood-stained. There is intense thirst, and attempts to allay it provoke further vomiting. The purging is accompanied by excessive tenesmus; the motions may be blood-stained, or they may be like rice-water. Collapse sets

in; the surface is cold and clammy, the pulse feeble and irregular, there is great restlessness and anxiety, the mind often remaining clear to the end. Cramps in the legs not unfrequently occur, and there may be convulsions. In fatal cases death from exhaustion usually takes place in from one to four days.

THE GENERAL TREATMENT OF POISONING.—The indications are: to remove or neutralise any poison that may be in the digestive tract, to counteract the effects of that which has been absorbed, and to promote its elimination; to keep the patient alive until the effects of the poison have passed off, and to alleviate pain and other general symptoms.

Emetics.—Half-drachm doses of zinc sulphate, dissolved in a few ounces of warm water, repeated if necessary, act quickly without causing depression; or a dessert-spoonful of mustard may be given in a tumblerful of warm water. A teaspoonful of ipecacuanha wine is a good emetic for children. In addition to the emetic, the patient should be made to drink copiously of warm water in order to aid the vomiting and to wash out the stomach. In some cases it is preferable to give a hypodermic injection of one-tenth of a grain (ten minims of the B.P. injection) of apomorphine hydrochlorate. In the absence of an emetic the fauces may be tickled with a feather, or even with the finger, and copious draughts of warm water given.

The *stomach-pump* or tube is the most efficacious means of emptying the stomach. When using the stomach-pump a hard, rigid tube is to be avoided lest it should injure the œsophageal walls. The tube should be passed down the œsophagus, keeping it well against the posterior wall; some water is always to be injected into the stomach before any fluid is extracted, the object being to wash out the stomach without entirely emptying it, which might cause injury to its coats. Five or six feet of small india-rubber tubing, one end of which has been slipped over a funnel, is a good substitute for the pumps, and indeed is sometimes preferable. The free end is passed down the œsophagus, and then a pint or more of warm water is poured into the funnel, which is now held vertically over the patient's mouth as high as the tubing permits. When the funnel is nearly empty, the tube close to it is pinched tight with the finger and thumb, and the funnel inverted, is depressed until it is lower than the stomach; on removing the finger and thumb the tube acts as a syphon and empties the stomach. As with the stomach-pump, the alternate introduction and withdrawal of warm water is to be persevered in until the washings are free from odour and consist of clear water. If the stomach contains much lumpy, solid matter, an emetic may be necessary before using the pump or tube, in order to avoid clogging. Neither the stomach-pump, nor the tube, nor emetics are to be used in cases of corrosive poisoning; the proper treatment in such cases is to neutralise the poison. Carbolic acid constitutes an exception to this rule. The stomach-pump or tube is especially useful in cases of poisoning by opium, chloral hydrate, alcohol, the vegetable and most of the mineral irritants, phosphorus and the alkaloids. In strychnine poisoning it will be necessary to administer chloroform before the tube can be passed. After evacuating the stomach a purgative should be given unless spontaneous purging has occurred.

Antidotes are remedies which counteract the effects of poisons; their action is either mechanical, chemical, or physiological. Flour mixed with water acts mechanically when given in poisoning by cantharides, magnesia acts chemically in the case of the mineral acids, and atropine is regarded as a physiological antidote to morphine.

Elimination of the poison which has been absorbed may be promoted by purges, diuretics, and special remedies in the case of central poisons. The means to be taken to keep the patient alive until the effects of the poison have passed off are determined by the nature of the poison; artificial respiration in hydrocyanic acid and severe opium poisoning, external warmth in poisoning by chloral hydrate and carbolic acid, and the administration of stimulants in many forms of poisoning, are illustrations. In addition, excessive pain, useless vomiting and purging, with other general symptoms, are to be controlled by appropriate means.

POST-MORTEM APPEARANCES PRODUCED BY POISONS

The most characteristic appearances are produced by corrosives, irritants, and blood poisons. Many of the more important alkaloidal poisons do not produce characteristic appearances. The indications afforded by corrosives and irritants comprise hyperæmia, softening and ulceration of the mucous membrane, with perforation of the wall of some portion of the digestive canal. Hyperæmia is a common effect of an irritant poison; it is usually most marked in the stomach, and may be either diffuse or in patches; it is most commonly seen at the cardiac end, less frequently at the pyloric end. The surface of the injected mucous membrane is often covered with viscid mucus, which may be blood-stained. Softening of the mucous membrane of the stomach is chiefly caused by corrosives, and is due to immediate chemical action. It is not a common condition, but is almost invariable in poisoning by the alkalies. Ulceration of the mucous membrane of the stomach occasionally results from irritant poisoning, due to intense local action, or to the occurrence of infarcts. Ulceration must be distinguished from the removal of patches of mucous membrane by the action of corrosives; the former is a pathological

process set up by the poison, the latter is purely chemical. Perforation caused by poisoning is usually due to the action of a corrosive, and the mode of its production leaves characteristic appearances. There is no indication of limitation by inflammatory processes, the margins of the opening, instead of being thickened, are partially disintegrated, and the aperture itself is often large and of irregular outline; if caused by sulphuric acid, the contiguous parts are charred and blackened. As a result of irritant poisoning, perforation is of exceptional occurrence, and the appearances would more nearly resemble those due to an ordinary gastric ulcer which had given way.

Some of the above-described appearances are very similar to those due to disease; others are only met with as the result of poisoning. Acute inflammation of the mucous membrane of the stomach is exceedingly rare, except as the result of irritant poisoning. The colour of the inner surface of the stomach is not to be relied on as an indication of inflammation; unnatural redness may be caused by food or medicine which possesses pigmentary properties, and during the process of digestion the gastric mucous membrane is reddened. Post-mortem staining produces redness, which, however, is limited to the posterior part of the stomach, and, moreover, the appearance is different from that due to vital processes; there is no glairy mucus on the surface of the membrane, which itself is not thickened. The early stage of putrefaction leads to softening of the whole thickness of the stomach wall; whereas softening due to inflammation is usually limited to the mucous membrane, which may be detached in patches. Ulcer of the stomach is much more frequently due to pathological processes than to poisoning. When an ulcer is caused by an irritant poison, it more nearly resembles an erosin, with inflamed margin, and without the thickening and matting together of the tissues which are characteristic of the more slowly formed idiopathic ulcer. Post-mortem perforation of the stomach may take place owing to the solvent action of the gastric juice; but the margins of the irregular aperture are not coloured as by the action of a corrosive, and the surrounding mucous membrane is usually swollen and gelatinous.

Inorganic Poisons—Corrosives

The Mineral Acids.—Of the mineral acids, sulphuric acid is by far the most energetic corrosive; it chars and destroys the tissue with which it comes in contact, and enters into chemical combination with albumin. Nitric acid ranks second as a tissue destroyer. Hydrochloric acid acts much less energetically.

The *symptoms* of poisoning by the mineral acids are those previously described (p. 50) as due to corrosives. The fumes given off by nitric and hydrochloric acids, especially the latter, are prone to attack the larynx and air-passages, and thus give rise to additional symptoms.

The *treatment* consists in neutralising the acid as speedily as possible. Calcined magnesia is the best antidote; but, since saving of time is all-important, any alkaline substance at hand should be made use of. In ordinary households the substance that is most likely to be available is whiting, which is impure calcium carbonate; failing this, whitewash may be scraped off the wall or ceiling. Any of these substances should be powdered, and administered suspended in water. White of egg, or soap and water, or even water itself as a diluent, should be given if nothing better can be obtained. The stomach-pump must not be used. As soon as the poison is neutralised, pain should be assuaged by hypodermic injections of morphine.

Sulphuric Acid.—*Fatal Dose.*—Half a teaspoonful caused the death of a child one year old. One fluid drachm is the smallest amount that has been fatal to an adult. Death has occurred within an hour; it usually takes place within thirty hours. Recovery has ensued after one ounce of the strong acid was swallowed.

Nitric Acid.—The smallest recorded fatal dose is two drachms. Recovery has occurred after half an ounce. Death has occurred in less than two hours; from twelve to twenty-four hours is the average fatal period.

The *fumes* from nitric acid, which has been accidentally spilt, have on several occasions caused death. Usually the dangerous symptoms, which resemble those of capillary bronchitis, do not come on until a few hours after the vapour has been inhaled.

Hydrochloric Acid.—The smallest fatal dose is one teaspoonful. Recovery has followed two ounces. Death has occurred in two hours, the usual period being from eighteen to thirty hours.

Post-mortem Appearances.—Externally there may be stains on the lips and chin produced by direct contact of the poison. The clothing should be examined for indications of the action of the acid and for the presence of early vomit, and any stained parts should be cut out and examined chemically. The mucous membrane of the mouth will probably be disorganised and detached in parts. The most profound changes will be found in the stomach, and, to a lesser extent, in the duodenum. When strong *sulphuric acid* is the poison the inner surface of the stomach is usually darkened in colour, not unfrequently being quite black where the acid has acted most powerfully. Where not entirely disorganised, indications of intense inflammation are seen. Of all poisons sulphuric acid most frequently causes perforation of the stomach, and when this is the case the contiguous viscera may be corroded on their peritoneal surfaces by the acid which has escaped through the perfora-

tion; the colon has thus been perforated from without inwards.

When *nitric acid* is the poison the corrosive effects are rather less pronounced, and the tissues tend to show a yellowish or brownish coloration. Perforation of the stomach is less frequent than with sulphuric acid.

When *hydrochloric acid* is the poison the gastric mucous membrane is ashy grey in colour and is probably eroded; it may display indications of acute gastritis with blackened patches. Perforation is exceptional.

Tests.—Sulphuric acid gives a white precipitate with a solution of barium chloride, which is insoluble in hydrochloric acid. If some of the precipitate is mixed with an equal bulk of sodium bicarbonate, and fused in the reducing flame of the blowpipe, it is converted into a sulphide. A fragment of this placed on a clean silver coin and moistened with water produces a brown or black stain of silver sulphide.

Nitric Acid.—Free nitric acid develops a bright red when brought into contact with a crystal of brucine. If the acid is combined a drop of strong sulphuric acid will liberate it, and then the brucine reaction may be obtained.

Hydrochloric Acid.—A solution of silver nitrate gives a white curdy precipitate which is soluble in ammonia, but not in nitric acid.

Oxalic Acid not only acts as a corrosive, but it also exercises a special influence on the nervous system and on the heart.

Symptoms.—When swallowed in concentrated solution the local effects resemble those produced by the mineral acids; if the solution is less concentrated the local symptoms are less severe, and the specific action of the poison may be more obvious. The nerve symptoms are very irregular; they consist of paræsthesia, or anæsthesia of the trunk or limbs, aching and shooting pains in the loins, numbness of the tips of the fingers, tenderness of the muscles of the calves, and convulsions. Sometimes oxalic acid has a narcotic action.

Treatment.—Chalk, or whiting, suspended in a small quantity of water or milk neutralises the acid with the formation of inert salts. The alkalies and their carbonates should not be given, as the resulting salts are soluble and poisonous. All antidotes should be given in as small amount of water as will serve to suspend them, in order to limit diffusion and absorption of the poison. After the poison has been neutralised the bowels should be emptied by enemata or aperients.

Fatal Dose.—One drachm caused the death of a boy aged sixteen. Recovery has followed one ounce and a quarter. Death has occurred in ten minutes; it usually takes place within two hours, but it may be delayed for several days.

Post-mortem Appearances.—If a strong solution of oxalic acid has been swallowed the usual indications produced by corrosives are met with; the stomach may be either reddened, or it may be black, as is the case with sulphuric acid. The coats of the stomach may be softened, and, exceptionally, perforation occurs. A white zone of crystals of calcium oxalate may sometimes be seen between the cortical and the medullary portions of the kidneys.

Tests.—A solution of calcium chloride added to a solution of oxalic acid produces a white precipitate of calcium oxalate, which is insoluble in acetic acid, but is soluble in hydrochloric acid. The reaction is aided by neutralising the solution of oxalic acid before adding the calcium chloride. A solution of silver nitrate gives a white precipitate which is soluble in nitric acid and in ammonia.

Potassium Binoxalate, or salt of sorrel, or salt of lemons, is nearly if not quite as poisonous as oxalic acid, and produces similar symptoms.

Acetic and Tartaric Acids act as corrosives, or as irritants, according to the strength of solution in which they are swallowed.

Treatment.—Neutralisation with magnesia and the alkalies, with subsequent administration of morphine.

Carbolic Acid.—Although designated as an acid, phenol, or carbolic acid, has no acid reaction, but it coagulates albumin and destroys tissue, and therefore is regarded as a corrosive poison. Like oxalic acid its action is both local and remote; locally it is a corrosive, and remotely it exercises a complex influence on the nervous system.

Symptoms.—The chief difference in the action of carbolic acid as compared with other corrosives is that vomiting is not only commonly absent, but that it is actually difficult to procure; this is due to the anæsthetic action of the poison on the gastric mucous membrane. The urine is usually dark in colour, or becomes so on exposure to the air, due to the presence of an oxidation product of phenol—hydroquinone. The body temperature is low from the first, and death is threatened from cardiac and respiratory failure.

Treatment.—As already stated, carbolic acid constitutes an exception to the rule that the stomach-tube should not be used in corrosive poisoning. A soft tube should be used, and the greatest care should be exercised in passing it in order to avoid perforating the œsophagus. The stomach should be well washed out with lukewarm water, preferably with some magnesium sulphate or saccharated lime dissolved, so as to produce an innocuous combination with the phenol. White of egg and milk may be given. The application of warmth externally by means of hot bottles and flannels is of the greatest importance. Stimulants should be administered, and if failure of the respiration seems imminent, artificial respiration should be performed.

Fatal Dose.—One drachm has proved fatal, and recovery has occurred after one ounce. Recovery after much larger amounts has been recorded, after six ounces for example, but in such cases a crude form of carbolic acid was taken, containing only about 14 per cent of phenol. Sometimes death occurs very rapidly; in one case, in three minutes; the usual period is from three to four hours. Exceptionally a case may not end fatally for forty-eight or more hours. The external application of carbolic acid, and its injection into abscess cavities, have respectively caused death.

Post-mortem Appearances.—The odour of the poison will be perceptible in the mouth and also on opening the stomach. The mucous membrane acted on by the poison is either of a white or ash-grey colour in the mouth, and usually brown in the stomach and duodenum, where it is often corrugated and toughened, or it may be softened. Erosion is uncommon.

Tests.—Bromine water gives a yellowish white precipitate of tri-bromophenol. A few drops of ammonia water and a small quantity of bleaching-powder are added to an aqueous solution of carbolic acid, and the mixture is gently warmed; a blue colour is produced, which becomes red on acidulation. The well-known odour of carbolic acid constitutes a most delicate test.

The Alkalies: Potash, Soda, Ammonia

Symptoms.—When a strong solution of one of the alkalies is swallowed the usual symptoms of corrosive poisoning occur, with the addition that purging is not infrequent. When ammonia is the poison its vapour tends to attack the larnyx and thus to lead to suffocation. Death from broncho-pneumonia has been caused by the inhalation of gaseous ammonia. Recovery may ensue in ammonia poisoning, even when death appears imminent. When recovery from the immediate effects of any of the alkalies takes place, stricture of the lower end of the œsophagus is a common sequence.

Treatment.—Vegetable acids in the form of dilute vinegar or lemon juice should be given, with olive oil, demulcents, and opium. The stomach-tube must not be used. When ammonia is the poison a steam-tent, and possibly tracheotomy, will be needed.

Fatal Dose.—Potash and Soda.—Death has followed forty grains. An average fatal dose would probably be three or four drachms. Death has occurred within a few hours; more frequently it results, after weeks or months, from secondary effects.

Ammonia.—Two drachms have proved fatal, and recovery has taken place after one ounce. Death has occurred in a few minutes, the usual period being twenty-four to forty-eight hours. Life may be prolonged for months and even years, death taking place from œsophageal stricture.

Post-mortem Appearances.—Those of corrosives. The mucous membrane of the stomach is softened; it may be bright red or dark brown; it may also be eroded. Perforation is unusual. In ammonia poisoning the laryngeal mucous membrane is infiltrated and thickened, and is sometimes covered with an exudation, forming a kind of false membrane. The irritating effects of gaseous ammonia may penetrate as far as the smaller bronchi, in which tubular casts have been found. The kidneys may be inflamed.

Tests.—For *potash* and *ammonia*. A solution of platinic chloride gives a yellowish-white crystalline precipitate. A saturated solution of tartaric acid gives a white crystalline precipitate. *Soda* may be identified by exclusion: if the suspected substance has a strong alkaline reaction, and does not respond to the above-named tests, nor to those which demonstrate the presence of an alkaline earth, it may be assumed that soda is present.

Irritants

Potassium Nitrate or saltpetre in large doses produces violent pain in the stomach and abdomen, with vomiting and purging. Nerve-symptoms, such as unconsciousness and convulsions, pain in the loins, cramps in the legs, muscular twitchings, abnormalities of sensation, paralysis, and aphonia, have been observed. Death is usually preceded by coma; it may occur suddenly from heart failure.

Treatment.—Wash out the stomach with the tube, and give ice and opium, and, if necessary, stimulants. Warmth and the recumbent posture must be maintained.

Fatal Dose.—The smallest recorded is two drachms. Recovery has followed one ounce. Death has occurred in from five to sixty hours.

Potassium Chlorate acts both as an irritant and as a blood poison. It causes pain in the stomach and bowels with vomiting. The patient becomes delirious and somnolent; the blood is brownish in colour and viscid, due to destruction of the red corpuscles and the transformation of some of the liberated hæmoglobin into methæmoglobin. The white corpuscles are increased in number. The skin may be jaundiced and the liver and spleen enlarged. The urine may contain albumin, hæmoglobin, or hæmatin.

Treatment.—Wash out the stomach, and administer diuretics and vapour baths, along with general symptomatic treatment.

Fatal Dose.—Six and a half drachms have caused death. The fatal period ranges from six hours to as many days.

Post-mortem Appearances.—The mucous membrane of the stomach may be softened and may exhibit ecchymoses. The blood is dark brown in colour, is inspissated, and yields the spectrum of methæmoglobin; in it are many free granules of colouring matter. The kidney tubules are filled with the débris of red blood

corpuscles. The spleen and liver may be enlarged.

BARIUM CHLORIDE.—This salt acts locally as an irritant and centrally as a nerve poison.

Symptoms. — Shortly after the poison is swallowed the usual symptoms of an irritant poison manifest themselves. In addition, the action of the heart becomes feeble and irregular, and pain may be felt in the cardiac region. The breathing is slow and laboured; the air-passages may become filled with mucus, causing dyspnœa and cyanosis. Singing in the ears, diplopia, pains in the limbs, coma, and in some instances paralysis, afford further evidence of the action of the poison on the nervous system.

Treatment.—The stomach-pump or an emetic, unless vomiting has already occurred. Sodium, or magnesium sulphate, should be given in half-ounce doses. Hypodermic injections of morphine and external warmth are useful.

Fatal Dose. — One teaspoonful has caused death. The fatal period has extended from one hour to seven days.

Post-mortem Appearances.—The usual diffusely inflamed appearance due to irritant poisoning, with spots of ecchymoses, are usually found in the stomach.

Tests.—By dipping a clean platinum wire into a solution containing a salt of barium, and then placing it in the flame of a Bunsen burner, a green colour is imparted to the flame. Dilute sulphuric acid, or a solution of an alkaline sulphate, produces a white precipitate when added to a solution of a barium salt—the precipitate being insoluble in nitric acid.

The salts of STRONTIUM are not poisonous.

MAGNESIUM SULPHATE, or Epsom salts, taken in large doses, has caused death. In one case four, in another, two ounces, and in a third case one ounce, respectively caused death. Vomiting and purging may be absent.

ARSENIC.— All combinations of arsenic are poisonous. The form in which it is most frequently used as a poison is arsenious oxide, commonly called white arsenic. When fresh it is glassy-looking and has a smooth, vitreous fracture; after being kept some time it becomes opaque and porcelain-like. When powdered it looks like flour. It has little taste, and no colour nor odour—properties which singularly fit it for homicidal purposes. Arsenious oxide is but slightly soluble in water; cold water dissolves about half a grain to a grain per ounce. Boiling water dissolves much more, the greater part, however, being deposited on cooling. When powdered arsenious oxide is added to water or liquid food, the finer particles float like a white scum on the surface, and cannot be got rid of by stirring; this appearance is very significant. White arsenic cannot legally be sold in quantities of less than ten pounds unless it is mixed with soot or indigo. When arsenious oxide is combined with potash or soda it is much more soluble. Some kinds of "fly-papers" are saturated with sodium or potassium arsenite and then dried; by soaking such papers in water a strong solution containing arsenic may be obtained, a fact that has been utilised for criminal purposes. In a higher degree of oxidation, as arsenic acid, arsenic is used in the manufacture of aniline colours, and also in combination with sodium as a fly-poison. Copper arsenite, or Scheele's green, is a pigment in common use, and occasionally gives rise to chronic arsenical poisoning by being detached in a state of minute division from wall-papers, fabrics, or woodwork which have been coloured with it. Arsenietted hydrogen is an extremely potent blood poison, which from time to time causes fatal poisoning to workmen who inhale the gas. Some organic compounds of arsenic are extremely poisonous, others are but feebly so. The combination of cacodylic acid belong to the latter group, and on this account have been used medicinally, especially sodium cacodylate, of which six to ten grains (equal to about four to six grains of arsenious oxide) have been administered daily for weeks without ill effects. On the other hand symptoms of arsenical poisoning have been caused by very much smaller doses.

Symptoms of Acute Arsenical Poisoning.—In from half an hour to an hour after a poisonous dose of arsenious oxide has been swallowed, a sensation of heat, which rapidly develops into a violent burning pain, is felt in the throat and stomach; then follow nausea and uncontrollable vomiting. Violent purging sets in, and, after the normal contents of the bowels have been evacuated, the motions tend to assume a rice-water appearance, and they may contain blood. The purging is accompanied by distressing tenesmus and by a burning sensation in the rectum. There is extreme thirst, any attempt to relieve it being immediately followed by rejection of the swallowed fluid. Indications of collapse now set in; the face assumes a very anxious appearance, the features are sunken, the surface is cold, moist, and cyanosed, the pulse is small and thready, respiration is laboured, and the voice is hoarse. The tongue, at first thickly coated with white fur, often becomes red at the tip and round the edges; sometimes it is unnaturally red all over. Owing to the vomiting and the low arterial tension the urine is very scanty. Cramps in the calves add to the patient's distress. Consciousness may be maintained to the end, or death may be preceded by coma, in which condition clonic or tonic spasms may occur.

In exceptional instances the symptoms vary from the above description. The interval between the swallowing of the poison and the commencement of the symptoms may be much shorter, only ten minutes, or it may be prolonged to twelve or eighteen hours, the condition

of the stomach as to food being the principal determining factor. Occasionally there is extreme collapse from the first, the gastro-intestinal symptoms being relatively insignificant; in such cases death may occur in from six to twenty-four hours. Repeated injurious doses of arsenic are frequently given in cases of criminal poisoning, the symptoms being more or less modified in consequence. As the symptoms produced by a dose of the poison are passing off a second dose is given, which may be followed by others; the result is that certain symptoms which are more commonly met with in chronic arsenical poisoning are superadded: itching of the margins of the eyelids and reddening of the conjunctivæ, with hyperæmia of the mucous membrane of the fauces, causing the patient to have a sensation as of a hair in the throat, which he tries to dislodge by constant hawking. Numbness and tingling in the fingers and toes, tenderness of the muscles of the legs, and paralysis of the feet, due to multiple neuritis, may severally occur, and, on more than one occasion, have followed a single large dose of arsenic. Death has resulted from the introduction of arsenic into the vagina, and also from the application of arsenical paste to morbid growths.

Treatment.—The stomach should be washed out with the tube, and freshly precipitated ferric oxide administered. The oxide is prepared on the spot by adding some solution of ammonia or of potassium carbonate to the tincture of perchloride of iron; the ferric oxide which is thrown down is strained off, and then given suspended in a little water. Demulcents and morphine, with the application of external warmth, should afterwards be resorted to.

Fatal Dose.—Two grains of arsenious oxide have caused death. Recovery has taken place after a teaspoonful of powdered arsenious oxide. Death usually occurs in from twelve to forty-eight hours. Death has occurred in twenty minutes, and, not unfrequently, in from two to three hours. On the other hand, the fatal period has been prolonged to the sixth, eighth, and even the sixteenth day. In these instances there were remissions in the course of the symptoms.

Post-mortem Appearances.—Externally there is nothing of significance. Internally the stomach presents signs of intense inflammation, the mucous membrane having a velvety appearance; on the general red surface darker dots or streaks are usually seen, due to small hæmorrhages; occasionally there are more extensive submucous hæmorrhages. The mucous membrane is sometimes softened and easily detached; less frequently it is eroded, and very exceptionally the stomach wall has been perforated. In fatal poisoning, it is to be noted that by whatever channel the arsenic has obtained entry into the body—by application to a malignant ulcer, for example—the stomach will be found inflamed after death; arsenic is partly eliminated through the stomach irrespective of the mode of administration. The duodenum also is usually inflamed; and occasionally the lower part of the intestinal canal, especially the rectum, shows signs of inflammation. When life has been prolonged beyond the usual period, and sometimes when not so prolonged, the liver and kidneys show distinct appearances of fatty changes.

Chronic Arsenical Poisoning.—When the ingestion of small doses of arsenic is spread over a prolonged period, the symptoms produced are different from those due to acute poisoning. Arsenic is well-nigh ubiquitous, and may be unconsciously received into the system in a variety of ways; from the inhalation of particles given off by arsenical wall-papers, green baize, painted woodwork, and toys, and from its presence in minute quantities in many substances which are taken with food. All foodstuffs into the preparation of which sulphuric acid enters are liable to be contaminated with arsenic. A very extensive outbreak of chronic arsenical poisoning occurred in 1900 in Manchester and the neighbourhood due, as shown by Reynolds, to the introduction of arsenic into beer, through the unsuspected contamination of some of the ingredients manufactured with the aid of sulphuric acid used in brewing the beer. In certain trades chronic arsenical poisoning is liable to occur, as in furriers who dust the skins of the animals from which furs are obtained with various substances that act as preservatives, arsenic entering into the composition of some of these powders.

Symptoms.—The early indications consist of disorders of the digestive system, along with headache, alternate constipation and diarrhœa, with loss of appetite. Then follow irritation of the eyelids with suffusion of the eyes; laryngeal irritation; a red, moist, and painful condition of the soles of the feet and the palms of the hands; various skin eruptions—erythematous, urticarial, herpetic, bullous, or eczematous; disorders of sensation in hands and feet, with excessive tenderness of the muscles of the legs, followed by more or less paralysis (multiple neuritis), with high-stepping or tabetic gait; keratosis of the hands and feet, especially the latter; pigmentation varying from freckle-like spots to complete bronzing of the surface. There is great tendency to heart-failure, and in fatal cases death is usually due to this cause. When recovery takes place it is very slow.

The *elimination* of arsenic takes place primarily by the kidneys and bowels, and secondarily by the skin and its appendages. In the ordinary sense of the word, arsenic is not a cumulative poison, and when a single poisonous but not fatal dose is swallowed, elimination commences forthwith, and no evidence of the poison can be obtained from the urine later than ten or twelve

days. On the other hand, when successive small but not minute doses are taken over a long period, as in chronic poisoning, the elimination-rate does not appear able to keep pace with the rate of ingestion, and consequently a certain proportion of arsenic is backed up in the tissues, and shows itself in the urine for, possibly, several weeks after the last reception of the poison; I have found it fifty-nine days after the final dose. After death, arsenic, which has been absorbed, is found in the largest amount in the liver; the kidneys, spleen, brain, and other soft organs contain much less. In chronic poisoning arsenic may be found in the horny scales formed by the keratosis, in the nails, and in the hair; it is also found in the bones. When a considerable quantity of arsenic remains in the tissues at the time of death putrefaction is delayed; in acute poisoning after a minimum lethal dose putrefaction would probably follow the ordinary course.

Tests.—When arsenic is in solution Reinsch's method is a good way of demonstrating its presence; this method can be used when arsenic is in organic admixture. The first and absolutely indispensable step is to prove that the reagents about to be used are free from arsenic. Into a clean flask pour three drachms of pure hydrochloric acid and two ounces of distilled water; drop in a piece of bright copper foil about three-eighths of an inch square, and allow the acidulated water to gently boil on a gauze-covered tripod over a Bunsen flame for half an hour. Empty the fluid in such a way as to allow the copper to remain in the flask; if the copper is bright and its colour is unchanged the absence of arsenic may be assumed; if the copper is discoloured the reagents must be rejected as being contaminated. Should the copper be unchanged it is left in the flask, and some of the fluid which is suspected to contain arsenic is added, along with one-sixth its volume of hydrochloric acid from the same bottle from which the first supply was obtained, and the flask is replaced over the flame. The length of time that the fluid should be allowed to boil depends upon the amount of arsenic present; if there is a considerable amount a few minutes will suffice; but if very little be present, half an hour or even an hour will be required to obtain a deposit. On removing the flask from the tripod the liquid is poured out, the copper being left as before, and the flask is partly filled with water and gently shaken so as to wash the foil; this may be repeated once or twice. The copper, purple-tinted or possibly steel-grey, is then shaken out of the flask on to a piece of filter paper, by means of which it is dried. It is then cut in half with a pair of scissors, and the two pieces are introduced into a clean, dry sublimation tube. The tube is rapidly passed over a Bunsen flame to warm it, and the lower end of the tube is then brought to the margin of the flame and held there for a few seconds until the copper changes colour, showing that the film of arsenic has been volatilised. When the arsenic leaves the copper it combines with oxygen and deposits on the walls of the tube, a short distance above the foil, in the form of tetrahedral crystals of arsenious oxide, which present a very characteristic appearance under the microscope. If the crystals are dissolved, with the aid of heat, in a drop of water, and when cold a drop of a solution of ammonia-nitrate of silver be added, a yellow precipitate of silver arsenite is formed. In addition to arsenic, antimony, mercury, silver, bismuth, and tin are deposited on copper when boiled with it in acid solution; of these only antimony and mercury yield sublimates. With solutions containing organic matter the copper may be stained although no arsenic is present; the sublimation test, therefore, must never be omitted. When urine is submitted to Reinsch's test it is preferable to previously evaporate it down to one-fourth its volume. Solid organic substances should be pulpified and mixed with a sufficiency of distilled water as to render them fluid.

Marsh's Test.—A flask, furnished with a stopper through which a long thistle-funnel and an exit-tube pass, is charged with some pure zinc and dilute sulphuric acid. The exit-tube is fitted with a wider tube, which is filled with fragments of calcium chloride in order to dry the gas which passes through it; a length of combustion tubing drawn out small at its free end is attached to the drying tube by means of a short piece of rubber tubing. When the hydrogen, which is evolved by the action of the sulphuric acid on the zinc, has replaced the air in the flask, a Bunsen flame is allowed to play on the combustion tube a little before the narrow part is reached. If, after the flame has heated the combustion tube for half an hour, no stain is visible in the narrow part, the acid and zinc may be regarded as being free from arsenic. The suspected fluid is now introduced into the flask through the thistle-funnel when, if the arsenic is present, a stain will begin to show itself where the tube is contracted. The length of time required for the stain to develop, and the density of the resulting deposit, depend upon the amount of arsenic that is present in the flask: if there is a considerable amount the deposit begins to appear in a minute or less; if there is only little more than a trace, fifteen or twenty minutes elapse before it shows itself, in which case the experiment must be continued for an hour at the least. The deposit in the tube, if of medium density, is brownish and of a metallic lustre; when of greater density it may be black. The tube is now detached from the flask and the stained portion is heated in the Bunsen flame; the film readily volatilises, and deposits on a

cooler part of the tube in the form of crystals of arsenious oxide, which may be tested with ammonia-nitrate of silver as previously described. A less delicate method of demonstrating the presence of arsenic in the gas coming from the flask is to ignite it as it issues from the end of the combustion tube, and to allow the flame to play on a cold porcelain surface such as a crucible lid; the dissociated arsenic deposits on the porcelain as a brownish, glistening film which is freely soluble in a solution of bleaching powder, and but only slightly so in ammonium sulphide. Arsenic that is in combination with organic matter requires to be separated from it before it is added to a Marsh-apparatus, as almost all organic fluids lead to the formation of froth on the surface of the contents of the flask which mounts up to the exit-tube and spoils the results.

ANTIMONY.—The preparation of antimony that is most commonly encountered as a poison is the potassio-tartrate, or tartar-emetic. This salt is very soluble in water.

Symptoms.—Immediately after a poisonous dose of tartar-emetic is swallowed, an astringent, metallic taste is experienced, followed in a few minutes by a hot, burning pain from the mouth down to the stomach, along with a sensation of constriction in the throat. Violent vomiting then occurs, and shortly after diarrhœa. Exceptionally, blood may be present in the vomit. Cases have occurred in which vomiting was delayed for an hour after the poison was swallowed. The depressant effects of tartar-emetic soon show themselves: the pulse is small, frequent, and of low tension, the surface is cold and clammy, and may be cyanosed; the breathing is slow and laboured, and the patient is profoundly collapsed, and is probably unconscious. The urine is almost entirely suppressed. Clonic spasms may precede death.

Treatment.—The poison usually causes vomiting; if not, the tube may be used to wash out the stomach; or, probably, it will suffice to tickle the throat with a feather in order to induce emesis. Tannin, or some substance containing it, such as stewed tea, should then be given so as to form an insoluble compound with any of the poison that remains in the stomach. Unnecessary vomiting should be checked by ice and morphine. External warmth and stimulants will be needed.

Fatal Dose.—Five to ten grains would probably be a fatal dose. Death has been caused by a grain and a half following a like dose taken twenty-four hours previously. Recovery has followed two hundred grains. Death may occur in from a few hours to several days. After a large dose, either a speedily fatal issue from exhaustion may be expected, or an almost equally speedy recovery.

ANTIMONY CHLORIDE, or butter of antimony, acts as a corrosive.

Post-mortem Appearances.—After a fatal dose of tartar-emetic the mucous membrane of the stomach may be inflamed and ecchymosed; it is covered with slimy mucus and is sometimes eroded. The duodenum may participate in these appearances. In some instances the gastric mucous membrane is not inflamed, but presents a pale-yellowish appearance. The liver and kidneys may show fatty changes.

The appearances produced by antimony chloride are those of a corrosive.

Tests.—Reinsch's method, as described under "Arsenic," yields an amorphous deposit in the sublimation tube, which is readily distinguishable from the crystalline sublimate of arsenic. If the antimonial sublimate is dissolved in a drop of a solution of tartaric acid, and then treated with a drop or two of sulphuretted hydrogen water, an orange-coloured precipitate of antimonious sulphide is produced. Marsh's method (*see* "Arsenic") may also be used. The deposit in the tube occurs nearer to the Bunsen flame, often on both sides of it. The deposit is dead black (not brown as with arsenic), or, if there be much antimony present, it may have a metallic lustre like mercury. On applying heat the deposit is volatilised with difficulty, and if the tube be filled with air, it comes down on a cooler part of the tube as a white, non-crystalline, amorphous cloud, which may be dissolved in a solution of tartaric acid and tested with sulphuretted hydrogen water, as with the sublimate obtained by Reinsch's test.

MERCURY. — The chief poisonous salt of mercury is the mercuric chloride; occasionally the red and the white precipitates give rise to poisoning.

Symptoms of Acute Mercurial Poisoning.—Immediately after a poisonous dose of the perchloride has been swallowed, an acrid metallic taste, accompanied by a sensation of constriction in the throat, is experienced. A burning sensation quickly develops, which spreads from the mouth, down the œsophagus, to the stomach, and then radiates over the abdomen. Vomiting of white slimy masses, frequently tinged with blood, quickly follows. Diarrhœa, with severe tenesmus and colicky pain in the abdomen, then occurs, the motions being watery, often blood-stained, and mixed with shreds of mucous membrane. The mucous membrane of the mouth and pharynx is white and swollen; the voice is hoarse, and the breathing difficult and noisy. The patient is profoundly collapsed; the surface is cold, clammy, and cyanosed; the pulse is small and irregular; the urine is suppressed, any that may come away contains albumin. There may be convulsions. If the patient survives, salivation will probably set in twenty-four or more hours after the poison was swallowed.

Strong solutions of corrosive sublimate ap-

plied to ulcered surfaces have caused death. Intra-uterine injections of a solution of 1 in 2000 of corrosive sublimate have also caused death. Absorption of a solution of corrosive sublimate may take place through the unbroken skin, and accidents occasionally occur from the extensive use of this salt as an antiseptic.

The symptoms produced by red and white precipitates are those of an irritant; the toxic action of these salts is much milder than that of corrosive sublimate.

Treatment.—In corrosive sublimate poisoning, if vomiting has not occurred, white of egg should be administered, followed by an emetic; the former to combine with the poison and render it inert, and the latter to dislodge the compound, as it is soluble in excess of albumin. Demulcents, opium, and stimulants will then be required.

Fatal Dose.—Three to five grains of corrosive sublimate may be regarded as a fatal dose for an adult; recovery has followed three hundred and seventy grains. When death occurs it is usually within three or four days; it has occurred in a few hours, and has been delayed for seven or eight days. Red precipitate has been recovered from after a dose of a teaspoonful and more. Thirty-five grains of white precipitate have caused death. Recovery has followed two drachms.

Post-mortem Appearances.—After a fatal dose of corrosive sublimate the mucous membrane of the mouth and œsophagus is swollen, softened, and ash-grey or white in colour. The mucous membrane of the stomach is also swollen and softened, and may be deeply injected and ecchymosed; eschars may be present; sometimes there is little or no appearance of inflammation. The small intestines are less affected than the colon and rectum, which are often deeply injected and may be ulcerated. Interstitial nephritis is usually found to have occurred, and there may be a deposit of calcareous salts in the cortex of the kidney.

Chronic mercurial poisoning is almost exclusively due to the use of mercury, or its salts, in certain trades, such as looking-glass silverers, thermometer and barometer makers, and workers in quicksilver mines, and manufactories in which preparations of mercury are made; furriers and bronzers also use mercurial salts, and are liable to suffer from chronic poisoning. The symptoms comprise loss of appetite, of flesh, and of strength, with colicky pains, stomatitis, salivation, and tenderness of the gums, with extreme fœtor of the breath. Anæmia, occasional vomiting, and diarrhœa may occur, and eczematous or other eruptions of the skin. Later a fine tremor develops in the muscles of the tongue and face, which tends to spread to the arms and legs. As a rule more or less paralysis occurs, and there may be disturbance of the mind.

Tests.—With mercuric salts potassium iodide gives a scarlet precipitate soluble in excess. Stannous chloride gives a white precipitate which changes to grey. With soluble mercurous salts potassium iodide gives a green precipitate.

LEAD.—The acetate, the carbonate, and the chromate are the salts chiefly met with in acute poisoning by lead.

Symptoms.—The salts of lead act as mild irritants. If an ounce of the acetate be swallowed a metallic taste is at once perceived, followed by the usual burning sensation, which is common to metallic irritants, in the gullet and down to the stomach. In about half an hour, vomiting of white, opaque masses, which may be tinged with blood, occurs. There is thirst and colicky pains in the abdomen, which come on in paroxysms, and which are accompanied by rigidity of the abdominal muscles; the patient attempts to obtain ease by bending forward and compressing the abdomen. Usually the bowels are constipated, occasionally they are relaxed, the motions being black from the presence of lead sulphide. The urine is partially suppressed. There is great prostration, vertigo, pains in head and limbs, numbness, and paræsthesiæ, cramps in the calves, and occasionally paralysis. The tongue is coated, and the breath is offensive. The pulse is small and frequent. The blue line on the margins of the gums, which is characteristic of chronic lead poisoning, is rarely present. Most cases of acute lead poisoning recover.

Treatment.—The stomach should be washed out unless there has been free vomiting. Sodium and magnesium sulphates should be given in half-ounce doses, dissolved in half-pints of water; the resulting lead sulphate should then be got rid of by purgation. Demulcents and opium will be required.

Fatal Dose.—The fatal dose of lead acetate is unknown; recovery has followed one ounce. The *post-mortem appearances* are those of a mild irritant.

Chronic Lead Poisoning or Plumbism.—The extensive use of lead in trade pursuits, and the many ways in which beverages and articles of food may, accidentally, become contaminated with it, together with the characteristic cumulative properties it possesses, all tend to make chronic lead poisoning a much too common occurrence. Of trade pursuits in which lead is used there is no end; beginning with the extraction and reduction of the ore, an endless sequence is encountered of manufactures in which lead in one form or other is used. Workers in the manufacture of lead salts, especially of white and red lead, painters, plumbers, gas-fitters, file-cutters, printers, dippers, and other workers in potteries, glass-cutters, coach-makers, workers in enamelled sheet-iron ware, in the construction of electrical accumulators, in the preparation of white

vulcanised rubber, solderers, type-founders, and lead pipe-makers, comprise only a selection of the artificers who handle lead. Apart from trade risks there are countless channels by which lead may find its way into the system; snuff, tobacco, and certain kinds of sweets are frequently enveloped in so-called tin-foil, into the composition of which lead largely enters; beer is often drawn from the cask through leaden pipes; tinned fruits may be contaminated with lead which the acid of the fruit dissolves out of the solder; hair dyes are frequently composed of solutions of lead salts; the glaze of many earthenware vessels, and that of so-called enamelled iron-ware, readily yields lead to any acid substances, such as rhubarb, that are stewed in them. The most prolific source of dietetic lead poisoning is drinking-water. Notwithstanding the well-nigh universal knowledge of the risk which attends the introduction of lead into the system, the use of lead cisterns and pipes for the storage and conveyance of drinking-water is still common. New lead pipes are more dangerous than old ones, which gradually become protected with an insoluble coating of basic lead carbonate. The presence of carbonic acid, chlorides, nitrates, and nitrites in the water increases its solvent action on lead. Peaty water, and water containing other organic matter, is especially prone to dissolve lead; the action of bacteria on organic matter tends to the formation of nitrates and nitrites. The amount of lead in drinking-water need only be very small in order to cause chronic poisoning; $\frac{1}{100}$ of a grain per gallon is sufficient.

Symptoms.— Dyspeptic troubles, with loss of appetite, offensive breath, and unwonted constipation, are the earliest symptoms. Then follows anæmia, the skin acquiring a sallow, unhealthy appearance. Probably at this period a blue line will be found at the margin of the gums, due to the deposition of particles of lead sulphide in the papillæ of the mucous membrane. Small quantities of sulphur-containing food cling to the teeth, and, undergoing slow decomposition, give off sulphuretted hydrogen, which combines with the lead that is in a state of solution in the gums. Where the teeth are absent there is no blue line. The blue line may exist without any other symptom of plumbism, which, on the other hand, may be present, although no blue line is visible. At this period the patient will probably be thinner than usual owing to the disturbance of nutrition. The next symptom which usually occurs is colic as a rule, radiating from the umbilicus over the abdomen, the muscles of which are retracted and tense. It is often intermittent, with constant heavy pain between the attacks. It is relieved by pressure, except when it is extremely violent, and then the abdomen is tender and intolerant of pressure; in such cases, especially in young women, the patient rolls about the bed with contortions that might be mistaken for an hysterical seizure. During the attacks the pulse is slow and of high tension, there is little or no elevation of temperature; the bowels are usually obstinately confined, but occasionally they are rather relaxed. Deep-seated pain of a boring kind, most frequently about the knees, but sometimes affecting the elbows and shoulders, often occurs, to which the name arthralgia is given. After colic, one of the commonest symptoms of chronic lead poisoning is paralysis of the extensor muscles of the hands and fingers, producing "wrist drop"; the supinator longus usually escapes, and, as the affected muscles undergo atrophy, this muscle stands out in marked contrast to those that are wasted. When a patient suffering from wrist drop holds out his arms horizontally before him, with the palms downwards, the hands drop and cannot be raised, hence the name "wrist drop." Exceptionally the paralysis begins in the muscles of the upper arm, the deltoid, biceps, and coraco-brachialis being affected, and in this—the upper-arm type—the supinator longus is attacked. In each type both arms suffer, though one may be more affected than the other. The legs are not usually attacked, and never until the arms have been affected for some time; the extensors of the foot are the first to suffer; the tibialis anticus, like the supinator longus, usually escapes. In exceptionally bad cases the paralysis may spread to the muscles of respiration and deglutition. The characteristics of lead paralysis are: little if any disturbance of the sensory fibres, with the exception of occasional localised patches of anæsthesia, together with extreme atrophy of the affected muscles, the paralysis in ordinary cases being limited to the extensor muscles of the fore-arm.

In the course of chronic plumbism psychical disturbances may manifest themselves; sleeplessness, drowsiness, excitability, with hallucinations and wild delirium, may occur. Epileptiform convulsions are not uncommon, especially in women; sometimes they come on unexpectedly in the early stage of plumbism, before the commoner symptoms develop; more frequently they occur later in the course of the disease. They may be repeated at intervals for days, the patient remaining unconscious for a considerable time after each attack. Convulsions in lead poisoning are of serious import; they frequently portend a fatal result. In rare cases anomalous nerve symptoms may supervene—as general paralysis, ataxia, with girdle-sensation and loss of muscular co-ordination, probably due to neuritis, with possibly some changes in the cord; and, as observed by Oliver, a condition which closely resembled bulbar paralysis. Optic neuritis may occur as a consequence of plumbism; it may produce no noticeable impairment of vision, or it may lead

to absolute blindness which may be permanent. Not unfrequently some improvement of vision takes place even in the worst cases.

When lead is taken into the system for prolonged periods it tends to cause chronic nephritis, probably in the first instance of the parenchymatous variety, but leading eventually to interstitial nephritis with the formation of the granular kidney. As a result albumin will be present in the urine, but probably not until the system has been under the influence of lead for some time. The relation borne by granular kidney to lead on the one hand and to gout on the other has long been observed. There is no doubt that individuals with a gouty tendency succumb readily to the influence of lead, and that in such subjects gout is developed by the lead, but whether chronic lead poisoning can determine gout apart from the presence of a gouty diathesis is doubtful. Pregnant women usually abort when under the influence of lead —a fact so well known in some districts as to cause certain preparations of lead, *e.g.* diachylon-plaster, to be regarded as ecbolics.

Treatment.—The patient should be removed from the influence of lead and any special symptoms combated, colic and arthralgia by opium, paralysis by local massage and electricity, with rest. Potassium iodide is usually given, but with doubtful efficacy. Elimination is best promoted by fresh air, hot baths, general massage, moderate purgation, and a judicious amount of exercise.

Tests. — Sulphuretted hydrogen produces a brown or a black precipitate with lead salts. Potassium iodide gives a yellow precipitate which dissolves on boiling and crystallises, on cooling, in gold-coloured scales. Potassium chromate gives a yellow precipitate, and sulphuric acid a white precipitate.

Copper.—Metallic copper is but slightly, if at all, poisonous; copper coins, accidentally swallowed, have remained in the digestive tract for six and nine months without causing any symptoms of poisoning. The salts of copper by which acute poisoning is produced are the sulphate and the acetate.

Symptoms. — Immediately on swallowing a poisonous dose of one of the above salts a metallic astringent taste is experienced, followed, after a short interval, by pain in the stomach and abdomen, by vomiting and purging, and by collapse. The early vomit is green or blue, and probably the inside of the mouth and lips are tinted blue. The distinction between vomit coloured by a copper salt and by bile is determined by the addition of a little ammonia water: the coppery vomit is turned a deep blue, whilst the bilious vomit is unaltered in colour. Pain in the head often occurs, and occasionally convulsions. Jaundice has been observed.

Treatment.—Vomiting usually occurs spontaneously, and may be promoted by draughts of warm water; if no vomiting occurs the stomach-tube should be used. White of egg, barley water, thin arrowroot and milk should afterwards be given.

Fatal Dose.—Not known. One ounce each of the sulphate and of the acetate have severally proved fatal. Death may occur in a few hours, but is more usually delayed for several days.

Post-mortem Appearances.—Those of an irritant poison, with possibly bluish or greenish coloration of gastro-intestinal mucous membrane, which may be distinguished from bile-staining by the addition of ammonia water. The liver may show fatty changes.

Chronic Copper Poisoning. — Although the introduction of repeated minute doses of copper salts into the system does not produce symptoms of poisoning comparable with those produced by like doses of lead, it must not be assumed that repeated small doses of copper are innocuous. It is stated that workers in copper, even those engaged in the manufacture of verdigris, never suffer from chronic copper poisoning. Some observers, however, report cases which occurred in copper workers, that support the view held by Filehne and others—founded on experiments on animals—as to the resemblance between the toxic effects of copper and those of the other heavy metals. Chronic copper poisoning is mostly discussed in relation to the presence of salts of the metal in food; the contamination may be accidental, or it may be intentional. Accidentally, copper finds its way into food through the use of unclean brass cooking utensils; the copper boilers and hot-water cylinders in general use are also sources of contamination. Copper salts are purposely added to certain foods and condiments to improve their appearance; green-peas and some kinds of pickles are frequently thus treated. The law does not specially prohibit the addition of copper to aliments, it merely forbids the addition of any material which renders food injurious to health, and leaves it to the court before which the case is tried to decide what is, and what is not, injurious to health. This allows great latitude of opinion among expert witnesses, especially as it is difficult to obtain direct evidence of the toxic action of small doses of copper.

Tests.—Potassium ferrocyanide gives a chocolate-coloured precipitate, and ammonia water an azure blue colour with a solution of a copper salt. A piece of bright iron wire, or a steel needle, placed in a solution of a copper salt which has a slightly acid reaction, becomes coated by a film of metallic copper, the reaction being aided by heat. The wire, subsequently placed in a few drops of ammonia water, imparts a blue tint to the liquid.

Silver.—Acute poisoning by a salt of silver is very rare, most of the recorded cases being due to the accidental swallowing of a piece of lunar caustic whilst it was being used to

cauterise the throat. The *symptoms* are those of a corrosive and an irritant. The *treatment* consists in the adminstration of common salt, followed by an emetic. White of egg and ice, with hypodermic injections of morphine, are subsequently to be given. The fatal dose is unknown.

Post-mortem, there will be patches of greyish-white where the poison came in contact with the mucous membrane, in addition to the usual appearances produced by an irritant.

Chronic poisoning by silver is usually the result of prolonged medicinal treatment with silver salts. One of the chief symptoms is argyria; a lead-blue discoloration of the skin, due to the deposition of reduced silver in the papillary layer of the corium, which is permanent.

Tests.—Hydrochloric acid produces a curdy precipitate with a solution of a silver salt, which is soluble in ammonia water but is insoluble in nitric acid.

ZINC.—The salts by which acute poisoning is caused are the sulphate and the chloride. The *sulphate*, in poisonous doses, produces violent vomiting and the usual symptoms of an irritant. The treatment is limited to attention to the symptoms, as the poison is usually evacuated spontaneously and its toxic action is but feeble. The fatal dose is not known. Recovery has followed one ounce. The *chloride* is a powerful corrosive. Its action is to be combated by teaspoonful doses of sodium carbonate, white of egg, and opium.

Tests.—The formation of a white sulphide on the addition of sulphuretted hydrogen to a neutral or alkaline solution of a zinc salt, is a characteristic reaction. The gelatinous, opalescent precipitate given by potassium hydrate, and soluble in excess, is also characteristic. Potassium ferrocyanide gives a yellowish, gelatinous precipitate.

TIN.—Poisoning by tin-salts is very rare. It has usually occurred from the presence of the metal in the juice of tinned fruits, the acids of which, acting on the solder, or on the tinned surface of the containing vessel, have charged the juice with the corresponding salts of tin. The *treatment* consists in emptying the stomach and administering white of egg, milk, ice, and opium.

Tests. — Stannic salts give a yellow, and stannous salts a brown, precipitate with sulphuretted hydrogen. With mercuric chloride and a little hydrochloric acid, stannous salts give a white precipitate which turns grey and subsequently black.

BISMUTH. — Two drachms of bismuth subnitrate have caused death with symptoms of gastric irritation, together with stomatitis and foul breath. The treatment consists in evacuating the stomach and then giving ice, and opium if necessary.

IRON.—One ounce and a half of the tincture of perchloride of iron caused death after five weeks. The symptoms and treatment are those of an irritant.

CHROMIUM.—Half an ounce of a solution of *chromic acid*, 100 grains to the ounce, has caused death after being applied to papillomatous growths; chromic acid has produced severe toxic symptoms after being swallowed. *Potassium dichromate*, in two-drachm dose, has caused death in four hours; recovery has followed 270 grains. Lead chromate has also caused death. The symptoms produced are those of acute gastritis, with disturbance of respiration. The *treatment* consists in evacuating the stomach and then giving magnesium carbonate, or chalk suspended in water. Opium will probably be required and also stimulants.

NON-METALLIC ELEMENTS

PHOSPHORUS.—The toxic action of phosphorus is chiefly utilised as a means of committing suicide, for which purpose either rat-paste, which consists of finely divided phosphorus mixed with fatty matter artificially coloured, or the heads of matches, are swallowed. Phosphorus poisoning is more common in women than in men, partly because phosphorus is taken by women in order to procure abortion. Occasionally young children are fatally poisoned by sucking the heads of matches.

Symptoms of Acute Phosphorus Poisoning.—The primary symptoms are those of an irritant. They come on in from a few minutes to twelve, or more, hours. The early vomit and the patient's breath may appear luminous and yield a garlic-like odour. There is intense thirst, eructations, and a painful sensation in the gullet and stomach, with a foul tongue and a tender tympanitic abdomen. At this stage diarrhœa is more frequently absent than present. In rapidly fatal cases (which are the exception), the gravity of the symptoms increases until the patient dies in eight or ten hours. The more usual course is that the primary symptoms diminish somewhat in intensity, the diarrhœa, if present in the early stage, ceases, and the patient appears easier for one or two days, with persistence of the thirst and possibly occasional attacks of vomiting and a tender state of the abdomen. Then the secondary symptoms develop: the white of the eye and the skin acquire a jaundiced hue, pain is felt in the epigastric region, and the liver, and sometimes the spleen, are enlarged. The abdominal distension and tympany increase, the vomiting returns and is now accompanied by diarrhœa, both vomit and motions, probably, containing blood. The urine is high-coloured and scanty and contains albumin and bile pigments. There is a general hæmorrhagic tendency, shown by epistaxis, hæmaturia, purpuric spots, and, in women, metrorrhagia. The pulse is quick, but there is not much eleva-

tion of temperature. Stupor and coma supervene, and death soon follows: occasionally the mind remains clear. The temperature usually sinks towards the end, but, exceptionally, it rises and has been known to continue to rise for a short time after death.

The most characteristic feature of acute phosphorus poisoning is altered metabolism, shown by diminished oxidation of tissues and consequent modification of the excretory products. This is specially the case as regards the nitrogenous constituents of the urine: the urea is diminished, the deficiency being compensated by an excess of ammonia. Leucin and tyrosin are occasionally present in the urine. The altered metabolism is further shown by the presence of non-nitrogenous bodies in the urine, such as sarcolactic acid and, exceptionally, sugar.

Treatment.—Empty and wash out the stomach either with warm water or with a 0·1 per cent solution of potassium permanganate. Turpentine may be given in half-drachm doses. If diarrhœa does not occur, repeated movements of the bowels should be procured, as phosphorus may remain unoxidised in the intestines and may be absorbed; castor oil or other fatty matter should not be given, as fat is a solvent for phosphorus and would promote its absorption.

Fatal Dose.—The smallest recorded fatal dose is a grain and a half; recovery has followed six grains. Death has occurred under twelve hours; it is usually delayed from the second or fourth day to a week.

Post-mortem Appearances.—If death occurs early, the cavities of the body, when opened, may yield the odour of phosphorus, and occasionally phosphorescence has been observed. The mucous membrane of the stomach and duodenum is usually yellowish or greyish-white; limited ecchymoses and erosions may be present. The rest of the intestinal tract shows no changes except that small ecchymoses may be seen; exceptionally, the intestines have been found inflamed. The heart and kidneys show signs of fatty degeneration and the spleen is usually enlarged. The principal post-mortem sign of phosphorus poisoning is seen in the liver, which is large, of a yellow colour, and of doughy consistence. This appearance is due to the presence of a large amount of fat which replaces the normal liver tissue. In some respects acute phosphorus poisoning resembles acute yellow atrophy of the liver; both are of toxic origin, the latter being probably caused by the action of micro-organisms.

Chronic phosphorus poisoning is caused by inhalation of the fumes of phosphorus, which causes necrosis of the upper and lower jaw-bones. The phosphorus vapour acts on any bone, the periosteum of which is exposed; hence it attacks the jaws of work-people in lucifer-match manufactories, whose teeth are defective, obtaining access to the bone by way of the decayed teeth. If the teeth are sound, the vapour cannot reach the bone.

Iodine, *iodoform* and *potassium iodide* have each, on rare occasions, caused death,—mostly from the medicinal use of these substances.

Bromine has, on two or three occasions, caused fatal poisoning from being swallowed in the liquid form, and also from inhalation of bromine vapours. One or two cases of poisoning by *potassium bromide* are recorded, one being fatal.

Chlorine has caused death to work-people in chemical and bleach-works. The severe dyspnœa which the inhalation of chlorine produces is best relieved by putting the patient in a steam-tent.

Boric Acid has caused death by the injection of a 5 per cent solution into the pleural sac, after evacuation of an empyæma, by washing out the stomach, and by the use of a vaginal tampon of boric acid. The symptoms are depression, erysipelatous eruption on the face, vomiting, diarrhœa, and collapse. Skin eruptions, such as erythema and urticaria, on various parts of the body, are the most constant signs of boric poisoning.

Gaseous Poisons

Sulphuretted Hydrogen.—With the exception of accidents in chemical works, poisoning by sulphuretted hydrogen generally results from the inhalation of sewer-gas. When the percentage of sulphuretted hydrogen is limited, the symptoms produced are a sensation of "catching of the breath," a small pulse, oppression in the head, vomiting, dizziness, and probably diarrhœa, with great muscular prostration; with a large percentage, asphyxia and heart-failure, cyanosis, delirium and convulsions may occur, and very rapid death.

Treatment.—Artificial respiration, with friction over the cardiac area and along the limbs, and the application of external warmth, are helpful. If the surface is hot, drenching with cold water may be tried.

Post-mortem Appearances.—Putrefactive changes usually quickly follow death; but this is not always the case. Cadaveric rigidity has been noticed to be well marked. The blood is fluid and dark in colour, consequently the viscera are darker than usual. The muscles sometimes have a bluish tint. The lungs will probably be œdematous.

Carbon Dioxide.—Death from this gas may occur in coal-mines, after an explosion which gives rise to "choke damp," in deep wells and excavations, in brewers' vats, and in the neighbourhood of lime- and brick-kilns. The usual method of testing the respirableness of air suspected to be contaminated with carbon dioxide is to lower a lighted candle into it; if the candle goes out the atmosphere is poisonous; if it continues to burn, however, the air is not necessarily harmless, as a candle will burn in an atmosphere that is dangerous to life.

Symptoms.—Heaviness in the head, giddiness, noises in the ears, tightness of the chest and an inclination to sleep are experienced, and, shortly, the muscles lose power, the individual falling to the ground. Subsequently, the symptoms are those of asphyxia. In a concentrated atmosphere of carbon dioxide, as in a well, or a vat charged with the gas, immediate loss of consciousness and muscular power, followed speedily by death, occurs, not unfrequently to workmen who incautiously venture into such an atmosphere. No one should undertake the function of rescuer without having the noose of a rope round his body, the free end being in charge of bystanders; the omission of this precaution has often led to the multiplication of victims.

Post-mortem Appearances.—Those of death from asphyxia:—dark-coloured fluid blood, distended right heart and veins, hyperæmia of the lungs, with frothy mucous in air-passages.

Carbon Monoxide.—Poisoning from this gas may be due to the fumes given off by slow combustion stoves, or by coke burning in an open fireplace; the death of those who are in a building on fire, from which escape is cut off, is frequently due to carbon monoxide. Perhaps the most usual way in which poisoning by carbon monoxide occurs is from breathing air contaminated with coal-gas, or, more especially, with water-gas, which is now so commonly used either to adulterate or to replace coal-gas. An atmosphere containing 1 per cent of carbon monoxide would soon prove fatal to those who breathed it; much less might do so if breathed for a considerable time. Coal-gas contains from 4 to 8 per cent of carbon monoxide, whilst water-gas contains 40 per cent; when water-gas is mixed with coal-gas the percentage of carbon monoxide is reduced to about 16.

Symptoms of Acute Poisoning by Carbon Monoxide.—A sensation of heaviness in the head, dizziness, noises in the ears, quickening of the action of the heart and lungs, oppression on the chest, and occasionally vomiting, occur along with loss of muscular power; coma quickly supervenes, and convulsions may precede death. The pulse is small, the surface is cold and cyanotic, the conjunctivæ are hyperæmic, and the pupils somewhat dilated. The lips are often covered with froth.

The toxic effects of carbon monoxide are due to its affinity for hæmoglobin, with which it combines, and for which it has an affinity two hundred times greater than that of oxygen. The compound of carbon monoxide with hæmoglobin is much more stable than that of oxygen with hæmoglobin, and so long as hæmoglobin is combined with carbon monoxide it is useless as an oxygen carrier; if, therefore, more than a certain amount of the hæmoglobin be thus rendered functionless, death results from asphyxia due to arrest of internal respiration. The combination of carbon monoxide with hæmoglobin takes place progressively with each respiration made in an atmosphere contaminated with the gas; the action is strictly cumulative, so that the duration of the exposure as well as the amount of gas present has to be taken into account when considering the effects likely to be produced by a given admixture.

Treatment.—The most important object is to keep the tissues supplied with a sufficiency of oxygen to enable life to continue until the carbon monoxide is dissociated from the hæmoglobin with which it is combined; for although, in a certain degree, carboxyhæmoglobin is a stable compound, it gradually parts with the carbon monoxide whilst circulating through lungs which are breathing pure air. Artificial respiration, therefore, along with the inhalation of oxygen, when available, should be persevered with for a long time. External warmth must be maintained and stimulants administered either by the rectum or hypodermically (in the form of ether), should the patient be unable to swallow. Bleeding, followed by the transfusion of an equal amount of defibrinated human blood, has been resorted to, but not so successfully as, on theoretical grounds, might have been expected.

Post-mortem Appearances.—Externally, the appearance is very characteristic: in a well-marked case the surface of the body is rosy-red, and the post-mortem stains are bright pink in colour. On opening the body the colour of the tissues is equally striking; the intestines are of a light red colour and the whole of the viscera are much brighter in colour than usual. The blood itself is cherry-red in colour, and, for the most part, is fluid. Froth may be found in the air-passages, and the lungs may be œdematous. The tissues and the blood from a case of poisoning by carbon monoxide resist putrefactive changes for a much longer time than is usual after death from other causes.

Blood that is charged with carbon monoxide yields a spectrum which closely resembles that of oxyhæmoglobin, the two bands being slightly nearer the violet end of the spectrum than those of oxyhæmoglobin. The critical difference lies in the fact that the spectrum of carboxyhæmoglobin undergoes no change on the addition of a reducing agent to the blood. When ammonium sulphide is added to ordinary blood, the oxyhæmoglobin is reduced and its spectrum is changed from two bands to one broad band; the same reagent produces no effect on being added to blood which is fully charged with carbon monoxide. It is to be observed, however, that death usually takes place before the hæmoglobin is fully saturated with carbon monoxide; the result being that whilst the hæmoglobin that is combined with carbon monoxide resists the action of a reducing agent, that which is uncombined is reduced in the ordinary way; therefore the spectrum yielded by the blood of

those who have died from carbon-monoxide poisoning usually shows a broad band of reduced hæmoglobin, with the two narrow bands of carboxyhæmoglobin superimposed.

Chronic poisoning by carbon monoxide occurs among those who work for long hours in badly ventilated rooms heated by slow combustion stoves, or gas stoves, which give off carbon monoxide, but in too small amount to produce acute poisoning. Furnacemen, stokers, and the workmen in gasworks where water-gas is made are also liable to chronic poisoning. In private houses, the use of coke in open fireplaces, leaky, slow combustion stoves, and ill-ventilated gas-cooking stoves are sources of danger. A most insidious risk to health, now unfortunately very widespread, arises from the introduction of water-gas into private houses as a means of illumination; the percentage of carbon monoxide is so high that the slightest leakage in the household gas-fittings may give rise to chronic poisoning.

Symptoms.—The early symptoms are easily mistaken for simple anæmia and malnutrition. They comprise headache, neuralgic pains, loss of appetite, insomnia, anæmia, loss of flesh and breathlessness, and palpitation of the heart on exertion. Subsequently, peripheral neuritis and psychical disturbances may occur. The treatment consists in removing the patient from the source of mischief, and in dealing with the anæmia and other symptoms by appropriate medicines and change of air.

Cyanogen Compounds

Hydrocyanic, or *prussic acid*, is one of the most deadly poisons known. *Oil of bitter almonds* contains a varying amount of hydrocyanic acid which, in the crude oil, reaches to from 5 to 15 per cent. *Potassium cyanide* is a common and dangerous cyanogen salt.

Symptoms.—When a fatal dose of hydrocyanic acid is swallowed, the symptoms usually appear within a few seconds; they may be delayed for half a minute, and have been known to be delayed for over a minute. After a few gasps the patient falls to the ground in an unconscious condition; the surface is cold, the face pallid, the eyes are staring with insensitive pupils, the breathing is gasping, and quickly ceases with a few convulsive efforts. Tetanic spasms often occur in the early stage, followed by complete muscular relaxation. The pulse is extremely rapid; it can rarely be felt at the wrist. The lips may be covered with froth; vomiting and involuntary micturition and defæcation are not uncommon. Death usually occurs within from five to ten minutes; it has been known to be delayed for over an hour. When life is prolonged for half an hour there is good chance of recovery.

Treatment. — Immediate evacuation of the stomach with stomach-pump or an emetic is indicated, but, on account of the extremely rapid action of the poison, it can rarely be put in practice. Artificial respiration, faradisation of the phrenics and the diaphragm, subcutaneous injections of ether, brandy by the rectum, and, if the surface is not cold, effusion with cold water, alternated with vigorous friction and the application of hot cloths or bottles. For all practical purposes antidotes are useless.

Fatal Dose.—Half a drachm of the B. P. acid caused death in one hour and twenty minutes. One grain of the anhydrous acid constitutes an ordinary fatal dose; the ultimate result is not influenced by dilution, though the action may be slightly delayed. Recovery has taken place after half an ounce of the medicinal acid, equal to 4·8 grains of the anhydrous acid.

Post-mortem Appearances.—The eyes, glassy-looking, with dilated pupils, are usually prominent; the fingers and the jaws are clenched and the lips are covered with froth; the post-mortem stains may be light red in colour. On opening the body, the odour of the poison may be perceived; the blood is almost always fluid and is sometimes light-coloured, in which case the various mucous membranes and other tissues may also be light-coloured.

Potassium cyanide acts like hydrocyanic acid with the addition of erosion and softening of the mucous surfaces, with which the poison came in contact, due to the action of the potash. The inner surface of the stomach may be deeply injected and softened, or eroded. Five grains of the salt has caused death, and recovery has followed forty grains.

Oil of bitter almonds in a teaspoonful dose has caused death.

Tests.—Place a small drop of a solution of silver nitrate on the concave surface of a watch-glass and invert it over a substance suspected to contain free hydrocyanic acid; in a short time, if the acid be present, the transparent drop becomes milky white from the formation of minute crystals of silver cyanide, which are soluble in hot, concentrated nitric acid. This test may be applied over the mouth of a dead body recently poisoned by hydrocyanic acid, or over the stomach after it has been opened. If a drop of a solution of potassium hydrate be substituted for the silver solution and, after exposure over the acid, a drop of a solution of ferrous sulphate is added and then a drop of hydrochloric acid, a precipitate of Prussian blue is obtained. If potassium cyanide was the poison, sufficient tartaric acid to produce an acid reaction must be added to any suspected fluid, before it is tested as above, in order to set free the hydrocyanic acid.

Organic Poisons

Alcohol.—Acute poisoning by alcohol varies from what is easily recognised as alcoholic intoxication to a profoundly comatose condition

which cannot be distinguished from apoplexy and many other causes of total insensibility. The face may be either flushed or pale, the conjunctivæ are injected, the pupils may be either contracted or dilated, the breathing is slow and more or less stertorous, the pulse is scarcely perceptible, the surface is cold and clammy, and on account of the extremely low arterial tension and laboured breathing the face is usually cyanotic. If the case goes on to a fatal issue, the lungs become œdematous and cardiac failure occurs.

Treatment.—The stomach-pump or an emetic is to be resorted to, and hot coffee administered, which probably can only be accomplished with the aid of a tube. Friction applied to the surface, alternating with cold douches should the skin be hot, along with attempts to rouse the patient from time to time, are helpful. Should the patient not respond, the faradic current may be applied, and in extreme cases artificial respiration may be needed. If there is any doubt as to the cause of the coma, keep the patient under observation as a possible cerebral lesion, until the diagnosis is obvious. The diagnosis may be aided by adding a few drops of potassium dichromate to some of the patient's urine in a test-tube and then allowing some strong sulphuric acid to flow down the side of the tube to the bottom, where a green colour is developed if alcohol be present.

Post-mortem Appearances.—If the patient died shortly after drinking a large quantity of spirit, the indications will be characteristic. The stomach and various cavities of the body yield the odour of the spirit which was swallowed. The mucous membrane of the stomach may be injected or pale. The lungs are hyperæmic, and the right side of the heart and veins are filled with dark-coloured fluid blood. The bladder usually contains a large quantity of urine, which may be tested as above described. If the deceased was an habitual drunkard, the usual tissue changes due to chronic alcoholism will also be present.

CHLORAL HYDRATE.—Soon after a poisonous dose has been swallowed the patient becomes drowsy and then comatose. The face is cyanotic, or is pallid, and the entire surface is very cold and moist. The respirations are slow and laboured; the pulse is small and, in the later stage, is slow. The pupils are usually contracted. Death takes place from heart-failure.

Treatment.—The stomach should be emptied by the tube or an emetic, and hot coffee injected into it. The body should be wrapped in blankets and hot bottles applied. Friction of the surface is useful. If breathing fails, artificial respiration must be carried on, and hypodermic injections of a twenty-fifth of a grain of strychnine administered. Alcohol may be given by the mouth or rectum, and ether subcutaneously. Attempts should be made from time to time to rouse the patient.

Fatal Dose.—Twenty grains have caused death to an adult, and three grains to a child one year old. Recovery has followed 420 grains. Death has occurred in fifteen minutes; it may be delayed for six or more hours. There is no characteristic post-mortem appearance.

Test.—To a test-tube nearly full of a solution of chloral hydrate add a single drop of ammonium sulphide, and mix by closing the tube with the thumb and inverting it. Then heat the upper stratum of the liquid in the flame, when it at once darkens in colour and afterwards becomes turbid.

CHLOROFORM.—When a poisonous quantity of chloroform is taken into the system, either by inhalation of its vapour or by swallowing the poison in the liquid state, the face becomes pale and cyanotic, the features are sunken, the entire surface is cold and clammy, the pupils are insensitive to light and are frequently dilated; the corneal reflex is abolished, the breathing is stertorous, the pulse is small and slow and of low tension. Death occurs from paralysis of the respiratory centres or from cardiac failure. When chloroform is swallowed in the liquid state gastro-intestinal irritation is produced by direct contact of the poison with the mucous membrane.

Treatment. — When the poison has been swallowed the stomach should be emptied and washed out with the tube. The inhalation of amyl nitrite and artificial respiration will probably be needed whether the poison was swallowed or its vapour was inhaled. The patient should be placed in the horizontal posture and external warmth applied. If heart-failure threatens, invert the patient so as to keep the brain supplied with blood.

Fatal Dose.—Swallowed in the liquid state seven fluid drachms caused the death of an adult, and one drachm caused the death of a boy four years old. Recovery has occurred after four ounces. Death has taken place in one hour, and has been delayed for forty-eight hours.

Post-mortem Appearances.—With the exception of signs of irritation and softening of the mucous membrane of the stomach, and possible fatty changes in the liver, kidneys, and heart, no indications of the action of the poison are present.

SULPHONAL has frequently given rise to symptoms of poisoning, and on several occasions has caused death. One of the most characteristic indications that sulphonal is acting injuriously is an abnormal coloration of the urine. After sulphonal has been taken, in medicinal doses, for some time, the urine has often been observed to vary from burgundy red to reddish black in colour; this is due to the presence of an unknown pigment associated with hæmatopor-

phyrin, an iron-free derivative of hæmatin, which demonstrates that the drug is acting as a blood poison. Vomiting and pain in the stomach and abdomen, profuse perspiration, accelerated pulse and respiration, reeling gait, ataxia, and suppression of the urine, have also been recorded. Death has taken place from cardiac failure. Enormous doses of more than an ounce—three ounces in one case—have been recovered from. When sulphonal, or trional, is being given for prolonged periods, the urine should always be inspected at frequent intervals, and if it be discoloured the drug should at once be stopped.

Carbon Bisulphide.—Acute poisoning by this substance is very rare. In one instance half an ounce caused death in a little over two hours; recovery has followed two ounces. The cause of death is paralysis of the respiratory centres. Tha treatment consists in emptying the stomach, applying warmth to the body, and using artificial respiration if the breathing flags. Elimination of any of the poison that has been absorbed should be promoted by purgatives and diuretics. The tendency to drowsiness which is usually manifested should be combated as in poisoning with narcotics generally.

Chronic poisoning by carbon bisulphide is met with among those who are engaged in certain departments in rubber factories. The initial symptoms comprise nausea, vomiting, headache, anorexia, sleeplessness, colicky pains, and mental instability—the patient is either exalted or depressed. Later on symptoms of multiple neuritis occur: burning, numbness, and tingling in the hands and feet, followed by paralysis, producing wrist- and ankle-drop. The field of vision has been found restricted for all colours; amblyopia and scotomata, without retinal changes, are common.

Petroleum and Paraffin Oil have produced dangerous symptoms and even death, but they cannot be regarded as active poisons. Some kinds appear to be much more noxious than others; on more than one occasion half a pint of petroleum, or of paraffin oil, has been swallowed without causing more than temporary disorder. On the other hand severe symptoms have occasionally been produced by lesser quantities, and in one or two instances death has resulted. The symptoms produced are not constant; they comprise vomiting, abdominal pain, the odour of the poison in the breath, drowsiness, collapse, feeble pulse, sighing respiration, cyanosis, and the presence of some of the oil in the motions and even in the urine. The motions have been mixed with blood. The pulse and respiration rates have been much accelerated; the pulse to 140, and the respirations to 50 in the minute.

Benzene, also called benzole, produces toxic symptoms which resemble those caused by alcohol. Fatal poisoning has resulted from the inhalation of the vapour of benzene. In addition to the odour of benzene in the breath, the respirations are slow—may be only eight to the minute—and stertorous; the pulse is usually quick, and when death takes place it has been observed to be due to cardiac failure. When recovery has taken place the odour of benzene in the breath has persisted for two or three days. One ounce has proved fatal.

Nitro-Benzene has caused death in small doses, such as twenty drops; recovery has followed nearly one ounce. The symptoms comprise giddiness; loss of power; vomiting; quick, feeble pulse; shallow, irregular breathing; subnormal temperature, and a peculiar cyanotic appearance of the face and fingers, with dark blue lips. The treatment, as with benzene, is to wash out the stomach, to apply external warmth and friction, and, if necessary, to resort to artificial respiration.

Coal-Tar Derivatives.—Aniline, often called aniline oil, speedily causes symptoms of poisoning when swallowed; six drachms have caused death. The symptoms resemble those caused by nitro-benzene, the most remarkable being a peculiar blueness of the body, more marked than ordinary cyanosis, which persists after the breathing has become natural, and may occur without any disturbance of the respiration.

The *treatment* is the same as for nitro-benzene.

Antifebrin has frequently produced "cyanosis" and other poisonous symptoms when administered under medical supervision. Of recent years the sale of "headache powders," which contain from four to ten grains of antifebrin, has placed the drug within the reach of ignorant people who freely resort to it, and take it in a most reckless way. In one instance, a woman took six of these powders every day for a month, which caused the skin to become bluish grey for more than a fortnight. The application of antifebrin, as an antiseptic, to raw surfaces has caused death.

Antipyrin has occasionally given rise to symptoms of poisoning, but not so frequently as antifebrin. In addition to the blue discoloration of the cheeks and lips, excessive doses of antipyrin have caused urticarial and other rashes to break out over the body.

Phenacetin on two occasions has caused death, and has often caused serious symptoms, such as dyspnœa, vomiting, diarrhœa, headache, and vertigo, together with the blue—sometimes almost black—hue of parts of the surface of the body, especially of the face; the mucous membrane of the mouth also may be discoloured. The urine may become chocolate-coloured, due to the presence of methæmoglobin. All these coal-tar derivatives, by prolonged administration, tend to act as blood-poisons.

Salicylate of Soda sometimes produces untoward effects, such as hæmorrhage from the gums, epistaxis, retinal hæmorrhages, hæma-

turia, albuminuria, vomiting, and urticaria. In one case great dyspnœa and stridulous breathing, with extreme slowness of the pulse, were produced by excessively large doses

Creasote when swallowed in poisonous doses produces abdominal pain, nausea, vomiting, and diarrhœa. Serious symptoms have from time to time been recorded after large or prolonged medicinal doses, and, in an exceptional case, death occurred in five days to a woman of fifty-two, who swallowed three six-drop doses in milk; in this case erosions were found in the œsophagus, the stomach was injected, and the kidneys were acutely inflamed. On the other hand enormous doses have been administered in the treatment of tuberculosis without ill results; the dose has been gradually increased from one up to a hundred and more drops, and in one case up to two or three quarter drachms twice a day; in this case, however, the patient was made very ill.

Alkaloids and Vegetable Poisons

Acute alkaloidal poisoning in general is characterised by the absence of local mischief, and by the presence of symptoms which are due to absorption of the poison and to its effect on the nerve centres. This is in marked contrast to the effect produced by acute poisoning with inorganic substances, which for the most part is attended by considerable local disorganisation or inflammatory mischief, often with relatively insignificant absorptive effects. The non-alkaloidal organic poisons stand between the inorganic and the alkaloidal poisons as regards the symptoms they produce; they cause more obvious derangement of the tissues than the alkaloids, and they exercise a greater influence, due to absorption, than do the inorganic poisons. In consequence of the absence of local effects, the alkaloids leave scanty post-mortem indications of their action; so that the sole means of confirming the diagnosis of poisoning made during life consists in the detection, by chemical analysis, of the poison in the viscera after death. Some of the alkaloids, strychnine, for example, are very stable, and, by chemical processes, can readily be separated from organic matter, and recognised even when present in very small amount; others, such as aconitine, by reason of their instability, are difficult to isolate, and when isolated their identity is not capable of being definitely determined by chemical reagents, but demands additional experimentation on some of the lower animals.

Strychnine is the most commonly used alkaloidal poison, both by murderers and by suicides; it also frequently causes accidental death.

Symptoms.—In three or four minutes, to a quarter of an hour and occasionally longer, muscular twitchings, accompanied by a feeling of anxiety and impending suffocation, are experienced, followed immediately by violent convulsions. The arms and legs are stretched out, the hands are clenched, and the whole of the skeletal muscles are taut and hard; then clonic spasms come on with progressively increasing violence, the head and feet are jerked backwards and the trunk forwards; very soon the spasm becomes tonic, and every muscle is stretched and contracted to the uttermost, the body often being supported on the back of head and the heels (opisthotonos). During the stage of tonic spasm respiration is arrested, or thereabouts, and the patient becomes cyanosed, the pulse being very rapid and feeble. The terror-struck face, prominent eyeballs, and dilated pupils demonstrate the physical pain and the mental anguish experienced by the patient, who is fully conscious of his sufferings and dreads immediate death. When he can speak he cries out for something to be done to relieve his agony. After a minute or more the muscles relax, the eyeballs cease to be prominent, and the pupils resume their usual size; the respiration becomes easier, the cyanosis disappears, and the pulse slows down. The patient lies exhausted, dreading a return of the spasm, which sooner or later recurs, the period of remission lasting from a few seconds to five or ten minutes. If the case is about to terminate fatally the spasms succeed each other rapidly, and death usually takes place within two hours, either from asphyxia or from exhaustion. When recovery takes place the spasms are separated by longer and longer intervals of quiescence, and are less and less violent, the patient usually recovering from the sequential state of feebleness and exhaustion in a few days.

Treatment. — Chloroform should be administered by inhalation, so as to enable the stomach-tube to be introduced, with which the stomach is then washed out. If the stomach-pump is not available an emetic should be given, as spontaneous vomiting rarely occurs. Chloral hydrate may afterwards be given—if necessary by being injected under the skin—in ten- or twenty-grain doses, dissolved in water and repeated until the spasms cease. If the patient can swallow, twenty grains may be given by the mouth and repeated as often as necessary. If death from asphyxia is threatened artificial respiration should be resorted to.

Fatal Dose.—Half a grain of strychnine sulphate caused death in twenty minutes; a little over a grain has frequently proved fatal. Recovery has taken place after ten and even twenty grains. Exceptionally, the symptoms do not commence for one or even two hours after the poison was swallowed. Death also may be delayed for three, or even seven hours; when a narcotic is taken with the strychnine still longer periods of survival have been recorded.

There are no characteristic post-mortem appearances produced by strychnine.

Tests.—Strychnine has a bitter taste which,

unless disguised by strongly flavoured substances, can be detected in solutions containing 1 part in 70,000. If a fragment of strychnine is mixed with a couple of drops of strong sulphuric acid, and a few granules of manganese dioxide are stirred into the mixture with the point of a glass rod, a blue colour is produced which rapidly becomes purple and then gradually changes to red. A few drops of a solution of strychnine injected into a frog produce convulsions in a few minutes.

Nux Vomica, which owes its toxic activity chiefly to the strychnine it contains, has occasionally caused death. Brucine, the other alkaloid of nux vomica, acts like strychnine, but much less powerfully. It is almost unknown as a poison, not being accessible to the public.

Cocculus Indicus, the active principle of which is picrotoxin, in small doses produces symptoms like those of alcoholic intoxication, and in larger doses it causes clonic spasms like those due to strychnine.

Opium and Morphine.—The medicinal preparations—official and not official—are very numerous, and almost all of them, from time to time, give rise to symptoms of poisoning. Laudanum contains 0·75 per cent. of morphine. A drachm of paregoric is equal to a thirtieth of a grain of morphine hydrochlorate, and the strength of Dover's powder is equivalent to 1 per cent of morphine. Many of the soothing drugs which are often improperly given to children contain small amounts of opium. Dalby's carminative contains about two and a half minims of laudanum to the fluid ounce; Atkinson's preserver, three minims to the ounce; Godfrey's cordial contains from half a grain to a grain and a half of opium to the ounce. Chlorodyne contains about four grains of morphine hydrochlorate to the ounce.

Symptoms.—The first effect of a poisonous dose of opium or of morphine is excitation of the nerve-centres, which comes on in from half an hour to an hour after opium is swallowed, and in from a few minutes to a quarter of an hour if the poison be morphine. The action of the heart and of the lungs is accelerated, the face is flushed, and the mental condition is exalted; this stage is very evanescent, sometimes so much so as to pass unrecognised. The next stage begins with a feeling of lassitude, heaviness in the head, giddiness, and a progressively increasing tendency to sleep. The patient becomes drowsy and sinks into a condition of stupor. The pupils are contracted, the pulse is slow; the breathing at this period being deep and regular. The patient can be roused somewhat by being shaken and loudly spoken to, and if the dose of opium has not been very large, he may be kept more or less conscious by various methods of stimulation. When a large dose has been taken the stupor deepens into coma, and then external stimuli utterly fail to evoke any response: the muscles are relaxed, the surface is cold and clammy, the features are shrunken and either pallid or cyanotic, the pupils are extremely contracted, the pulse is still slower, and the breathing is laboured, irregular, and stertorous. If the case ends fatally the patient becomes more profoundly collapsed, the breathing more embarrassed, the pulse more irregular, and the general appearance more death-like. The pupils may dilate immediately before death, and the heart may beat for a short time after respiration has ceased. In fatal cases death usually occurs in from six to twelve hours.

If opium itself, or the tincture, was taken, the odour of opium may be perceptible in the breath. All the secretions, save that of the skin, are suppressed or diminished, so that the bowels are confined, the urine is feebly secreted, and is retained in the bladder, on account of loss of sensitiveness and of expulsatory power, and the mouth is parched from arrest of the salivary secretion. In children convulsions may occur; they are exceedingly rare in adults. Occasionally the urgent symptoms mitigate to such an extent as to remove all fear, and then, after several hours of distinct improvement, the patient relapses into coma and dies.

Treatment.—If the poison has been swallowed the stomach should be well washed out with a solution of potassium permanganate—ten to fifteen grains in six or eight ounces of water, repeated at half-hour intervals; in default of the stomach-pump an emetic may be given, or eight to ten drops of injection of apomorphine (B.P.) may be administered under the skin. Attempts should be made to rouse the patient by means of external stimuli, such as flapping the face and chest with a wet towel, or the application of the faradic current to various parts of the body. In the mild form of opium poisoning the patient may be walked about between two assistants, and, if the surface of the body be warm, occasional douching with cold water may be resorted to, the patient being kept awake, by these various means, until the effects of the poison have passed off. In cases of severe opium-poisoning the treatment is different; in such cases it is worse than useless to drag a comatose person about. He should be put to bed, warmth should be applied to the surface, and, if necessary, artificial respiration should be diligently performed. Hot coffee may be injected into the stomach or the rectum, and hypodermic injections of atropine sulphate ($\frac{1}{20}$ of a grain) may be given at not too frequent intervals. Hypodermic injections of strychnine ($\frac{1}{30}$-$\frac{1}{20}$ of a grain) have proved very efficacious. Ether also may be injected under the skin.

Fatal Dose.—To adults, four grains of opium in one case, and in another two drachms of the tincture proved fatal. Recovery has taken

place after three ounces, and even after eight ounces of laudanum. One grain of morphine hydrochlorate has caused death, and recovery has followed thirty, and even fifty-five grains. Recovery has followed the subcutaneous injection of about twelve grains of morphine. To infants opium is very dangerous; one drop of the tincture may give rise to serious symptoms, and even to death. Recovery, in an infant three months old, occurred after it had swallowed a teaspoonful of laudanum. In another case an infant six days old survived a grain and a half of opium.

Death has been caused by sprinkling an open sore with morphine.

Post-mortem Appearances.—There is nothing characteristic to be found after death other than the presence of some of the poison in the stomach. The blood may be dark and fluid, and there may be hyperæmia of the brain and its membranes.

Tests.—A drop of nitric acid added to a minute fragment of morphine produces an orange-yellow colour. If a centigramme of molybdic acid is dissolved, with the aid of gentle heat, in a cubic centimetre of sulphuric acid, and a drop of the mixture (when cold) be mixed with a fragment of morphine, a reddish purple colour is produced, which changes to blue. An aqueous solution of iodic acid shaken up with a little carbon bisulphide imparts to it no coloration; the addition of a trace of morphine liberates iodine from the iodic acid which, on agitation, colours the carbon bisulphide pink or rose-red.

Chronic opium-poisoning, also called opium-eating and morphinism.—Opium is a poison towards which an extraordinary degree of tolerance may be acquired by persons who habitually take it in gradually increasing doses; probably due to a progressively increasing power of the tissues to destroy the morphine, rather than to an acquired indifference to its action. The habit is frequently initiated by the medicinal use of opium to relieve pain or to procure sleep. After a time the patient feels depressed and uncomfortable when not under the influence of the drug, and the temptation to take it surreptitiously is more than many people can withstand. Once begun the practice is extremely difficult to give up; hence it behoves medical practitioners to guard against exposing patients to the allurements of opium, and when the drug needs to be prescribed for a time, to earnestly warn the person taking it against prolonging its use beyond the specified term. One of the most pronounced characteristics of a person addicted to the habitual use of opium is the total absence of the moral sense, especially as regards the means used to secure indulgence in the drug. Whilst capable of special efforts, he shuns methodic work and routine duties; at the time when he feels the stimulating effects of the opium he is at his best and is companionable; at other times he is dull, restless, and captious. He will resort to any lengths to obtain the craved-for indulgence, and, after succeeding, will deny the fact with an astounding earnestness even to those who are intimately acquainted with his habits. In these cases the heart's action is irregular, the appetite is capricious, nutrition is impaired, and emaciation occurs which is often associated with an unhealthy appearance like that due to malignant disease. The patient may suffer from visceral neuroses which often give rise to attacks of violent pain in the abdomen, resembling gallstone colic; also to periodical vomiting, like gastric crises. Later on indications of peripheral neuritis may appear, such as paræsthesia, numbness of the hands and feet, with ataxic gait.

Amongst better-class people the use of the hypodermic syringe has, to a great extent, replaced the more crude method of drinking laudanum. Enormous doses of morphine are thus self-administered; as much as forty grains of morphine acetate, as a daily dose, has been reached and even exceeded. Under no circumstances should a medical practitioner leave a hypodermic syringe in the possession of the patient. On some occasions the baneful habit has been brought about by the medical attendant, innocently, and without any idea of the evil that was likely to ensue, leaving a hypodermic syringe in charge of a patient who is subject to some recurrent painful affection, in order that he might be able to afford the sufferer immediate relief at a moment's notice. Sooner or later the patient takes the matter into his own hands, and uses the syringe secretly, with disastrous results.

The best treatment of chronic opium poisoning is to cut off the supply at once, which, however, is extremely difficult to accomplish unless the patient is placed under restraint. The abrupt cessation of the habit is held by some to be dangerous; there may be cases where it is so, and then gradual withdrawal, combined with some special treatment, may be needed. It is risky to substitute some other drug for opium, as a means of weaning the *habitué* from its use; such a procedure is likely to complicate the issue and to prolong the cure. Care must be taken, both during and after the cure, to restrain the patient from the abuse of alcohol—an occurrence not at all improbable. In addition to an intense craving for the drug so long indulged in, severe gastric pains, due to irritation of the sensory nerve-endings in the mucous membrane of the stomach by the gastric juice, are often experienced by those who have recently ceased the opium habit; the pains may be allayed by occasional doses of sodium bicarbonate. Morphine, whether swallowed or injected subcutaneously, is chiefly eliminated by the stomach; consequently, during the regular daily reception of the drug the nerves of the gastric mucous membrane are kept benumbed; when the be-

numbing influence is withdrawn, the nerves for a time are hyperæsthetic, and as the gastric juice at the same time tends to be abnormally acid, much suffering results.

BELLADONNA.—Poisoning by belladonna is usually due to the swallowing of a preparation of the drug which is intended for external use, or to eating the fresh berries. Atropine has caused death by being applied in the form of an ointment to a blistered surface; it has frequently produced symptoms of poisoning when a solution containing it has been dropped into the eye in order to dilate the pupil.

Symptoms.—Soon after a poisonous amount of belladonna has been swallowed the symptoms begin and rapidly increase in severity. A hot, dry sensation, and a feeling of constriction, are experienced in the throat, which are accompanied by thirst; the tongue is dry, the saliva is inspissated, and swallowing is almost impossible. The pupils are dilated to the uttermost, leaving only a narrow ring of the iris visible, and they are insensitive to light. The conjunctiva are suffused. The pulse is quick—120 to 160 in the minute—and is often so small as scarcely to be felt at the wrist. In the early stage the skin is hot and dry, and is often covered with a scarlatina-like rash, which may be followed by desquamation; in the later stage the skin may be cold. Dizziness and indistinctness of vision occur, and may be succeeded by complete loss of sight and incapacity to walk. Next to the extreme dilatation of the pupils the most characteristic symptom of belladonna poisoning is the peculiar type of delirium that occurs; it is excitable, lively, and imitative; the patient is voluble, and pours forth a stream of incoherent, stammering utterances, interspersed with laughter and shouting. Imitative actions are performed with a gravity of demeanour and of persistence that is ludicrous; the patient will go through the movements of counting coins, or of putting something in his mouth and chewing it, for a prolonged time, the movements being almost machine-like in their exact sequence. Numbness of the hands and feet is frequently present, the patient usually being unable to stand. The intestines and the bladder are generally paralysed. Recovery is slow; many days elapse before the pupils regain their normal size, and visual accommodation and perception are still longer defective. After severe poisoning the memory may be enfeebled, especially for names and persons and things, producing a kind of aphasia. A large proportion of cases of belladonna poisoning recover, including those in which the symptoms were severe.

Treatment.—Wash out the stomach or give an emetic. Afterwards strong tea and coffee may be administered. Hypodermic injections of pilocarpine, one-third to half a grain of the hydrochlorate, act antagonistically. If pilocarpine is not available, quarter or half-grain doses of morphine may be substituted. During the hot stage douching and sponging with cold water is beneficial; in the more advanced stage of severe poisoning artificial respiration may be required.

Fatal Dose.—One drachm of belladonna liniment, and the same amount of the extract, have respectively caused death after being swallowed. Recovery has taken place after ten drachms of the liniment, and after half an ounce of the extract. Fourteen belladonna berries have caused the death of an adult. and recovery has followed fifty. Children are less susceptible to the action of belladonna than adults; recovery in children has followed thirty berries. Belladonna applied externally, either as a plaster or in the form of the extract, has often produced toxic effects. Two grains of atropine have caused death; recovery has followed five and a half grains. A child, two and a half years old, recovered from a quarter of a grain of atropine.

Post-mortem Appearances.—No characteristic indications are afforded after death from belladonna poisoning unless the berries have been eaten, when fragments of them, or their seeds, may be found in the intestinal canal.

Tests.—A drop of a solution containing atropine, even only in infinitesimal amount, if instilled into the eye of a young cat, causes dilatation of the pupil. This test may be used to confirm diagnosis; a drop of the patient's urine will dilate the pupil of a kitten if dropped into its eye.

HENBANE and STRAMONIUM produce toxic symptoms resembling those produced by belladonna. Another mydriatic, DUBOISINE, acts similarly. The active principle in each case is isomeric with atropine.

The *treatment* is the same as for atropine. The fatal doses are not known. One-eighth of a grain of hyoscyamine, swallowed along with one-eighth of a grain of morphine sulphate, caused death. The hypodermic injection of one-thirtieth of a grain of hyoscine caused severe symptoms, followed by recovery. Seventeen or eighteen grains of extract of stramonium caused death. The instillation of one-hundredth of a grain of duboisine sulphate into the eyes produced severe toxic symptoms, followed by recovery.

No characteristic post-mortem appearances are produced by any of these atropaceous plants.

INDIAN HEMP (*Cannabis Indica*) is a hypnotic which, in the form of *haschisch*, is used in the East to procure sensuous hallucinations. It has often caused toxic symptoms and, occasionally, death, but the fatal dose is not known. The treatment would be the same as for opium.

GELSEMIUM.—Poisoning by yellow jasmine is usually due to excessive medicinal doses, or to idiosyncrasy on the part of patients for whom it has been prescribed. The symptoms are:—Giddiness, loss of speech, clonic spasms, un-

certainty of movement of the limbs, unconsciousness, and dilatation of the pupils. The treatment consists in evacuating the stomach with tube or emetic, and in the subsequent administration of stimulants, with the application of external warmth and artificial respiration if needed. Hypodermic injections of one-twentieth of a grain of atropine, or one-thirtieth of a grain of strychnine, may be given.

Cocaine.—This alkaloid has frequently given rise to dangerous symptoms when used to procure local anæsthesia. The symptoms vary somewhat. There may be clonic spasms, or convulsions, unconsciousness, rapid pulse and respirations, dilatation of the pupils, with delirium in some cases. The treatment consists in washing out the stomach, if the poison was swallowed, in administering stimulants and also chloroform vapour, if the clonic spasms interfere with respiration, in which case artificial respiration may be required. If the cocaine was injected subcutaneously the treatment is the same except as regards washing out the stomach. Ten grains of cocaine hydrochlorate caused the death of an adult within fifty minutes after the poison was swallowed; recovery has taken place after upwards of forty grains. Death has been caused by the injection of twenty minims of a 4 per cent solution of the hydrochlorate into the urethra. Three and a half grains of cocaine injected into the breast of a woman preparatory to an operation caused immediate epileptoid convulsions, and death in twenty minutes. In almost all fatal cases death occurred within an hour, and in most cases within ten or fifteen minutes.

Chronic cocaine-poisoning is an analogue to chronic morphinism. It is initiated by the medical administration of cocaine by hypodermic injection, and by nasal and laryngeal insufflation of atomised solutions. The *habitué* frequently adheres to the original method, so that if the drug was prescribed for naso pharyngeal treatment, he will continue to take it in the same way, many times a day, for an indefinite period. Mental apathy and moral degeneration result, and the patient is miserable unless he constantly resorts to the one thing which he has come to regard as necessary to his existence. Anomalous pains and disorders of the digestive organs, with corresponding emaciation, occur. A peculiar disturbance of sensation is often produced by the habitual use of cocaine: the patient feels as though grains of sand, or small round bodies, or in some instances worms, were under the skin. The treatment, obviously, is withdrawal of the drug. To accomplish this it may be necessary to place the patient, temporarily, under surveillance.

Tobacco.—Nicotine, the active principle of tobacco, is one of the few liquid alkaloids; it paralyses the cardiac nerves, and causes contraction of the peripheral blood-vessels. Acute poisoning by tobacco is usually accidental. It has occurred from the injection of an infusion into the rectum to destroy worms; from the application of an infusion to the skin in order to kill parasites; and, as a practical joke, from pouring the juice out of a well-smoked pipe into beer, or spirits, about to be drunk by a partially inebriated person. In one notable instance nicotine was used homicidally, the victim dying five minutes after the poison was administered. Severe toxic effects have been produced by smoking tobacco—on one occasion, at least, ending fatally. When the poison has been swallowed the treatment consists of washing out the stomach, or in administering an emetic, and then placing the patient in the recumbent posture, giving stimulants, promoting external warmth and, if necessary, performing artificial respiration. One twenty-fifth of a grain of strychnine may be injected subcutaneously with advantage, and repeated if required. There are no characteristic post-mortem appearances: the odour of the poison may be perceptible on opening the stomach, the mucous membrane of which may be injected or ecchymosed.

Chronic tobacco-poisoning is due to excessive or prolonged smoking of strong tobacco, to chewing tobacco, to taking snuff, and to the inhalation of fine particles of tobacco by workers in tobacco manufactories. The symptoms comprise dyspepsia, chronic catarrh of the mucous membrane of the pharynx and trachea, blunting of the senses of taste and smell, irregular action of the heart, faintness, tremor, and amblyopia. The symptoms that usually cause the patient to seek advice are the excessive and irregular action of the heart and the defect of vision. Tobacco amblyopia is probably due to pyridine, or to picoline, as much as, or more than, to nicotine, which is decomposed when the tobacco is burnt. With rare exceptions tobacco-amblyopia is only met with in old smokers—those who have indulged in the habit freely for fifteen or more years; so that it is uncommon before forty years of age. It is due to the presence of a central scotoma, especially for red and green; the periphery of the field of vision remains unaltered. The symptoms of chronic tobacco-poisoning pass away on cessation of the habit which caused them; the amblyopia may not disappear for several months. The administration of strychnine is said to be helpful.

Spotted Hemlock (*Conium maculatum*), like tobacco, yields a liquid alkaloid—conine—which possesses a characteristic mousy odour. It paralyses the motor nerve-terminals, and subsequently the motor centres of the cord and brain, death being due to respiratory paralysis. The symptoms produced comprise a burning sensation with a feeling of constriction in the throat, nausea, vomiting, pain, oppression in the stomach, and diarrhœa. The pupils dilate and the surface becomes cold; the respiratory move-

ments become slower and feebler, and there may be delirium and coma. When the paralysis reaches the respiratory muscles the patient becomes cyanosed, and dies asphyxiated, death being frequently preceded by convulsions. The treatment consists in evacuating the stomach and administering stimulants along with the application of external warmth. Artificial respiration will be needed, and should be perseveringly kept up; by this means life may be saved when the condition appears to be hopeless. There are no characteristic post-mortem appearances—simply the indications of death from asphyxia.

FOXGLOVE.—This is a true heart poison; in fatal cases the heart usually ceases to beat before the respiration ceases, although the respiration rate may be reduced in frequency shortly before death. In addition to the specific action of digitalis the poison acts as an irritant to the digestive canal, causing pain with a feeling of oppression about the stomach, nausea, often obstinate vomiting, and possibly diarrhœa, with colicky pains in the abdomen, and much thirst. The specific effects of the poison are shown by giddiness, faintness, headache, clammy, cold surface, especially of the limbs, collapse, and various nerve disturbances such as dimness of vision, noises in the ears, hallucinations, and delirium. These symptoms are due to the extremely low arterial tension which is further evinced by the feeble, fluttering action of the heart; any movement towards the upright posture is immediately followed by threatened or actual syncope, which may be instantaneously fatal. The breathing is slow and of the sighing type. A tendency to drowsiness or to coma is common, and cyanosis with asphyxic convulsions may precede death.

Treatment.—Wash out the stomach or give a stimulating emetic such as a dessert-spoonful of mustard in warm water. As soon as the stomach is freed from the poison stimulants must be freely given, external warmth applied, and the recumbent posture rigorously maintained for several days. Friction and the application of mustard to the region of the heart, with the administration of hot coffee, to which brandy may be added, are helpful. If the vomiting is prolonged the coffee and the stimulants may be given by the rectum, or ether may be injected subcutaneously. An occasional fragment of ice will help to check the vomiting. It must be borne well in mind that the condition of the heart renders the patient extremely liable to fatal syncope for several days after the immediate effects of the poison have passed off; hence the absolute necessity for keeping him closely confined to bed until the dangerous period is passed.

The *fatal dose* of digitalis is uncertain: nine drachms of the tincture have caused death, and recovery has taken place after more than three ounces. Dangerous symptoms have been caused by the medicinal preparation called digitalin, which is an uncertain drug of no fixed chemical constitution, its activity being determined by the process by which it is prepared. In fatal cases of poisoning by foxglove death may take place within twenty hours, or it may be much longer delayed. There are no characteristic post-mortem appearances; some indications of the action of a gastro-intestinal irritant may be present.

COLCHICUM, in poisonous doses, causes a burning pain in the throat and down the œsophagus to the stomach, followed by copious vomiting and purging, with violent griping and strangury. There is intense thirst, with symptoms of collapse, the appearances being those previously described as due to a typical irritant poison. After the bowel-contents have been evacuated the alvine discharge becomes serous and may be blood-stained. Muscular spasms, or, occasionally, convulsions may occur. The mind usually remains clear until death approaches, although, exceptionally, stupor attacks the patient at an earlier stage.

The *treatment* consists in emptying the stomach by the tube or an emetic, and then giving strong tea and brandy, with friction, external warmth, and, if necessary, artificial respiration. If the vomiting be excessive an ether injection should be substituted for the brandy, unless the latter is administered by the rectum. A hypodermic injection of a third of a grain or half a grain of morphine will probably be required to allay the intense griping.

Fatal Dose.—Three and a half drachms of colchicum wine have caused death, and recovery has followed ten drachms. Six grains of colchicine—the active principle of colchicum—caused the death of an adult in thirty-one hours. Death from colchicum usually takes place within thirty hours; it has occurred as early as seven hours, and has been delayed for seven days. Beyond the possible presence of signs of inflammation of the intestinal mucous membrane there are no post-mortem appearances.

HELLEBORE contains a number of alkaloids of which one—*veratrine*—produces a very irritating effect on the nasal mucous membrane.

Symptoms.—An acrid, burning sensation beginning in the throat and extending to the stomach is quickly experienced, which is followed by vomiting and possibly by diarrhœa. There is great thirst, the pulse is feeble, the respirations are slow, and rapid collapse occurs. There may be disturbances of sensation and delirium.

Treatment.—Evacuate the stomach and administer hot coffee and stimulants. Artificial respiration, with the application of external warmth and friction, and the maintenance of the recumbent posture, may severally be required. There are no characteristic post-mortem appearances.

MONKSHOOD.—The alkaloid of this plant—*aconitine*—is probably the most virulent poison known. It most unfortunately happens that several preparations are known as aconitine, and that they differ enormously in potency. English and French aconitines are much more powerful than German aconitine.

Symptoms.—Tingling, followed by numbness, is felt in the lips, mouth, and throat soon after the poison is swallowed; the tingling subsequently spreads all over the body. Nausea, usually followed by vomiting and sometimes by purging, with pain in the stomach and abdomen, occurs. Imperfect vision, restlessness, anxiety, twitching of the muscles, darting nerve pains in the legs, prostration, feeble intermittent pulse, lowering of the temperature, and collapse with possibly convulsions, precede death.

Treatment.—Empty the stomach by tube or emetic. Administer brandy by mouth or rectum, or ether subcutaneously. External warmth, friction, artificial respiration, and the recumbent posture will be required.

Fatal Dose.—One drachm of aconite root, two grains of the pharmacopœial extract, one ounce of the liniment, and one drachm of the tincture have each caused death. The fatal period is from three-quarters of an hour to twenty hours; the usual period being from three to four hours.

The post-mortem appearances are not characteristic.

PILOCARPINE, the chief active principle of jaborandi, is used medicinally in order to promote the action of the skin. It also stimulates the secretion of mucus, and in this way occasionally produces symptoms of suffocation by causing sudden blockage of the bronchial tubes. One-sixth of a grain injected hypodermically has given rise to alarming symptoms. Vomiting, purging, and pain in the head are also produced. The effects may be combated by hypodermic injections of one-fiftieth of a grain of atropine.

PHYSOSTIGMINE, or ESERINE, an alkaloid of the Calabar bean, is used for ophthalmic purposes in order to contract the pupil. It has produced toxic effects when a stronger solution than usual has been dropped into the eye. When the bean is swallowed, pain in the stomach, vomiting, small slow pulse, salivation, thirst, cold moist surface, and occasionally diarrhœa, are produced. The *treatment* would be to evacuate the stomach if the poison was swallowed, to use artificial respiration if necessary, and to administer one-fiftieth of a grain doses of atropine until the pulse-rate is quickened. Hypodermic injections of strychnine, a fiftieth to a twenty-fifth of a grain, are useful.

SANTONIN occasionally produces symptoms of poisoning in children to whom it has been given as an anthelmintic. A common symptom, often seen without other ill-effect, is what is called "yellow vision," probably due to primary stimulation with subsequent paralysis of the violet-seeing retinal elements. Singing in the ears, dizziness, pain in the stomach, vomiting, stupor, convulsions, and a tendency to asphyxia, may also occur. The urine is of a dark yellow colour, and, on the addition of sodium hydrate, becomes red. If rhubarb be present in the urine it yields the same reaction; but if milk of lime be subsequently added and the urine is filtered, the filtrate is colourless, whereas urine containing santonin does not lose its colour when similarly treated. Two grains of santonin caused the death of a child five and a half years old; recovery in a child has followed ten grains. The treatment is to evacuate the poison, to give stimulants, and, if convulsions occur, potassium bromide.

ABORTIFACIENTS

Among the substances which are taken by pregnant women with the object of procuring abortion are: savin, yew, pennyroyal, tansy, saffron, colocynth, croton oil, gamboge, aloes, hellebore, ergot, cantharides, phosphorus, and salts of iron, lead, mercury, and arsenic. With one exception none of these substances has a specific action on the healthy uterus, but abortion may follow their administration under one or both of two conditions: that there is an unhealthy relation between the uterus and its contents; or, that the drug is administered in such doses as to produce severe symptoms of general poisoning. It is well known that pregnant women suffering from syphilis, and from other conditions which are not well understood, abort very readily and from trivial causes; in such women a smart purge may induce abortion, so that with them many of the above-named substances, which are cathartics and irritants, are competent to cause abortion without being administered in poisonous doses. It is equally well known that perfectly healthy pregnant women may be subjected to considerable and even excessive violence from accidental causes without the progress of gestation being interfered with. Such women will not respond to any so-called ecbolics unless the drug is administered in sufficiently poisonous doses as to seriously derange the entire organism, often with fatal results. It not unfrequently happens, when powerful drugs are taken with the object of causing abortion, that the woman is fatally poisoned, and dies undelivered; in other cases abortion is speedily followed by the death of the mother. The inclusion of the salts of iron in the list of abortives is due to false deduction. Women know that doctors often prescribe iron in anæmic amenorrhœa, and have drawn the inference that the iron acts directly on the uterus, and compels it to resume its monthly function, and therefore that iron will re-establish menstruation which by any

cause has been arrested. Even among medical men there is often an indefensible prejudice against the administration of iron to pregnant women; if, for the purpose of medicinal treatment, the health of a pregnant women calls for the administration of iron, there is no reason why it should not be given. Of the general poisons which indirectly cause abortion, two are exceptionally active—phosphorus and lead; with one exception the vegetable ecbolics are simply purgatives and irritants. The only substance in the list of ecbolics that, as such, merits discussion is ergot. This is a drug that undoubtedly is capable of acting directly on the uterus; it has, however, but slender claims to be entitled an abortifacient. In the later months of pregnancy, when uterine action has once commenced, the administration of ergot induces powerful tonic contraction of the uterine musculature, whether, at the moment, natural contraction is in progress or is not; but if ergot is given for the purpose of initiating labour it will probably not be successful unless it produces profound disturbance of the entire organism. In the early months of gestation (when criminal abortion is resorted to, the uterus at the time being passive) the administration of ergot is still less likely to induce uterine action, unless acute ergot-poisoning is produced; and even then pregnant women have died from acute ergot-poisoning (the drug having been taken for the purpose of inducing abortion) without abortion taking place.

The *symptoms of acute ergot-poisoning* are: pain in the stomach, vomiting, a feeling of weight in the cardiac region, cramps, dyspnœa, numbness and tingling in the limbs, beginning at the fingers and toes, followed by extreme coldness, delirium, coma, and convulsions. Subcutaneous and submucous hæmorrhages, with the appearance of blood in the vomit and the motions, not unfrequently occur. The treatment is to empty the stomach and the bowels, unless the spontaneous evacuation has taken place. Stimulants and external warmth will be required. Inhalation of amyl nitrite or the administration of nitroglycerine by the mouth ($\frac{1}{100}$ to $\frac{1}{50}$ of a grain) may be tried.

Post-mortem Appearances.—The surface may be somewhat jaundiced, and there may be subcutaneous hæmorrhages. Extravasation of blood may also be found internally, on the surface and into the substance of the viscera. The liver and kidneys are often pale and bloodless, and they show the presence of fatty changes, sometimes very advanced, so much so as to give rise to suspicion of phosphorus-poisoning. When ergot has been taken to produce abortion, the fœtus, on several occasions, has been found in the uterus after the death of the mother.

As regards abortifacients generally, the inference to be drawn is that there is no known drug which will infallibly induce abortion without imperilling the life of the mother.

Poisoning by Food-Stuffs

Many food-stuffs may become poisonous owing to partial changes in their composition, caused by the action of micro-organisms; others are hurtful by reason of the unwonted excess of a normal constituent.

Vegetable Foods which may cause Poisoning

Fungi (Mushroom-poisoning).
Grain, especially rye (Ergotism).
Vetch (Lathyrism).
Maize (Pellagra).
Potato.

Fungi.—Many fungi are inherently poisonous in the same sense that hemlock is; others, without possessing any essential toxic principle, occasionally act as poisons. The only fungi eaten in England are the common mushroom (*Agaricus campestris*) and the champignon (*Agaricus oreades*). On the Continent many other varieties are used as food. Fungi, being of extremely rapid growth, quickly undergo changes after being gathered which may convert those of a harmless variety into poisonous bodies; consequently, only fresh fungi should be eaten. Mushrooms which have been cooked should not be eaten after having been put aside and warmed up again.

The symptoms of fungi-poisoning are *gastro-enteric* and *neurotic*; usually both occur, with relatively varying intensity, in the same patient. *Gastro-enteric symptoms* may not appear for eight or ten hours, or more, after the fungi are eaten. A painful sensation develops in the stomach and abdomen, and the latter becomes hard and tender; then follow nausea, vomiting, and diarrhœa; the motions may be serous, and may contain flakes of lymph and sometimes blood; the diarrhœa is often persistent. The other symptoms of irritant poisoning, tending to collapse, with occasionally jaundice, follow. *Neurotic symptoms* comprise muscular twitching, tetanic spasms, convulsions, delirium, disorders of vision, dilatations of the pupils, and coma. Sometimes the symptoms are exclusively neurotic; more frequently they are associated with gastric symptoms. The fly-fungus (*Amanita muscaria*), so-called because it acts as a fly-poison, causes delirium, clonic spasms, dilated pupils and quick pulse; in its mildest form the condition resembles that of alcoholic intoxication. The active principle of this fungus (*muscarine*) produces somewhat different symptoms, the assumption being that a second active principle, of antagonistic properties, is also present in the fungus. *Amanita phalloides* is another poisonous fungus, which, in addition to producing other symptoms, acts as a blood poison and causes fatty changes, especially in the liver, along with a hæmorrhagic tendency,

closely resembling the effects of acute phosphorus-poisoning. The fungus *Helvella esculenta* acts much in the same way.

Treatment of Fungi-Poisoning.—Empty the stomach by means of an emetic, and the bowels with castor oil, and then treat the symptoms. External warmth and stimulants will be required, and morphine if the gastro-enteric symptoms are violent. If the symptoms partake of the muscarine type, atropine in one-twentieth of a grain doses may be given subcutaneously.

The *post-mortem appearances* vary in accordance with the action of the fungus; indications of gastro-enteritis with erosions and submucous hæmorrhages may be present, and the liver and other viscera may show fatty changes, which in the case of the liver may be very pronounced. If the symptoms during life were purely neurotic the post-mortem appearances will be indefinite.

Ergotism, or chronic ergot-poisoning.—Ergot is the mycelium of a fungus—*Claviceps purpurea*—which grows on various species of grain, especially on rye. It is mostly met with in Russia and Germany, where, after a rainy summer, widespread epidemics of ergotism often occur, which are due to the inhabitants eating bread made from the diseased grain.

The *symptoms* commence with disturbance of the digestive tract; nausea, occasional vomiting, a sense of oppression in the stomach, loss of energy, and either constipation or diarrhœa. Subsequently local symptoms manifest themselves, either in the form of gangrene, or of spasmodic contraction of muscles, with other nervous disturbances; both these types may occur in the same patient.

Gangrenous ergotism is initiated by patches of anæsthesia or by redness and burning of the skin, which is followed by gangrene, usually of the dry type, in the fingers and toes; this may spread as far as the elbows or knees, but the trunk is rarely affected. At the line of demarcation the gangrenous part separates by slow ulceration, unless it is removed by surgical operation. Very exceptionally the skin alone is attacked.

Spasmodic ergotism is preceded by various paræsthesiæ, such as a creeping sensation, beginning in the fingers and toes, or there may be anæsthesia, followed by spastic contraction of the muscles, by which the fingers and toes are drawn together and, along with the hands, are flexed—presenting the appearance of the clenched fist with the thumb drawn in towards the palm; the feet are extended, the heel being so powerfully drawn up as to cause the leg and foot to form a straight line. The dorsal muscles may also be contracted so as to produce opisthotonos. The spasms are excessively painful, and in the intervals the patient suffers from an unbearable burning pain in the affected limbs. The spasms may be tetanic in character, and the diaphragm may participate, so that respiration is impeded; sometimes the contractions are clonic, resembling epilepsy. Spastic contraction of the bladder, diplopia, deafness, aphasia, and changes in the visual field for colour, have been observed. The pulse is quick, and in the later stage is feeble. Psychical disorders, delirium, mania, stupor, and ataxic symptoms have been recorded; the knee-jerks are absent in these cases, and sclerosis of the posterior columns of the cord, implicating the root-zones, has been found. Both forms of ergotism are probably due to persistent contraction of the smaller arteries, which deprives the tissues implicated of their blood-supply.

The *treatment* consists in preventing further use of contaminated bread, and in dealing with the symptoms, medically and surgically.

Lathyrism.—Several species of vetch—*Lathyrus sativus*, *L. cicera* (chick-pea), and *L. Clymenum*—may give rise to poisoning, cases of which have occurred in France, Spain, Africa, and India, through eating grain derived from one or other of the above-named species. The symptoms begin with pain and stiffness in the back, stiffness with an intolerable creeping sensation in the limbs, tremor of the hands, and spastic gait; occasionally the gait is ataxic. The knee-jerk is exaggerated. Disorders of sensation, hyperæsthesia and paræsthesia, followed by anæsthesia, may occur. Usually the legs only are affected; the arms, if at all, participate but slightly. But little is known of the morbid anatomy of lathyrism; the symptoms point to degenerative changes in the lateral columns of the cord; but any changes that take place cannot be very profound, inasmuch as after the grain is no longer eaten the symptoms usually subside in time, the course of lathyrism being much less severe and prolonged than that of the antecedently described disease—ergotism, or of the disease next to be described—pellagra.

Pellagra (*pelle*, skin, and *agra*, rough) is a disease produced by eating diseased maize, which in some countries is used as a substitute for corn. Pellagra is met with in France, Spain, Italy, Roumania, Austria, Corfu, Mexico, and Egypt. It occurs in the early part of the year and subsides in the autumn. It is rare in children.

General Symptoms.—Headache, depression, paræsthesiæ, cramps, vertigo, and dyspepsia, with the tongue denuded of epithelium, may precede the characteristic manifestations of the disease. These comprise erythema, with an œdematous condition of the skin, and an intolerable burning and itching on those parts of the body which are exposed to the sun. The acute stage lasts about a fortnight, when it subsides, leaving a dry, thickened, scaly, pigmented, psoriasis-like skin. The hair and nails are not affected. There is usually a recurrence each spring. After repeated attacks (not until after the third

or fourth) the patient becomes melancholic; he suffers from delusious, and eventually from dementia. He may have epileptic seizures. The knee-jerk is exaggerated, ankle clonus is present, the condition resembling that of spastic paraplegia. Sensation is not affected. After many years the skin shrivels and the muscles atrophy; the knee-jerk is then lost.

The *morbid anatomy* comprises degeneration of the pyramidal tracts, mostly in the lower dorsal region; degeneration of Goll's column and also of Burdach's in the upper dorsal region, and there may be sclerosis of the lateral columns. Pigmentation of the anterior corneal cells has also been observed. In the very acute form of the disease diffuse myelitis is reported to have occurred.

POTATO, or *Solanum tuberosum*, has occasionally given rise to poisoning owing to the presence of an excessive amount of *solanine*, a poisonous constituent, especially of the seed-potato. It is probable, in cases of potato-poisoning, that the tubers which caused the mischief were sprouting, or that some young green potatoes were mixed with them.

The *symptoms* comprise frontal headache, colicky pains in the stomach and bowels, with swelling and tenderness of the abdomen, vomiting, diarrhœa, shivering, sweating, depression, slight stupor, oral and visual disturbances, congestion followed by pallor of the face, blueness of the lips, widely dilated pupils, initial acceleration with subsequent retardation of the pulse, elevation of temperature, syncope, and convulsions. In one case death took place on the third day.

The *treatment* is to clear the bowels of the potatoes and to give morphine, if necessary, along with stimulants and external warmth.

ANIMAL FOODS WHICH MAY CAUSE POISONING.—Meat, fish, sausage (botulism or allantiasis), milk, cheese.

Meat, from a variety of causes, may injure the health of those who eat it. The animal from whence the meat is derived may have been the subject of parasitic disease, such as *Cysticercus cellulosæ*, which affects the pig, or the *C. bovis* of the cow; giving rise respectively to *Tœnia solium* and *T. mediocanellata* in man. The *Trichina spiralis* is another animal parasite which renders meat affected with it very dangerous to those who eat it, the disease produced being known as *trichiniasis*. This parasite is most common in pork—a meat that in various forms is often eaten in an imperfectly cooked state. The symptoms produced are: a feeling of general prostration, pain, vomiting, diarrhœa, elevation of temperature, and profuse perspiration, followed by symptoms due to the presence of the parasite in the muscles; local pain, tenderness, and swelling, with rigidity of the affected muscles. The early symptoms of trichiniasis sometimes resemble those of enterica; at others the onset is sudden, and the condition might be attributed to cholera or to irritant-poisoning. Death, from ulceration of the intestine, from pneumonia, or from exhaustion, may result in four or five weeks, or earlier. The transmission of *anthrax* and of *tubercle* to human beings who eat the flesh of animals thus affected is a question that is still *sub judice*.

Apart from the specific conditions above described, meat may become dangerous as food: (*a*) from the presence of disease in the animal from which the meat is derived; (*b*) from micro-organisms which develop in the meat subsequent to slaughter; (*c*) from the presence of toxalbumoses or of ptomaines. The condition due to (*a*) and (*b*) is that of a true infection; that due to (*c*) is one of poisoning without infection. It might be supposed that the distinction between these two classes would always reveal itself—in the one case by a prolonged interval between the reception of the diseased meat and the commencement of the symptoms, and in the other by their rapid onset. Whilst it is true that in the cases of (*a*) and (*b*) a more or less prolonged period of incubation elapses after the meat is eaten before any indications manifest themselves, it is also equally true that in (*c*) the symptoms, though usually speedily developed, may be delayed for a considerable time.

The symptoms met with in (*a*) and (*b*) are those of an infectious disease. They include headache, anorexia, foul tongue, rigors, constipation followed by diarrhœa, pains in the back and limbs, elevation of temperature, photophobia, delirium, skin eruptions, meteorism, and enlargement of the spleen. The post-mortem appearances have a close resemblance to those of enterica—infiltration, ulceration, and sloughing of Peyer's patches, hæmorrhage into the bowel, enlargement of the spleen, with possibly depôts of pus. Sometimes the symptoms resemble those of the next group. The symptoms met with in (*c*) are those of acute gastro-enteritis—violent vomiting, purging, prostration, cramps in the legs, and collapse; the temperature is usually subnormal, but it may be elevated. The post-mortem appearances are those produced by gastro-enteritis, with hæmorrhages into the intestinal mucous membranes; the spleen is frequently enlarged, and Peyer's patches may be infiltrated.

Special symptoms occasionally ensue, such as dizziness, dryness of the mouth and throat, dyspnœa, heart-failure. When a number of persons have eaten of the same poisonous meat they may not all suffer in the same way; not only may some of the victims be much more severely attacked than others, but the character of the symptoms may also differ in the various individuals.

The substances which cause meat-poisoning (*c*) are frequently spoken of as "ptomaines";

but, for the most part, they would be more correctly designated "toxalbumoses"; that is, proteid substances which have been acted on by bacteria in such a way as to develop intensely poisonous properties. Toxalbumoses are unstable, and their constitution is but little understood. Ptomaines—a later product of bacterial action—are basic substances which possess a definite chemical composition. A ptomaine may or may not be poisonous; many ptomaines are practically harmless. All these substances—toxalbumoses and ptomaines—when present in actively poisonous forms, are liable to lose their toxic properties as decomposition advances: a meat pie, for example, which caused severe poisoning in human beings, and which killed mice fed on it, after being kept a little longer became putrid, and at the same time lost its poisonous character, so that it could be eaten by mice with impunity. Poisonous bacterial products, though unstable, are sometimes very resistent of a high temperature—that of boiling water, for instance; so that cooking cannot be depended upon invariably to render the meat harmless. The same applies to micro-organisms and their spores that may be present in meat; the outermost layer of the meat may have its nocuous properties destroyed by boiling or roasting, but the central portion may never reach the requisite temperature. Salting and smoking do not, or may not, remove poisonous properties that are present in the meat; neither does the exposure of the meat to a very low temperature, as in an ice-safe, necessarily inhibit bacterial decomposition. It is not essential that meat should acquire a suspicious odour in order to become poisonous; certain micro-organisms render meat dangerous without producing the ordinary accompaniments of decomposition. The kinds of meat which most commonly cause poisoning are: pig's flesh, veal, beef, various kinds of meat pies, potted meat, tinned meat, sausages, and brawn.

Allantiasis or *botulism* are names given to sausage-poisoning, due to eating "German sausage," which consists of finely divided meat mixed with other substances, imperfectly cooked, and frequently formed into large masses. A bacterial decomposition product, endowed with special toxic properties, is not unfrequently formed, to which the name *ptomatropine* has been given on account of a certain resemblance between the symptoms it produces and those produced by atropine.

The *symptoms* of allantiasis do not usually develop for from twelve to twenty-four hours after the poisonous sausage has been eaten. The patient has a foul tongue, with dryness in the throat and suppression of the salivary secretion, which causes the voice to be hoarse and the swallowing to be difficult or impossible; there may be a dry laryngeal cough. Nausea and vomiting with diarrhœa, or perhaps obstinate constipation with colic, follow. The pulse is rapid, the pupils are dilated and reactionless, and there may be ptosis and diplopia. Affections of the special senses, as sounds in the ears and dizziness, are frequent. In fatal cases death occurs in four or five days; occasionally its occurrence is earlier. There are no characteristic post-mortem appearances. The *treatment* is to evacuate the bowels and stomach, if this has not previously been done spontaneously, and to give hypodermic injections of pilocarpine hydrochlorate (one-third to half a grain), the patient being enveloped in blankets wrung out of hot water.

Fish.—Some fish are inherently poisonous; they are mostly found in tropical waters; others, such as mackerel, carp, barbel, and herrings, are liable to become poisonous; mackerel and herrings are especially prone to develop poisonous properties after they are caught. Something depends on the treatment of the fish; herrings, if they are not "gutted" immediately after they are removed from the water, quickly become unfit for food. Caviare, the roe of herrings and of other fish, dried cod-fish, preserved anchovies and sardines, have caused poisoning; a stale red herring caused the death of a man from gastro-enteritis. Shell-fish excepted, mackerel produces the greatest number of cases of fish-poisoning in this country. The symptoms of fish-poisoning may be purely gastro-enteric, or they may be partially or entirely neurotic—swelling of the tongue and face, intense itching of the skin, coldness of the hands and feet, thirst, dyspnœa, and convulsions. Tinned salmon and sardines have caused fatal poisoning, the post-mortem appearances indicating the action of a most potent irritant; in one case the stomach and intestines were so intensely inflamed as to be almost gangrenous. Sometimes the mischief produced is of such a character as to indicate the action of micro-organisms rather than of a pre-formed poisonous substance.

Shell-Fish, as mussels and oysters, may, during the lifetime of the fish, develop toxines which render them poisonous. This occurs if the fish are placed in stagnant water, especially if it is contaminated by decomposing organic matter; so that mussels obtained from docks, harbours, the mouths of rivers, or other places where there is a restricted tidal interchange, are liable to be poisonous; indeed almost all the bad cases of mussel-poisoning are due to this cause. A mild form of mussel-poisoning is characterised by the appearance of an urticarial or an exanthematous eruption on the body, often associated with oppression in the chest and difficulty of breathing; these symptoms usually pass off in a short time. In the severer forms there is gastro-enteritis, and in the most dangerous of all, paralysis. A man ate uncooked a large quantity of mussels obtained from the bottom of a graving dock; he became

unconscious and livid, with imperceptible pulse and widely dilated pupils, paralysis of the limbs, and slow, gasping respirations once or twice in the minute, death taking place in twelve hours from respiratory paralysis; the heart continued to beat for many hours after voluntary respiration had ceased. In this case there was neither vomiting nor purging. Death may occur very rapidly without any post-mortem indications. A man ate some mussels and very soon after his face became fiery red and he died in an hour; at the autopsy no signs of gastritis were found, nor was there any obvious cause of death. Oysters also, under the above-named conditions, may develop toxines, which may produce either gastro-enteric or purely neurotic symptoms, or a combination of the two. In the neurotic type of shell-fish poisoning death takes place from respiratory paralysis. In addition to causing poisoning shell-fish may convey disease. Oysters and mussels which have been placed in water contaminated with sewage containing typhoid bacilli are capable of communicating enterica to those who eat the fish in a raw state.

Milk may give rise to illness, either by being contaminated with pathogenic micro-organisms, or from the formation in it of decomposition products. Instances of the first-named group have occurred in which the milk was found to contain streptococci, and bacilli of the type of *B. coli communis*, which caused an outbreak of disease chiefly among children who had been fed with the milk. In other instances milk has conveyed typhoid fever, either on account of the milk-cans having been washed with germ-laden water, or by transference of germs to the milk from the hands of dairymaids who were nursing cases of typhoid fever. Cows are subject to a number of diseases which may render their milk dangerous to human beings. When milk is thus contaminated it is by no means easy to destroy the germs by subjecting the milk to heat; the spores of many micro-organisms resist a temperature of 212° F. maintained for three-quarters of an hour or more; some anaerobic bacteria are even still more resistent.

When milk, derived from healthy cows, is not cooled immediately after milking, especially in hot weather, it is liable to develop early decomposition products which produce nausea, vomiting, cramps, diarrhœa, and collapse. By exposing the milk, warm from the cow, in shallow basins surrounded with cold water or, if necessary, with ice, this danger may be avoided. The greatest risk is incurred when the warm milk, in hot weather, is conveyed to a distance by cart and rail; the continuance of an elevated temperature along with the constant agitation to which the milk is subjected are very liable to promote changes which lead to the formation of toxic products.

The *ice-creams* which are sold in the streets not unfrequently cause illness on account of the decomposition products they contain. In some instances children that have eaten such creams are attacked with symptoms which closely resemble those of meningitis—restlessness, dulness, insensibility, and retraction of the head; the symptoms usually subside within forty-eight hours. In other instances these ice-creams are profusely contaminated with micro-organisms, some of which may be pathogenic; this is due to carelessness and to the extreme insanitary conditions under which such creams are frequently made.

Cheese.—Instances of poisoning by cheese have occurred from time to time; the symptoms produced being: pain in the stomach, violent vomiting, diarrhœa, and cramps in the legs; the tongue, at first coated, becomes red and dry; the pulse is weak and irregular, and the face is pale and cyanotic. Old, decayed cheese has caused colic, diarrhœa, diplopia, precordial pain, and collapse.

The poisonous agent in cheese has been variously described as *tyrotoxicon*, a substance which closely resembles diazobenzene; as a toxalbumose, and as a tertiary amine — trimethylamine.

Toxicomania.—A diseased craving for poisons, or (sometimes) toxiphobia (*q.v.*).

Toxicosis. — A poisoned state or the condition resulting from the presence of toxic, *i.e.* poisonous, materials in the system; if these materials are developed in the system the state is called *autotoxicosis* or autointoxication, and if they are introduced from without it is named *exogenic toxicosis*.

Toxine.—A poisonous ptomaine or nitrogenous compound produced by animal or vegetable cells; it is analogous to a vegetable alkaloid; a toxalbumin. An *animal* toxine is one produced in animals by the cells of the body, *e.g.* snake venom; bacteria by their activity (as in diphtheria) produce *bacterial* toxines, and if the toxines are contained in the bacteria they are called *intra-cellular*, while if they are found in the surrounding medium they are *extra-cellular*. *See* Immunity (*Toxic Filtrates*); Immunity, Recent Views on; Tetanus (*Toxine*); etc.

Toxiphobia.—Morbid fear of being poisoned.

Toxoid.—A transformation product of a toxine no longer possessing toxic powers; a toxine possessing its haptophore group but having lost its toxophore one. *See* Immunity, Recent Views on.

Toxone.—A toxine incompletely combined with its antitoxine, and on that account

possessing toxic powers, although in a less degree than a toxine.

Toxophore.—The atomic group in a toxine molecule which produces the poisonous effect upon any cell with which it may unite; the union is effected by means of the other atomic group (*haptophore*), which anchors the toxophore to one of the receptors of the cell. *See* IMMUNITY, RECENT VIEWS ON.

Toynbee's Experiment.—The production of rarefaction of the air in the middle ear by swallowing with the mouth and nose closed.

Trabecula.—A band, usually fibrous in nature, which extends from the capsule of an organ into the interior of the same, and acts as a supporting structure. *See* PHYSIOLOGY, TISSUES (*Bone, Development and Structure*).

Trabs Cerebri.—The corpus callosum of the brain. *See* PHYSIOLOGY, CEREBRUM (*Corpus Callosum*).

Trachea, Affections of the.

MALFORMATIONS 80
INFLAMMATIONS 80
ULCERATION 81
PERFORATION 81
TUMOURS 81
TRACHEAL OBSTRUCTION 81
TRACHEAL RUPTURE 81
FOREIGN BODIES 82
TRACHEOTOMY 82

See also ANEURYSM (*Neck, Symptoms, Tracheal Tugging*); AORTA, THORACIC, ANEURYSM OF (*Symptoms, Respiratory, Deviation of Trachea*); ASPHYXIA (*Definition, Phenomena, Causes, etc.*); BRONCHI, BRONCHIAL GLANDS (*Pressure on Trachea*); BRONCHI, BRONCHITIS (*Treatment, Intratracheal Injections*); CAUTERY (*Red Heat in Tracheotomy*); DIPHTHERIA (*Intubation and Tracheotomy*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Examination of Heart, Tracheal Tugging*); LARYNX, EXAMINATION OF (*Examination of Trachea*); LARYNX, MALIGNANT DISEASE (*Treatment, Tracheotomy*); LUNG, TUBERCULOSIS OF (*Complications, Trachea*); LUNGS, SYPHILIS OF (*Trachea, Stenosis of*); MEDIASTINUM; STATUS LYMPHATICUS; SYPHILIS (*Visceral Trachea*); THYMUS GLAND; THYROID GLAND, SURGERY OF; TONGUE (*Glossitis, Treatment*); TONGUE (*Carcinoma, Treatment, Preliminary Tracheotomy*); TUBERCULOSIS (*Larynx and Trachea*).

MALFORMATIONS.—The chief malformations to which the trachea is liable are:—

1. The trachea is short and imperforate.
2. The trachea communicates with the œsophagus.
3. Fistula, by which the trachea communicates with the surface of the neck.

TRACHEITIS.—The two chief varieties of inflammation of the trachea are:—

1. Catarrhal tracheitis.
2. Diphtheritic tracheitis.

Catarrhal Tracheitis.—This is often associated with a similar condition in the larynx or bronchial tubes.

Causation.—Sudden alterations in the temperature of the air, the breathing of irritating vapours, and chill, are the most common exciting causes.

Morbid Anatomy.—There is swelling with hyperæmia of the mucous membrane. In severe cases ulceration even may be present.

Symptoms.—In acute catarrhal tracheitis there may be elevation of temperature with quickened pulse, while the patient complains of a tickling sensation in the trachea and of substernal pain. Cough is one of the most prominent symptoms, and is most marked when the patient lies down. It is at first dry and accompanied by very little expectoration, but afterwards the sputum becomes more abundant and more purulent. Nothing abnormal is heard by auscultation over the trachea.

Diagnosis.—This depends on examination. Thus, with the laryngoscope the presence of an accompanying laryngitis will be confirmed or contradicted, while if the glottis be widely open and a good view obtained, hyperæmia of the tracheal mucous membrane will be seen. By examination of the chest the presence or absence of bronchitis will be diagnosed.

In chronic catarrhal tracheitis there is little or no constitutional disturbance; but there is present a constant feeling of irritation in the trachea, with cough. The same physical examination must be made as in the case of acute affection.

Treatment.—At the commencement of an attack of acute catarrhal tracheitis the patient should, if possible, be put to bed and an aperient given, preferably calomel, followed by a saline. Given at this stage good results follow the administration of ℥xx. doses of vinum antimoniale, and, if there is much substernal pain, hot linseed poultices over the front of the chest give relief. The cough is best relieved by the frequent inhalation of the steam from a pint of hot water to which ʒj. of compound tincture of benzene has been added. As expectoration becomes more profuse and the cough loose, it is well to give a mixture containing ipecacuanha, iodide of potassium, ammonium carbonate, and senega. In the chronic condition tonics, such as iron and quinine, are indicated, and the patient should, if possible, have change of air. In obstinate cases a blister over the sternum is of great service.

Diphtheritic Tracheitis.—This nearly always occurs as an extension downwards of laryngeal diphtheria, but the deposit of diphtheritic membrane in the trachea is sometimes primary (*v.* article "DIPHTHERIA," vol. ii.).

Ulceration. — The varieties of tracheal ulceration are:—

1. *Simple ulcers* may form in the course of catarrhal tracheitis, and are caused by swelling and breaking down of the masses of lymphoid tissue.

2. *Syphilitis.*—In secondary syphilis mucous patches and superficial ulcers may form, but their presence will not usually be diagnosed.

In tertiary syphilis deep ulcers may form, and may cause necrosis of the cartilaginous rings. These ulcers heal under potassium iodide, but in their healing contract, and so cause a serious amount of tracheal stenosis. The lower part of the trachea is most commonly thus affected.

Symptoms. — The symptoms are those of chronic tracheitis. There is no sputum at first, but this afterwards becomes plentiful, muco-purulent, and streaked with blood. If situated high up the ulcers may be seen with the laryngoscope. The diagnosis generally depends on:—(*a*) the patient's history; (*b*) the presence of other lesions of a syphilitic nature; (*c*) the absence of tubercle bacilli from the sputum.

Treatment.—Give potassium iodide and a linctus, *e.g.* morphinæ hydrochlor. gr. $\frac{1}{24}$, syrup of balsam of tolu, and acid infusion of roses, āā ℥ss.

3. *Tuberculous.*—These occur in advanced cases of pulmonary and laryngeal phthisis (*v.* articles on those subjects).

Perforation.—This may arise:—

1. From bursting of an aneurysm. This is immediately fatal.

2. From bursting of an abscess into the trachea. This may cause death from asphyxia if the abscess is large. If this accident is not immediately fatal the treatment indicated is to provide a free external drain for the abscess.

Tumours. — Tumours of the trachea are uncommon, many of them extremely so. Elsberg classifies them thus:—

1. Benign
   - Papilloma; most common.
   - Fibroma.
   - Enchondroma.
   - Osteoma.
   - Myxo-adenoma.
2. Malignant
   - Carcinoma.
   - Sarcoma.

The benign tumours of the trachea have attention called to them by the occurrence of permanent difficulty of breathing, or of passing attacks of dyspepsia. For diagnosis see remarks on Tracheal Stenosis.

Treatment.—If situated high up, a papilloma of the trachea may be removed by the endo-laryngeal route. If low down, tracheotomy must be done, and the tumour, if possible, removed through the tracheal wound.

Tracheal carcinoma practically always occurs by extension from surrounding structures.

Tracheal Obstruction. — *Causes.* — 1. Stricture of the trachea from cicatricial narrowing of its wall, generally syphilitic.

2. A tumour within the trachea.

3. Pressure from without, *e.g.* by a thoracic tumour or aneurysm, by an abscess, by an enlarged thyroid gland, or by a dislocated clavicle.

Symptoms.—There is an obvious obstruction to respiration, and stridor is present. Inspiration is more noisy and more prolonged than expiration. The larynx moves up and down little, if at all, during respiration, while in laryngeal obstruction the larynx moves up and down freely. In extreme cases the lips have a bluish tinge, and the patient uses forcibly both the ordinary and the extraordinary muscles of respiration. By auscultation over the trachea a high-pitched, whistling sound is heard. The voice may not be affected at all. It may, however, be faint and weak; but this quality must be distinguished from the hoarseness generally present in cases of laryngeal obstruction. However, in a case of tracheal obstruction the voice may be hoarse, as pressure on the trachea is often accompanied by pressure on one or both recurrent laryngeal nerves.

These chronic symptoms may be associated with paroxysms of severe dyspnœa, which may be due to swelling of the tracheal mucous membrane, or to double abductor laryngeal paralysis due to pressure on both recurrent laryngeal nerves.

Diagnosis.—A laryngoscopic examination must be made, the presence of laryngeal obstruction being thus excluded, and the presence of a tumour or stenosis high up in the trachea perhaps diagnosed. Next the thorax must be examined for signs of the presence of an aneurysm and for signs suggestive of chronic mediastinitis. The neck, too, must be examined for evidence of aneurysm or tumour, and an œsophageal bougie should be passed. If the investigation of these parts has a negative result, then the obstruction must be in the trachea and due to contraction of its wall or an endo-laryngeal tumour. In the case of the former there will probably be a history of syphilis, while the latter may possibly be detected with the laryngoscope.

Treatment.—In many cases treatment is of no avail; in others a tumour pressing on the trachea has to be removed or an abscess evacuated; while in a third class, *e.g.* tracheal stenosis, treatment is possible, though often unsatisfactory. For stricture high up in the trachea a low tracheotomy should be done. Stricture low down in the trachea has been treated by passing flexible tubes through tracheotomy wounds.

Rupture.—Rupture of the trachea is very rare.

Causation.—Direct violence applied over the trachea.

Symptoms.—Obstruction to respiration, swell-

ing over the front of the neck. General emphysema is generally rapidly fatal.

Treatment.—Expose the trachea by a free median incision, clear away blood-clot. If the ends of the trachea are widely separated, suture them together. Put in a tracheotomy tube.

WOUNDS.—*Varieties.*—1. Incised wounds, *e.g.* in cases of cut throat.

2. Gun-shot wounds.

In the former the edges of the superficial wound should be left widely open, while the edges of the tracheal wound, if widely separated, may be sutured. Gun-shot wounds often result in tracheal stenosis.

FOREIGN BODIES IN THE TRACHEA.—*Symptoms.*—At the moment the body is drawn through the larynx there is a violent attack of dyspnœa due to spasm of the glottis; but when the body has dropped into the trachea the urgent symptoms subside. The absence of symptoms is only temporary, for directly the body is coughed into the larynx again, another spasm of the glottis occurs, with resulting obstruction to respiration.

Treatment.—When the history is given of recurrent acute attacks of dyspnœa, with free intervals following the supposed entry into the air-passage of a foreign body, tracheotomy should be at once done. The edges of the tracheal wound being held widely apart, the body is, as a rule, coughed into view and can easily be removed. If not at once seen, it should be searched for with forceps, and if it is still not found a tracheotomy tube should be inserted, and the trachea further explored on another occasion.

TRACHEOTOMY.—*Indications for the Operation.*—1. The presence in the larynx, pharynx, or upper part of the trachea of a serious obstruction to respiration, *e.g.* :—

(*a*) Diphtheritic membrane.
(*b*) Tumour of larynx or trachea.
(*c*) Œdematous laryngitis.
(*d*) Double abductor laryngeal paralysis.
(*e*) Cicatricial adhesion of the vocal cords.

2. Foreign bodies in the trachea.

3. As a preliminary to certain operations, *e.g.* removal of the tongue.

Varieties of Operation.—1. The high operation, in which the trachea is opened above the isthmus of the thyroid.

2. The low, in which the trachea is opened below the isthmus. The former operation is usually done.

Position of the Patient.—The patient should be on a narrow table in a good light. There should be a small pillow beneath the shoulders, so that the head is stretched back, and the chin should be kept accurately in the middle line.

The Operation.—The usual precautions as to asepsis having been taken, and the skin being steadied with the fingers of the left hand, an incision 1½ in. long is made accurately in the middle line of the neck with its upper end just overlapping the cricoid cartilage. The skin and fat being rapidly divided, the layer of deep cervical fascia overlying the pre-tracheal muscles is seen and divided. Beneath this fascia the anterior jugular veins run down on either side of the middle line, and there may be a communicating branch across the middle line. The interval between the sterno-hyoid and sterno-thyroid muscles of the two sides is then recognised in the middle line, and the muscles are separated and held apart with retractors. The finger should now be put into the wound and the trachea felt for, and steadied between the thumb and fingers of the left hand. The deep cervical fascia over the front of the trachea is now to be cleanly divided in the middle line, and the thyroid isthmus, which lies over the second and third rings of the trachea, pulled down with a blunt hook. Distended veins may be encountered at this stage, and should be clamped or drawn aside. The rings of the trachea being clearly exposed, a sharp hook is introduced into the middle line of the trachea just below the cricoid cartilage, and the air-passage pulled a little upwards and forwards. Then with a small scalpel the operator sharply plunges into the middle line of the trachea at about the third tracheal cartilage and cuts upwards. The rush of air into the trachea is now heard, and the tracheal dilator is immediately introduced and the blades opened. The tracheotomy tube is next introduced in the following way:—With the right hand hold the dilator in the trachea with its blades apart. Take the outer tube in the left hand, press its two halves together, and introduce it slowly and carefully into the trachea between the opened blades of the dilator. As soon as it is certain that air is passing in and out through the tube, remove the dilators while the tube is held securely in position. Then tie the tapes round the neck and introduce the inner tube.

If the low operation is being done the incision must be nearer the sternum, and the thyroid isthmus pulled upwards. Difficulty may arise from the inferior thyroid veins on the front of the trachea and from the occasional presence of the thyroidea ima artery running up the middle line of the trachea.

Comment on the Operation.—1. It is impossible to describe here all the varieties of tracheotomy tube. The most generally useful one is the ordinary bi-valve tube with a movable shield.

2. The chief difficulties likely to be met with are:—

(*a*) The patient may be an infant with a short, fat neck. This converts what is otherwise an easy operation into a difficult one, for the trachea is deeply situated, and the room for manipulation is limited. If the cardinal rules—to make a long enough incision, to cut

accurately in the middle line, and not to open the trachea till its rings are recognised—are observed the difficulty is reduced to a minimum.

(*b*) Distended veins may be found crossing the line of incision. These should, as far as possible, be secured and divided between forceps; but too long a time should not be spent in arresting venous bleeding, for this will cease as soon as the trachea is opened.

(*c*) In young, fat children there is very little space left above the thyroid isthmus. In this case the isthmus should be divided in the middle line. The bleeding will be slight, and can at once be stopped by applying forceps to each half of the isthmus.

(*d*) The tube may be introduced between the pre-tracheal fascia and the trachea. To avoid this divide the fascia freely enough, and introduce the tube carefully and without hurry.

(*e*) One limb of the outer tube may be put into the trachea, and one between the trachea and the fascia. This is avoided if ordinary care is taken. The two last occurrences are recognised by the fact that no air passes through the tube, in which case the tube must be taken out and reintroduced.

After-Treatment. —The patient should be in a bed surrounded by curtains, and in a temperature of from 62° to 65° F. A steam tent may be used, but is not necessary. A covering of six or eight layers of gauze should be kept over the tube and frequently renewed.

Membrane or secretion coughed up through the tube is to be caught on a mop, and must on no account be allowed to slip down the tube again.

The inner tube should be removed whenever it becomes blocked, and, under any circumstances, once every two hours at least. Before being put back it must be cleaned by boiling in soda solution.

Nourishment must be given frequently. Blunt hook retractors, tracheal dilators, and an extra tube must always be at hand.

On the fifth or sixth day an attempt should be made to leave the tube out. When this is done the surgeon should be near at hand for some time, as the tube often has to be replaced.

The remarks in this article apply chiefly to the operation when done for diphtheritic laryngitis. When it has been done for some conditions, *e.g.* growths or stenosis of the larynx, the tube may, of course, have to be worn permanently.

Difficulties met with in attempting to leave out the Tube.—1. *Fright.*—For a few minutes after removal of the tube the patient breathes through the tracheal wound, and directly the edges of the wound fall together the child feels the slight obstruction to inspiration and becomes frightened. A spasm of the glottis occurs, and the tube has to be replaced. This may occur several times.

Treatment.—Use a tube with an opening on the convexity, plug the outer end of the tube, and the patient unconsciously breathes through the larynx, and the tube can be removed.

2. Granulations may form in a mass at the junction of the trachea and larynx, or in the trachea on a level with the wound.

Treatment. — Use a well-fitting tube with a movable shield, and leave it out as soon as possible. If granulations should form touch them with silver nitrate. It may be necessary to frequently pass a flexible tube from the wound up through the glottis.

3. Stenosis of the larynx, varying from partial adhesion of the cords to complete blocking. In the former case dilate the rima-glottidis after division of the cicatricial tissue. In the latter thyrotomy must be done, and the cicatricial tissue cut away. In both conditions relapse is frequent, and so the tube has to be permanently worn.

4. Impairment of function of the laryngeal muscles, which may take one of the following forms:—

(*a*) Diphtheritic paralysis of the laryngeal muscles, which is more prone to affect the abductors of the cords than the adductors.

(*b*) The wearing of the tube interrupts the normal rhythm between the actions of the laryngeal muscles and those of inspiration.

In both these conditions the treatment is to wait, for the muscles will regain their functions in time.

5. In one instance, in which it was found impossible to leave out the tube, the writer found that the trachea was compressed by a large amount of cicatricial tissue formed round the tracheotomy wound.

Tracheal Tugging. *See* TRACHEA. *See also* ANEURYSM (*Neck, Symptoms*); AORTA, THORACIC, ANEURYSM OF (*Symptoms, Diagnosis*).

Tracheitis.—Inflammation of the trachea. *See* TRACHEA (*Inflammations*).

Trachelectomy.—Excision of the cervix uteri (Gr. τράχηλος, throat or neck).

Trachelismus.—Spasmodic contraction of the muscles of the neck.

Trachelo-.—In compound words *trachelo-* (Gr. τράχηλος, neck) means relating to the neck, *i.e.* either the neck proper or the neck (cervix) of the uterus or bladder; there are many words so formed, *e.g. trachelo-acromial, trachelo-bregmatic, trachelo-cystitis, trachelodynia* (pain in the neck), *trachelo-mastoid, trachelo-scapular*, etc.

Tracheloparasitus. — A group of double monsters in which the parasitic twin fœtus is inserted on the neck of the autosite, *e.g.* a supernumerary limb attached to the neck, an epignathus, etc.

Trachelorrhaphy. — Repair of the torn cervix uteri; Emmet's operation.

Tracheloschisis. — Congenital fissure of the neck; schistocormus fissicollis.

Tracheloterata. — Malformations and structural anomalies affecting the cervical region, *e.g.* fistula colli, cervical auricles and cysts, cervical ribs, etc.

Trachelotomy.—Incision into the cervix uteri.

Tracheo-.—In compound words *tracheo-* (Lat. *trachia*, the windpipe) means relating to the trachea, e.g. *tracheobronchitis* (inflammation of trachea and bronchi), *tracheolaryngeal* (belonging to trachea and larynx) *tracheoscopy* (inspection of the trachea by the laryngeal mirror), and *tracheostenosis* (narrowing of the trachea).

Tracheotomy.—The operation of opening into the trachea, either below or above the isthmus of the thyroid, either through the cricothyroid membrane or through the cricoid cartilage. *See* TRACHEA (*Tracheotomy*) and *Cross References.*

Trachielcosis.— Ulceration of the trachea. *See* TRACHEA (*Diseases of*).

Trachinus. *See* SNAKE-BITES AND POISONOUS FISHES (*Poison Wounds caused by Fish, "Weavers" or Trachinus*).

Trachoma. *See* CONJUNCTIVA, DISEASES OF (*Chronic Ophthalmia, Trachoma*); EYE, CLINICAL EXAMINATION OF (*Palpebral Conjunctiva, Granular Lids*); LARYNX, ACUTE AND CHRONIC INFLAMMATIONS (*Nodular Laryngitis or Trachoma of the Vocal Cords*).

Tracing. *See* PHYSIOLOGY, CIRCULATION (*Pulse Tracings*); PULSE (*Sphygmograph, Use of*).

Tract or Tractus.—A region of the body, *e.g.* of the spinal cord, which has its length considerably greater than its breadth or thickness, *e.g.* the tracts of white matter in the spinal cord and brain; also the alimentary or the genito-urinary canals. *See* PHYSIOLOGY, SPINAL CORD AND MEDULLA OBLONGATA (*Tracts*).

Traction, Axis. *See* LABOUR, OBSTETRIC OPERATIONS (*Forceps*).

Tractoration.—Treatment by metallic tractors. *See* PERKINISM.

Trades, Dangerous.

LEAD-POISONING 84
Symptomatology 86
Pathology 88
Diagnosis and Prognosis . . . 88
Treatment 88
MERCURY 89
Symptomatology 89
Pathology, etc. 90
Treatment 90
PHOSPHORUS AND LUCIFER MATCH-MAKING 90
Symptomatology 91
Pathology, etc. 92
Treatment 93
BISULPHIDE OF CARBON AND INDIA-RUBBER MANUFACTURE 93
Symptomatology 93
Treatment 94
INFLAMMABLE PAINTS AND BENZINE . . 94
Treatment 94
ANILINE AND DINITROBENZINE—
Description of Process of Manufacture —Symptomatology 95
Risks attending the Manufacture and Use of High Explosives . . 96
WORKERS IN THE CHEMICAL TRADES . . 96
The Manufacture and Use of the Bichromate Compounds 98
WORKERS IN ARSENIC 98
COPPER AND BRASS: BRASSFOUNDERS' AGUE 99
THE TEXTILE TRADES: COTTON AND LINEN 99

See also DERMATITIS TRAUMATICA ET VENENATA; DERMATITIS IN COAL-MINERS; TOXICOLOGY (*Lead-Poisoning, etc.*).

LEAD-POISONING

SYNONYMS.—Plumbism; Saturnine Poisoning; Painters' Colic; Colica Poitou; *Fr.*, Intoxication Saturnine; *Germ.*, Bleivergiftung.

OF all forms of metallic poisoning that caused by lead is far and away the commonest, owing to lead being so widely used in the arts and in commerce. The peculiarity of lead-poisoning is that it may develop so insidiously that a person is sometimes precipitated into a state of serious ill-health without much warning.

The causes of plumbism may, roughly speaking, be regarded as three: accidental, industrial, and self-induced. Under accidental poisoning are to be included those cases in which plumbism has been caused by drinking water that has become contaminated either through being stored in a lead cistern or through having lain overnight in the conduit-pipe that leads from the main into the interior of the house, through eating canned food that has become adulterated in consequence of the acid juices having acted upon the solder, also through eating food that has been cooked in cheap enamelled pans. A few cases, too, can be traced to the use of certain hair-dyes. Under the head of industrial lead-poisoning must be included a variety of trades, *e.g.* the smelting of lead, the manufacture and packing of red and white lead, dye-works using chrome or yellow lead compounds, house- and coach-painting, electric accumulator works, plumbing, manufacture of pottery, file-cutting, glass-polishing, etc. Self-induced lead-poison-

ing is generally due to lead having been taken, not with the view of causing death suicidally, but to induce abortion in pregnancy.

Accidental lead-poisoning is mostly due to drinking contaminated water. Under such circumstances the malady assumes almost an epidemic character. In a town or locality several people are found to be suffering at the same time from plumbism. Certain waters are known to exercise a greater solvent influence upon lead pipes than others. Water that is soft and of moorland origin owing to the presence therein of free acid that has been generated during the decomposition of peat, also water that contains iron, are capable of dissolving out a sufficient quantity of lead from the pipes to cause in a very short time symptoms of such severity as to threaten life or to incapacitate the individual for weeks, or even months. Several towns in Yorkshire have within the last few years suffered from plumbism traceable to this source. The addition of unburnt lime or of magnesian limestone to the filter-beds, or allowing the drinking water to gravitate through layers of sand, confers upon the water that degree of hardness, or gives to it such salts as carbonates and silicates, whereby an insoluble coating is deposited on the inside of the pipe which, in the absence of free carbonic acid, forms a protective covering.

It is to the numerous industries that we look for the largest number of cases of lead-poisoning, and among these the manufacture of red and white lead, manufacture of certain kinds of pottery, and the occupation of house-painting may be specially mentioned. In Britain the getting of the raw ore is not attended by any risk to the miner from plumbism, but at the Brocken Hill mines in Australia, where the ore exists very largely in the form of carbonate, there has been much sickness among the miners due to lead. In Britain the danger commences with the smelting of the lead ore. There is little risk from the manipulation of metallic lead compared with that incurred by the handling and inhalation of lead compounds. The more soluble these compounds are, the greater is the danger. Among the recognised dangerous compounds may be mentioned the red oxide, carbonate or white lead, acetate, and chromate. In any industry in which these salts of lead are made or handled the workpeople, unless they are particularly careful, run the risk of losing their health sooner or later. In the manufacture of pottery the earthenware is glazed by being dipped in a composition that contains a large quantity of lead carbonate or oxide. When the ware is dried and is being cleaned or brushed there is given off a considerable amount of dust, the inhalation or swallowing of which induces plumbism. Lead compounds, too, are used for colouring pottery, and especially for what is known as majolica ware. Workers in pottery are, in many ways, therefore, exposed to the risk of lead-poisoning. The frequency of lead-poisoning, too, in house-painters has conferred upon the malady, through one of its symptoms, the name of painters' colic. Within recent years several new industries have developed. Of these the one that is specially productive of plumbism is the manufacture of electrical batteries for telegraph and telephone purposes. In the manufacture of these batteries perforated lead plates are used. The openings in the grid have to be filled by a composition that looks like red clay, but which is a mixture of red oxide of lead. During the mixing of the dry powders that form the composition a considerable amount of dust rises and is inhaled, and during the rubbing of the paste into the openings in the plate, which is usually done by the hands encased in india-rubber gloves, some of the paste, owing to a tear in the gloves, gets rubbed into the skin and causes plumbism. Of the other trades risky to health from the handling of lead or its compounds may be mentioned file-cutting by hand, house- and coach-painting, the dyeing of wool by means of chromate of lead, and, especially on the Continent, the handling of type by compositors for printing newspapers, etc. In Britain and America newspaper printing is now so largely done by means of the linotype machine instead of by hand-set type, that while linotyping is not absolutely free from the possibility of lead-poisoning, the risk is so extremely small compared with that incurred by setting up the type by hand, that the two methods can scarcely be contrasted. In hand-file cutting and in pottery manufacture, in addition to the risks from plumbism, the death-rate from pulmonary phthisis is extremely high, owing to the dusty nature of the occupation.

In addition to the accidental and industrial forms of lead-poisoning, allusion has been made to that which is self-induced. Lead salts are very rarely resorted to for suicidal purposes; but as lead is known to possess distinctly ecbolic properties, the ignorant often have recourse to diachylon pills for the purpose of procuring abortion. Self-administration of the pills has, in the Midlands, where the practice is more prevalent than in other parts of England, caused death in some instances, and in others induced such serious symptoms as paralysis and loss of vision.

Of the sexes females are, I think, on the whole more susceptible to lead than males, so too are young persons and all who are ill-nourished, anæmic, and intemperate in the use of alcohol.

The channels by which lead effects an entrance into the system are the alimentary canal and respiratory passages, and also the skin. Workers in white lead not only inhale

the dust, they swallow it. When they eat their food with unwashed hands the lead which is swallowed becomes dissolved by the hydrochloric acid of the gastric juice, and is converted into a soluble chloride, some of which may be absorbed as chloride, or it may be converted into an albuminate, while a large quantity escapes by the fæces in the form of sulphide. That lead is capable of entering the system through the skin has been demonstrated by the use of cosmetics and hair-dyes that contain lead. When the poison is circulating in the blood symptoms of acute plumbism show themselves, often on the side of the nervous system; but there is a tendency for the metal to be deposited in the tissues, probably in the form of the rather insoluble albuminate. In several of my fatal cases of plumbism, Dr. Bedson, Professor of Chemistry in the University of Durham College of Science, extracted from the liver, kidneys, brain, and muscles lead in varying quantities. The poison is unevenly distributed throughout the body, but in my own cases the liver was the organ that generally contained the largest quantity of lead.

Symptomatology.—One of the earliest signs of a white-lead worker becoming poisoned by lead is a peculiar *pallor* of the countenance. Probably for several weeks the friends of the workman have noticed that he has been losing the ruddy appearance of health. Not only does the face become pale, but the features tend to become expressionless. There is a peculiar fulness, too, in the lower half of the face. On examining the blood microscopically it is noticed that there are fewer red corpuscles than in health, and that the hæmoglobin is proportionally diminished, while the number of leucocytes remains about the normal. Since lead is often found in the bones after death, the probability is that the function of bone marrow as a blood-cell-forming agent is seriously impaired early in the course of plumbism. Sometimes without any very marked, but usually with a considerable degree of pallor, the worker is gradually or suddenly seized with acute *pain* in the abdomen, which is referred to the neighbourhood of the umbilicus just above or to either side of it. The pain is sometimes so severe that he writhes in agony. He can allow nothing to touch his abdomen. In other instances not only is pressure well borne but relief is obtained by the patient placing a pillow on the back of a chair and leaning all his weight upon it. Accompanying the pain there is often vomiting and a considerable amount of restlessness. Usually the bowels are obstinately confined, but instead of constipation there may be diarrhœa. The abdomen may be retracted, but not necessarily so. During the attack of colic, sulphocyanides disappear from the saliva to reappear on cessation of the pain. The cause of lead colic has not been definitely determined. The character of the pain is suggestive of spasm, and it is therefore probably due to extreme peristaltic contraction of portions of the small intestine. In animals poisoned by lead I have found the small intestine so firmly contracted at places as to have its calibre completely obliterated, while the intervening portions were either normal or very slightly dilated. The pain therefore might be due to the distended portions of the intestine trying to send on their contents into the portions spasmodically contracted, or be due, as is more probable, to the spasmodic contraction itself. It is difficult to say whether lead acts primarily upon the muscular fibre of the bowel, or upon the sympathetic ganglia and nerve-fibres. During colic the radial pulse is small, slow, and feeble; it may fall to forty beats in the minute. At other times the pulse is one of high tension, and the two radial pulses are unequal. The urine is scanty; sometimes only eight or ten ounces are passed in the day. In uncomplicated cases it is free from albumin, but albumin may be present, persist through the colic in some patients, and disappear with it. This temporary albuminuria may be due to irritation of the renal epithelia by the toxic blood, or it may be that it marks the commencement of a parenchymatous nephritis, which becomes confirmed as time goes on, and ultimately ends in the development of contracted kidney. Frequently lead colic has been mistaken for appendicitis, and *vice versa*. The question has been sometimes raised as to whether poisoning of the musculature of the bowel by lead may become a cause of appendicitis. Certainly a patient suffering from lead colic can develop an appendicitis; but even under these circumstances I should regard intestinal micro-organisms as the more probable cause of the appendicitis. It is not always easy to distinguish between lead colic and inflammation of the appendix. As a rule lead colic only lasts two or three days, the pain is referred to a part higher up in the abdomen than the seat of the appendix, there is more sickness, also an absence of the elevated temperature observed in appendicitis. On examining the gums there is usually to be seen a *blue line* close to the margins of the teeth, especially noticeable where the teeth are decayed. Such a line, although corroborative evidence of plumbism, is not a proof of lead-poisoning being present at the time of its detection by the medical practitioner, for a blue line may for months be seen on the gums of lead-workers without these persons presenting any symptoms of lead-poisoning at all. It indicates that lead is in the system. This blue line, which was first described by Burton, is due to the deposition of particles of sulphide of lead in the deeper epithelial cells of the gums, and particularly in the large connective-tissue cells situated in the ulcerated portion of the

gums. Where there are no teeth there is no blue line, and where work-people are in the habit of using the tooth-brush regularly it may be absent. This circumstance suggests that the coloured line is due to the decomposition of particles of food that have been arrested between the teeth, the development therefrom of sulphuretted hydrogen, and the reaction of this gas upon lead salts in solution, and the deposition of the insoluble black sulphide in the interior of the cells Many of my patients have had large, bluish-black patches, the size of a sixpenny piece or larger, inside the lower lip or on the mucous membrane of the cheek, usually opposite a decayed tooth. At autopsies I have found similar large patches in the mucous membrane of the small intestine.

A common sign of lead-poisoning is *wrist-drop* In plumbism, as in all forms of paralysis due to poisoning, the loss of power is bilateral. Although the wrist-drop is double, one hand is usually more affected than the other In some instances muscular strain predisposes to the paralysis, but apart from this circumstance, lead itself, quite irrespective of the manner in which it has gained an entrance into the system, is capable of causing the loss of power The paralysis affects the extensors of the fingers and wrists Often the index finger escapes In other instances, both it and the little finger as well as the thumb cannot be extended, nor can the fingers be separated from each other owing to paralysis of the dorsal interossei. What strikes an observer in regard to the double wrist-drop is the utter helplessness of the patient. The hands fall powerless by his sides, the patient cannot raise a hand unless he places the other underneath it, nor can he pick up anything Any attempt to extend the fingers and wrists induces marked tremor of the muscles generally, including the flexors, which in time also suffer from loss of power. Preceding the paralysis, which may have been gradually or quickly developed, there is sometimes experienced by the patient a sense of tingling or of numbness, but only very occasionally is actual pain complained of. Even when there is pain it is never so severe when the affected muscles are grasped as in peripheral neuritis, due, for example, to alcohol The muscles sooner or later begin to atrophy One of the characteristic features of wrist-drop due to lead is that, while the extensors of the fingers and wrists are paralysed, the supinator longus, which is supplied by the same nerve, viz. the musculo-spiral, is not affected. In addition to wrist-drop, two other forms of paralysis are met with: (1) the *Duchenne-Erb* type, in which the deltoid, biceps, brachialis anticus and supinator longus are affected—a form of paralysis that is usually severe, and (2), the *Aran-Duchenne* type, in which the small muscles of the fingers, *e g* thenar, hypothenar, and interossei, are affected The appearances presented by this latter type resemble progressive muscular atrophy, only there are not the fibrillation and muscular tremor, nor the rapid loss of substance so noticeable in progressive muscular atrophy. In severe cases of plumbism the paralysis may extend to other muscles, *e g* those of the feet and toes, so that *ankle-drop* is produced. This form of paralysis constitutes the peroneal type. The muscles of the leg and thigh escape. The common extensors of the toes and big toe suffer so that the individual is unable to bear his weight on the outer border of his foot. When the muscles of the trunk become paralysed the patient is in a sad plight. He cannot turn in bed, he is perfectly helpless There is a danger of the paralysis extending to the muscles of deglutition and respiration. In some cases the muscles of the eyeball are affected. In plumbism the paralysed muscles lose their response to electrical stimulation Even under treatment, lead paralysis is slow to disappear. The knee-jerk is sometimes absent Occasionally lead-poisoned patients have symptoms not unlike those observed in tabes In standing or walking there may be staggering which is aggravated by closure of the eyes, also loss of the knee-jerk. In others the symptoms resemble those of general paralysis, there being loss of memory, muscular tremor of the lips and tongue, and embarrassment of speech, but instead of these symptoms, as in general paralysis, becoming worse, they tend to improve under treatment

In chronic cases of saturnine poisoning there is albumen in the urine. Consequent upon the anatomical changes induced in the kidneys and the impairment of their functional activity, structural alterations are subsequently set up in the heart and arteries, not unlike those observed in chronic interstitial nephritis. In the urine there is a deficiency of urea. The nitrogenous metabolism of the body is interfered with, so that where there is a tendency to uratosis, plumbism favours the development of gout The association of gout and lead-poisoning is observed with much greater frequency in the South than in the North of England or Scotland, a circumstance which suggests that other factors than plumbism are concerned, such, for example, as the habits of the people and the nature of the alcoholic drinks consumed In women plumbism often induces menorrhagia, and, if pregnant, causes them to miscarry. Female lead-workers, when pregnant, if they wish to go to term and have a living child, should therefore retire from the factory for a period. Not only does lead induce abortion in the mother, but it causes convulsions in her children. Most of the children of lead-workers die either at birth or shortly afterwards from convulsions.

There is a form of acute lead-poisoning which is rapidly fatal If it is not immediately fatal it is almost sure to be followed by sequelæ of

an unusually anxious character. It is known as *saturnine encephalopathy*. A lead-worker who, perhaps, has previously suffered from colic and mild wrist-drop, or who, perchance, has never exhibited any of these symptoms, except complained of severe headache, is suddenly seized with convulsions and unconsciousness. At the time of the seizure he may be in the factory. He is taken to a hospital, where it is found that the convulsions keep recurring, and although consciousness is not completely restored, he moans as if in pain, and is restless. In the urine, which is drawn off by catheter, there may be albumen; or if the patient is young and has only worked a few months in the lead factory, the urine may be found to be quite healthy but scanty. The pulse is small, and of fairly high tension. In one of the epileptiform seizures the patient perhaps dies, usually about the third day; or it may be that he recovers, and as consciousness is regained, the patient complains that he cannot see. This loss of vision may be temporary or permanent. On examining the fundus oculi ophthalmoscopically, well-marked signs of neuro-retinitis are observed, also retinal hæmorrhages; and as these occur in patients whose urine is often free from albumen, they are probably the result of the action of lead itself upon nerve-tissue, rather than, as some physicians maintain, uræmic in origin. In the milder forms of lead-poisoning there may be temporary amblyopia, without any marked changes in the disc and retina, a condition probably due to a toxic effect produced by the lead upon the cerebral visual centres. Premonitory to an attack of lead encephalopathy in females, it is no uncommon thing for them to exhibit what appears to be ordinary hysterical symptoms. This form of *toxic hysteria* is extremely fatal, and is apt to throw even careful observers off their guard. Out of the hysterical symptoms convulsions develop, and within three days the patient may be dead. Delirium of a very acute character, accompanied by high temperature, often follows an apparent recovery from lead encephalopathy, but it wears out the strength of the patient, and he dies within a few weeks afterwards; or after the acute delirium subsides there remain delusions which oblige the patient to be removed to an asylum. Females are more predisposed to lead encephalopathy than males.

Pathology.—In acute lead encephalopathy there may be practically no definite morbid anatomy. The surface of the brain may look dry and the convolutions feel hard and firm; the blood-vessels may be injected, and on slicing the brain the tissue may be extremely pale or watery, as in uræmia. On microscopical examination of the liver there may be signs of commencing intercellular cirrhosis. The kidneys in acute lead-poisoning in young subjects exhibit signs of commencing parenchymatous nephritis; in chronic cases there is well-marked interstitial nephritis. In chronic lead-poisoning with paralysis the muscles are pale and wasted: the nerves exhibit a thickening of their sheath and an increase of their interfibrillar connective tissue; the axis-cylinder is attenuated, it is interrupted here and there in its course, a condition which Gombault described as "periaxial" or "segmentary neuritis," in which healthy and diseased portions of nerve-fibre alternate with each other. The spinal cord is for the most part healthy. Occasionally distinct changes have been observed in the anterior horns of grey matter which lead to shrinkage of the large multipolar cells situated there, a circumstance to which many pathologists attribute the progressive atrophy of the affected muscles.

Diagnosis.—If to the fact of acute pain in the abdomen there be added anæmia, history of the patient having been exposed to lead, also the presence of a blue line on the gums, the diagnosis of lead colic is pretty certain. Appendicitis as a possibility should not be forgotten. The bilateral character of the wrist-drop suggests poisoning of some kind, and the fact that the paralysis affects the wrist and not the legs is in favour of lead as opposed to arsenic and alcohol as the cause. Saturnine encephalopathy might be mistaken for uræmia, especially when there is albuminuria. The presence of a blue line on the gums would be an aid in the diagnosis, and in any doubtful case the urine should be tested for lead.

Prognosis.—Lead colic is, practically speaking, always recovered from: less than 1 per cent die. Recovery from saturnine paralysis is slow and often incomplete; yet I have seen complete recovery even in the worst cases when the muscles of the arms, legs, trunk have all been involved. The most serious type of lead-poisoning is that known as saturnine encephalopathy. In the chronic form of plumbism, where the kidneys are affected, the tendency is for the symptoms to get worse, and for the patient to die from cerebral hæmorrhage and convulsions.

Treatment—Preventive.—Where plumbism is due to drinking contaminated water the recommendations made at the commencement of this paper should be followed. It cannot be too much insisted upon that lead-poisoning is much more likely to be caused by the continual entrance into the system of very minute quantities of lead than by one or two large doses. For industrial lead-poisoning the Home Office has drawn out a series of regulations, the main points in which are personal cleanliness, washing of the hands and also of the face before eating, for lead dust collects upon the beard and moustache, the wearing of respirators, and periodical medical examination of the workers. Acidulated drinks are provided in many of the factories, but too much reliance must not be placed upon these. They contain Epsom salts

and fresh lemon juice. Theoretically it is supposed that they convert the chloride of lead in the alimentary canal into the sulphate, but while this is a less soluble compound than the chloride, it is not so insoluble as to be free from risk.

Therapeutic.—For lead colic warmth to the abdomen, the administration, in the absence of severe sickness, of a mild purgative, *e.g.* Epsom salts or castor oil; a warm bath may be given If pain is not quickly relieved, a hypodermic injection of morphia may be necessary. When colic is more continuous and diffused over the abdomen, I have found monosulphite of soda in ten-grain doses thrice daily quickly relieve the pain. Olive-oil in tablespoonful doses relieves pain if it can be borne. If the bowels are not relieved, enemata must be administered. When there is repeated vomiting an effervescing bromide and belladonna mixture is worthy of a trial. Of the drugs given with the object of eliminating lead from the system, iodide of potassium is the most reliable. It must in the more acute forms of plumbism be given with caution, for experience confirms the statement made many years ago by Melsens, that potassium iodide is capable of dissolving out lead which has been deposited in the tissues, and of inducing symptoms of fresh poisoning For lead paralysis massage, electricity applied either directly to the limbs or by means of alternating currents in an electric bath, also the administration of potassium iodide and nux vomica, or the hypodermic injection of liquor strychniæ may be tried. Where there is marked anæmia and loss of strength syrup of the iodide of iron and bone-marrow tablets are often beneficial. For saturnine encephalopathy pilocarpine, administered hypodermically, or a disc placed upon the tongue of the patient, also nitrite of amyl inhalations, or saline transfusion, are well worthy of a trial.

Mercury

Until within recent years mirrors were *silvered* by means of quicksilver. The use of this metal was the cause of a considerable amount of poisoning in those who followed the trade. Since the introduction of the new method by means of silver nitrate and tartaric acid mirror-makers no longer incur the risks of former days In the manufacture of scientific instruments of precision, such as barometers, thermometers, etc, quicksilver is still largely employed Men who follow this avocation occasionally get poisoned, so too do felt-hat makers. It is found that the *felt* out of which hats are made can be more readily manipulated, and that it lies better, if it has been passed through a solution of nitrate of mercury. The danger from using this salt is less when the felt is wet than after it has become dry, for the particles of the mercuric salt are in this form inhaled. Should a workman get a cut on his finger when manipulating the wet goods, the wound is apt to become very painful and irritable. The introduction of American machinery, *e.g.* the Blake machine, into the manufacture of boots and shoes has, owing to the presence of a trough filled with quicksilver in the upper part of the machine in order to allow a revolving arm to move easily upon a joint, been followed by signs of mercurial poisoning. Mercury volatilises at a very low temperature, so that in the overheated and close rooms of a boot factory lit up on a winter's night by numerous jets of gas, of which there is always one on each machine, there is abundance of opportunity for the escape into the workroom of the metallic vapour. Until the California mines were opened most of the mercury used in our own country came from Almaden in Spain. The mining of quicksilver is a most unhealthy occupation, but newer methods of getting the metal and an improved system of ventilation have materially improved the lot of quicksilver miners Formerly the conditions in the Spanish mines were so bad that the work had to be undertaken by gangs of convicts, but so unhealthy was the occupation and so wanting in personal cleanliness were the men, that quicksilver-mining, although slower in its operation, became just as certain a means of shortening life as the execution of a death sentence by a quicker method. The Californian miners have not suffered like the Spanish There is in some people a peculiar idiosyncrasy to mercury and its compounds. I have a medical friend who is severely purged with one grain of calomel, and a short time ago one dose of two grains of calomel administered by me to rather a pale girl was followed by intense salivation, swollen and painful gums, and fetid breath for several days. Mercury is readily absorbed through the skin, hence this forms a useful channel for introducing, by means of inunction, the oleates of the metal.

Symptomatology.—The symptoms of mercurial poisoning may be acute, subacute, or chronic. Individual susceptibility to mercury has to be remembered. The most common symptoms are a disagreeable taste in the mouth, fetid breath, nausea, and vomiting, excessive flow of saliva, and ulceration of the gums with pallor of the face Persons suffering from anæmia and Bright's disease are readily influenced by mercury. Workers in trades in which quicksilver or compounds of mercury are used do not suffer severely from the mouth conditions just mentioned, but they are not altogether exempted, for they are found to be cachectic, and to possess also a fetid breath, their teeth are loose, and the margins of the gums ulcerated. In addition they are the subjects of muscular tremor, which, usually commencing in the tongue and lips,

extends to the muscles of the limbs and trunk. At first this tremor is only induced by exertion, but after a time it is more or less constant, although it usually ceases when the individual is asleep. The implication of the tongue and lips causes impairment of speech. Occasionally there is paralysis, but the loss of power is never so pronounced as in lead-poisoning, neither do the muscles waste so rapidly nor lose their electrical reaction. The knee-jerks may not be altered. The nutrition of the body is, generally speaking, deranged. It is an interesting fact that while by means of very small doses of mercury the metabolism of the body is quickened so that a patient when undergoing treatment by means of the drug may put on flesh, large doses have just the opposite effect, for owing to the continued entrance of the metal into the system there occur progressive emaciation, an earthy pallor or pigmentation of the skin, falling out of the hair, brittleness of the nails, rheumatic pains, albuminuria, and tremor.

In acute cases of mercurial poisoning, such as that caused by taking corrosive sublimate, there are extreme pain at the stomach, severe retching, vomiting, sense of great constriction in the gullet, profuse diarrhœa, a feeling of intense distress, a sense of great exhaustion, œdema of the glottis, and death from asphyxia or convulsions. The profuse diarrhœic discharges involuntarily expelled by a patient in the throes of corrosive sublimate poisoning render attention to the patient by a medical practitioner and friends a very unpleasant undertaking. Acute mercurial poisoning by the bichloride of mercury is generally suicidal. On an average in this country five deaths per year can be traced to corrosive sublimate. The bichloride of mercury is a powerful poison. Three grains have caused death.

Pathology.—When death has been caused by corrosive sublimate there is observed pallor of mouth, throat, and gullet, and on examining the interior of the body the mucous membrane of the alimentary tract is found ecchymosed in nearly its whole length. In cases of chronic poisoning mercury seems to have the power of dissolving lime salts out of the bones, and these are deposited in the tubules of the kidney in the form of small chalky masses. When there has been paralysis the nerves are found to have undergone degeneration, fatty changes being very noticeable in the myelin sheath.

Diagnosis.—If the case is one of acute mercurial poisoning the extreme pain at the epigastrium, severe vomiting and profuse diarrhœa with collapse, and the history of some drug taken suggest poisoning by corrosive sublimate. In chronic cases the earthy pallor of the skin or bronzing, the muscular tremor and paresis, with slight embarrassment and slowness of speech, recall disseminated sclerosis, but there are not the nystagmus and exaggerated knee-jerks which are present in that illness. The rather loose teeth, with ulcerated gums and absence of blue line and colic, show that it is not a case of plumbism. In some cases the symptoms are suggestive of general paralysis, but there are none of the grandiose ideas nor the pupillary and mental changes met with in that illness.

Prognosis.—If a large dose of corrosive sublimate in solution has been taken the patient will probably die. It is a strong irritant poison. Soothing substances therefore must be given. Milk with white of egg answers very well, for the mercury enters into combination with the latter, forming a rather insoluble albuminate. If possible the stomach should be washed out, but as the mucous membrane of the mouth and gullet has been rendered white and tender by the poison, care must be taken in passing the tube down the œsophagus. Vomiting is usually present; this should be encouraged by tepid drinks of sulphate of zinc and by a hypodermic injection of four minims of apomorphia. In industrial mercurial poisoning there ought to be better ventilation of the workrooms and the removal of dust by a Blackman's fan; personal cleanliness of the workers should be insisted upon; the men ought to be provided with astringent and antiseptic mouth-washes, be given at least a pint of milk daily, and have sulphur baths. In cases of paralysis recourse must be had to massage and electricity. To patients in whom decalcification of bone is taking place the internal administration of calcium phosphate is called for.

Phosphorus and Lucifer Match-Making

Poisoning by phosphorus occurs under two different circumstances: (1) suicidal or accidental, in which the symptoms are of an acute character; (2) industrial poisoning in lucifer matchmakers, in whom the symptoms are subacute or chronic. It is said that acute phosphorus-poisoning never occurs in lucifer matchmakers. Practically this is the case, for only one or two instances have been reported. Phosphorus is *allotropic*, *i.e.* is capable of existing under two different forms and of presenting different physical characters, yet retaining the same chemical composition. There are, consequently, two kinds of phosphorus, red and white or yellow. Red phosphorus is obtained from white by exposing the latter to a very high temperature in air-tight iron vessels. By this means its phosphorescence is lost and the metalloid ceases to catch fire on percussion or friction. White phosphorus is obtained from bone. It alone is dangerous. A very few grains of it will cause death. Persons who resort to phosphorus as a means of committing suicide usually take it in the form of rat or vermin paste, or they dissolve a number

of match heads in water. The composition with which matches are headed contains 5 per cent of white phosphorus, occasionally more. Phosphorus is also taken by ignorant and determined females with the view of producing abortion. In Sweden, a few years ago, the number of deaths from phosphorus taken for this purpose had become so large that Parliament was obliged to consider the advisability of interdicting the use of white phosphorus altogether, and had it not been for the commercial aspect of the question the Bill would assuredly have passed. If women only knew the amount of actual pain and general suffering that phosphorus causes during the few days of life that remain to them after swallowing the poison, they would not so light-heartedly resort to it as a means of self-destruction or for the purposes of producing miscarriage. Two grains of phorphorus have proved fatal. Match heads contain phosphorus varying from ·058 to ·116 grains, so that a solution of from 20 to 100 match heads might if drunk cause death. Suicides generally empty one or two boxfuls. Red phosphorus is not very poisonous. It can be administered to animals without serious consequences following. Risks to health in lucifer matchmakers are only incurred by those who have to do with white phosphorus. Matches made from red phosphorus strike only upon the prepared surface on the box. They are known as *safety* matches, and they are safe not only as regards accidental firing from percussion, but poisoning as well. It is different with white phosphorus. Male and female lucifer matchmakers alike suffer. Phosphorus affects the work-people probably by the fume given off during the mixing of the composition if done in an open vessel, during the dipping of the matches on an open iron slab, and during the drying and boxing of the matches. My experience of animals exposed to the fumes of phosphorus leads me to believe that the fume given off by the heads of matches is as destructive if not more so than that from pure phosphorus. In match-works the men who dip or head the matches are exposed to the fumes of the fresh phosphorus composition, and they suffer in consequence. So too do the women who handle the matches and fill the boxes. Their hands smell strongly of phosphorus towards the end of a day's work, and from the water in which they wash a certain amount of phosphorus can be recovered. In the act of filling the boxes some of the matches often take fire, and the thick clouds of smoke that rise render the atmosphere of the boxing-room thick and impure, also irritating to the respiratory passages when inhaled; but the phosphorus that poisons the work-people enters the system not by this means, but in the form of the more direct fumes from the metalloid itself.

Symptomatology.—When a person has swallowed vermin paste or drunk water in which match heads have been soaked for a sufficient length of time he will, within two or three hours, sooner if the stomach has been empty, complain of acute pain at the epigastrium, followed by retching and vomiting. The vomit smells strongly of phosphorus and may contain blood and biliary colouring matter. The retching and vomiting may continue for a few days, and there may be diarrhœa. If the case has not been treated early enough, that is, before absorption has taken place, jaundice comes on about the third day, and with the onset of jaundice the liver is usually found to be somewhat enlarged and tender. The pulse is small and rapid. The patient feels depressed and ill. Temperature may be unaffected. A high temperature is regarded by many as an unfavourable sign. The urine may or may not contain albumin, as a rule not. Crystals of leucin and tyrosin can be found in the urine if it is carefully manipulated and prepared for their detection. When the jaundice is not marked and the patient has been treated early, improvement may set in within a few days, but a deepening jaundice and the advent of coma point to a secondary poisoning of the patient through imperfect elimination caused by structural changes in the liver, kidneys, and blood-vessels. In severe cases death comes towards the end of the first week, but in children phosphorus-poisoning has proved fatal in a few hours. In addition to vomiting and abdominal pain there may be delirium and convulsions. The skin is often the seat of numerous small petechial-like hæmorrhages. Sometimes the red blood cells are increased in number, on other occasions they are diminished. Women, if pregnant, usually miscarry. The fœtus is dead. In severe cases of poisoning, completion of the labour is followed by increase of the jaundice, greater tenderness over the liver, diminution in the size of the organ, coma, and death.

The symptoms of industrial phosphorus-poisoning are more of a local than a general character. Acute poisoning, practically speaking, never occurs in lucifer matchmakers. The principal accident that happens to them is necrosis of the jaw-bone. At its commencement this is a very painful affection, and it is not always relieved by extraction of the decayed tooth. It is a periostitis followed by inflammation of the bone and then by necrosis. For many months pus keeps oozing into the mouth from the affected socket, and as some of this during sleep may trickle down the pharynx or be swallowed with the food, the patient runs the risk of infection, especially as the pus contains numerous micro-organisms. Among these Professor Stockman detected a few tubercle bacilli. The glands below the jaw are sometimes enlarged and tender. In France a con-

stitutional condition described as *phosphorism* has been observed in lucifer matchmakers, the principal symptoms of which are a peculiar pallor, attended by slight yellow tinging of the skin and by albuminuria.

Pathology.—In acute phosphorus-poisoning death may supervene in from three to five days. The cadaver is usually jaundiced. In the skin and serous membranes, also in the muscles and between them, may be seen numerous petechial hæmorrhages, the blood in which is dark. On opening the internal cavities a distinct odour of phosphorus may be detected. Lungs may present nothing abnormal. The pericardium is often the seat of numerous small hæmorrhages, so too is the surface of the heart and the myocardium. The diaphragm, too, may be the seat of small hæmorrhages. The liver is greenish in colour, enlarged in the early stages, and so fatty that the greasy material adheres to the knife when the organ is cut. The spleen is usually not enlarged. Kidneys are pale.

On microscopical examination the myocardium and the muscles of the body generally are found to have undergone fatty degeneration, consequent upon imperfect oxidation of cellular protoplasm during life. The numerous ecchymoses are the result of fatty degeneration of the walls of the small blood-vessels. The liver cells are observed to be extremely fatty, and the biliary canals are the seat of catarrh, due to the unhealthy bile having induced a subacute inflammatory condition of their lining membrane. It is to this circumstance that the jaundice is attributed. The kidneys, too, are the seat of well-marked fatty degeneration. By means of osmic acid the fat can be demonstrated in the form of black granules. It is largely owing to the destructive changes induced in the cells of the liver and kidney that there occurs retention of animal products within the body, which is the cause of the coma that is invariably fatal. On examining the stomach in very recent cases the mucous membrane is found to be red, and the seat of hæmorrhages. The brain and nervous system exhibit nothing very abnormal.

In lucifer matchmakers phosphorus-poisoning takes the form of necrosis of the jaw-bone, more frequently of the lower than the upper. It has not yet been definitely settled as to how exposure to phosphorus causes death of the bone. Formerly it was considered to be the sole result of the action of phosphorus fume, *i.e.* a mixture of phosphorus and phosphoric oxide, operating through a decayed tooth upon the socket and setting up periostitis. It is generally admitted that work-people with decayed teeth are more susceptible than others. Professor Stockman of Glasgow found tubercle bacilli in the pus that escaped from a necrosing jaw of a matchmaker, and he therefore considers the process to be tubercular; but as there are many kinds of micro-organisms in the mouth, and also in decayed teeth, it is difficult to say whether the necrosis is entirely tubercular. Exposure of dogs and monkeys to the fumes of phosphorus, with, in addition, inoculation of the gum by a culture of tubercle bacilli, is not followed by necrosis of the jaw-bone. There is something peculiarly human underlying this question of phosphorus necrosis in lucifer matchmakers. Once the disease is established and the decayed tooth removed, the affected bone, if not treated surgically, may keep discharging pus for several months, when a sequestrum comes away, and the bone heals, leaving often very little deformity. Where the necrosis affects the upper jaw-bone there is a tendency for meningitis to be induced, which becomes purulent, and is rapidly fatal. In some of the patients tubercular lung disease is established, and in others there is albuminuria, which may be due to amyloid degeneration of the kidneys.

At Grammont in Belgium, which is one of the principal seats of the manufacture of matches in that country, I had the opportunity of discussing the question of necrosis with Dr. Brocorens, who has had a large experience of the maladies of lucifer matchmakers. During twenty-five years he has met with thirty cases of spontaneous fracture of the long bones of the limbs in matchmakers who had previously suffered from necrosis of the jaw due to their occupation. This accident suggests that in addition to the local effects produced upon the maxillæ, phosphorus is capable of setting up pathological changes in other bones, whereby they break without any strain at all. Fracture of the femur in matchmakers in England is not unknown. Dr. Dearden of Manchester reported the case of two dippers, each of whom had at different times both thigh bones broken in the simplest manner possible.

Diagnosis.—Acute and subacute phosphorus-poisoning resembles acute yellow atrophy of the liver, but in the latter there is usually deeper coma, and the liver diminishes rapidly in size, whereas in phosphorus-poisoning the onset of jaundice is attended by enlargement of that organ. The spleen, if enlarged at all, is usually more so in acute yellow atrophy of the liver than in phosphorus-poisoning. Occasionally even at the autopsy it is difficult to differentiate between the two. Acute yellow atrophy is prone to develop during pregnancy, and it is when women are pregnant that they often, as we have seen, resort to phosphorus to cause miscarriage. Dr. Vivian Poore maintains that clinically acute yellow atrophy of the liver and phosphorus-poisoning are indistinguishable. Hypertrophic cirrhosis of the liver has been mistaken for phosphorus-poisoning, but the symptoms are usually not so acute. Uræmia with jaundice might under certain circum-

stances also resemble it, but an examination of the urine for albumen would to some extent help to settle the diagnosis.

The *prognosis* is grave if a large quantity of phosphorus has been taken. Fully one-third of the patients die, many of them within three days of taking the poison. Those patients who survive this period may die from syncope, due to fatty heart.

Treatment.—The main object is to prevent absorption of the poison from the stomach, and thereby the pathological changes in the liver and kidneys upon which the secondary poisoning and coma depend. Emetics and purgatives should be administered early, but it is preferable to wash out the stomach with plenty of lukewarm water until all odour of phosphorus has disappeared, adding to the water towards the end of the lavage copper sulphate (1 in 100), since this forms, with any phosphorus that has been left in the stomach, an insoluble phosphide of copper. Permanganate of potassium may be substituted for the copper salt. The administration of spirits or oil of turpentine will be found to be as near an antidote to phosphorus as possible. All fatty food, *e.g.* milk and oily substances, must be avoided, for these dissolve phosphorus, and therefore favour its absorption. The essential oils, such as turpentine and cloves, also such a liquid like ether, check the glow or phosphorescence of phosphorus, that is to say, its oxidation; it is when in this transitional state that phosphorus is harmful. Saline aperients should be administered. Castor oil, for the reasons just stated, must be avoided. The possibility of syncope coming on should be anticipated by the administration of heart tonics and stimulants, *e.g.* digitalis and ammonia. Treatment of the necrosed jaw-bone may be expectant. By means of antiseptic mouth-washes the effects of the diseased bone may be materially held in check. A sequestrum of varying size may come away after a few months, or the piece of the jaw-bone may be removed by the surgeon. Both lines of treatment give practically the same results. Most of the patients recover.

To check industrial phosphorus-poisoning regulations have been framed by the Home Office. Of these, personal cleanliness of the work-people, the introduction of fans for improving the ventilation of dipping- and drying-rooms, also of the tables where the boxing is carried on by hand, and periodic dental inspection of the workers are the principal points. Efforts are now being made to manufacture strike-anywhere matches without yellow phosphorus at all. In France they have succeeded in making matches from sesquisulphide of phosphorus. These have most of the advantages of the strike-anywhere matches, without being poisonous. The demand by the public for safety matches should be encouraged.

Bisulphide of Carbon-Poisoning and India-Rubber Manufacture

The india-rubber trade gives employment to a large number of men, women, and young persons. The increasing uses to which electricity is being put has given a stimulus to this industry. India-rubber is used for insulating electrical wires, also in the manufacture of waterproof garments, toys for children, tobacco-pouches, etc. In the factories where these articles are made, large quantities of naphtha are used to dissolve the rubber. The pungent vapour of the naphtha is at first very irritating to the eyes and the respiratory passages, but the workers soon get accustomed to this. What the work-people complain of is that the taste of the naphtha lingers in the mouth long after they have left the factory, so that it prevents them taking food with any relish. Partly owing to this circumstance, and to the fact that the work is carried on often in overheated and ill-ventilated rooms, the girls become anæmic, and suffer much from headache. Beyond these facts and the discomfort which naphtha vapour sometimes causes, it cannot be said that any serious illness has been traced to its employment. It is otherwise with those processes in the manufacture of india-rubber goods in which *bisulphide of carbon* is used. India-rubber has to be vulcanised, in order to render it capable of withstanding alternations of heat and cold. Vulcanisation of rubber goods is generally done by means of sulphur burned in close chambers or by using bisulphide of carbon. It is with the latter we are chiefly concerned. Bisulphide of carbon confers pliancy and elasticity upon india-rubber, and prevents it from becoming hard. The finer goods, such as children's balloons and tobacco-pouches, are dipped in bisulphide of carbon, a liquid that gives off a most offensive and repellent odour; it is also very volatile and its vapour is inflammable. All manufacturers are agreed that it is an excellent compound for the purpose to which it is put. Several substitutes have been tried, but as yet none has replaced it. For the vulcanisation of india-rubber 2 to 10 parts of chloride of sulphur are generally added to 1000 of bisulphide of carbon. As it is a very inflammable substance, the processes in which it is used are carried on in the top story of the factory, and in rooms without a fireplace or naked lights.

Symptomatology.—Inhalation of the vapour of bisulphide of carbon has a peculiar intoxicating effect upon some of the work-people. Prolonged exposure to the vapour causes peripheral neuritis. Men and women lose the power in their arms and legs, and once paralysis is developed it is slow to disappear. Before the paralysis occurs there is often observed a staggering gait. Inhalation of the bisulphide vapour causes the work-people to be drowsy. They sleep heavily over night, and waken often with a splitting

headache in the morning. The girls and young women who dip balloons and tobacco-pouches in the bisulphide suffer from headache and vomiting, accompanied by dizziness, and they complain of tasting the nauseous compound in their food. On going home from the factory at night they sometimes stagger and reel as if intoxicated. They even fall. After reaching home they are so tired that they often go to sleep before touching their evening meal. This sleep is heavy and unrefreshing. Next morning they proceed to their work languid and feeling nervous and shaky. They do not feel comfortable until they have again inhaled some of the vapour of the bisulphide. Upon some of the workers the carbon compound exercises a more injurious influence than that just described. It causes acute mania. Often the outburst is as sudden as it is unexpected, and in their frenzy affected work-people have been known to throw themselves from the top story of a factory. Bisulphide of carbon also causes temporary loss of vision; in some instances it acts as an ecbolic, while upon young women it occasionally induces symptoms of a peculiar hysterical nature—a mixture of hysteria and intoxication. This toxic hysteria, like that which occurs in plumbism, is often the prelude to a deeper and more serious form of poisoning, and should on no account be underestimated.

Diagnosis.—In acute cases the history of the occupation of the patient and the presence of a disagreeable odour that may possibly be about his clothes would suggest bisulphide of carbon poisoning. The bilateral character of the paralysis would also suggest poisoning, but not its cause. Absence of blue line on the gums would show that it was probably not due to lead. The affected limbs are not so tender to the grasp as in arsenical poisoning. Speaking generally, apart from the history of the case, it would be difficult to say what is the cause of the paralysis.

Treatment.—(1) *Preventive.* Removal of the patient from work. Free ventilation of the workroom and withdrawal of the vapours away from the face of the workers by means of fans. Alternation of employment in the factory. (2) *Medicinal.* In the acute forms of poisoning bromide of potassium with sulphate of magnesia; for paralysis massage should be tried and the application of electricity; internally nux vomica or liq. strychniæ supported by a liberal diet.

Inflammable Paints and Benzine

Inflammable Paints.—The use of composite or spirit paints has been frequently followed by symptoms of poisoning in men engaged in painting ships' holds or confined spaces in ships. These composite paints dry very quickly, and in consequence they are much in demand for ships, to the owners of which time is money. The drawbacks to these spirit paints are mainly two: (*a*) they are so very inflammable that a man cannot or ought not to be allowed to work with them in a confined space with a naked light; (*b*) since they contain large quantities of petroleum or methylated spirit combined with benzine, inhalation of the vapour causes the workmen to become intoxicated, and sometimes to be violently sick. In several instances the men, overcome by stupor, have been unable to leave the compartment, and have had to be removed by means of ropes. On bringing them up to the deck the workmen have been found to be unconscious, to be pale rather than cyanosed, and they have vomited, the vomited matter smelling strongly of the spirit paint. Some of the men, too, have had epistaxis. After being roused into consciousness not a few of them have lapsed into a deep sleep for several hours.

Treatment.—Too great care cannot be exercised so as to prevent the vapours from the paints catching fire. No naked lights should be allowed where men are using them, nor should the men be allowed to work in close and confined compartments for more than an hour or two at a time. They should come out into the open air. On taking up this particular kind of work the dangers of the occupation ought to be clearly pointed out to the men by their employers. Where the symptoms are those of acute intoxication the best treatment is to place the patient in as pure air as possible, so that he may, by ventilating his lungs, rid his blood of the deleterious vapour, care being taken in cold weather to have the body well covered with rugs to avoid being chilled. Subsequently a saline aperient should be administered.

Benzine.—Soiled gloves and garments are cleaned by means of benzine in "dry cleaning" establishments. Occasionally naphtha is used for the same purpose. Benzine is a very inflammable and highly explosive compound. Fires break out during the summer months in dry-cleaning establishments in the most unaccountable manner. They have been attributed to friction, and to electric sparks, while in a few instances they have doubtless been due to accidental ignition of lucifer matches concealed in the clothing sent to be cleaned. As a rule, all the pockets of the clothes are turned inside out and all metallic buttons are removed before the clothing is placed in the revolving cylinders that contain the benzine. Two risks are run by the work-people: (1) danger from fire and explosion; (2) effects due to inhalation of the benzine. Too great caution cannot be exercised in regard to the manipulation of benzine. There should be no naked lights about at the time it is being handled. Benzine, when inhaled, often causes young female workers to become extremely hysterical,

loquacious, and excited. Frequently, too, it causes headache and giddiness. If the work has been conducted in rather a close room, the men and women become mildly intoxicated. Benzine vapour produces a heavy, sleepy feeling, which obliges the work-people to go out into the fresh air.

Treatment.—Preventive by having the work-room well ventilated. In the case of fire the best way to extinguish the flames is by means of sand. Blankets ought also to be in readiness to throw around any worker whose clothing may have caught fire. When there are symptoms of intoxication the work-people should be taken into the fresh air. This is generally sufficient to restore them.

Aniline and Dinitrobenzine

The discovery of the coal-tar products has revolutionised chemistry as applied to the arts and manufactures. Many of these bodies, *e.g.* the picric acid compounds, are used in the manufacture of the higher explosives, while others are used for dyeing purposes. Dearden has described the symptoms observed in workmen employed in the dyeing of fast aniline blacks. Dyeing by means of aniline black agents is indirectly brought about through the medium of an aniline salt or aniline oil. The salt employed is obtained by acting upon aniline with hydrochloric acid in *closed* vessels. When not obtained in this manner a considerable amount of fume is given off, inhalation of which causes great prostration, dyspnœa, intermittency with rapid action of the heart, lividity of the face, blueness of the lips and finger nails. Symptoms of a similar character have been observed by Prosser White in men employed in the manufacture of nitro-benzole. Since nitro-benzole is a frequent impurity in aniline oil, some of the symptoms just detailed may probably be due to this substance. In dye-works the cloths that have been dipped in aniline liquor must be dried and mangled, and subsequently the dye is oxidated and chromed. In the ageing- or chroming-room large quantities of sodium bichromate are used, and workmen who carelessly allow the edge of the cloth to run through their fingers often suffer from chrome ulcers on the hands.

There is an idiosyncrasy to aniline-poisoning. Some persons are more susceptible to it than others. The symptoms of acute aniline poisoning are nausea with prostration amounting at times to collapse; the features become livid and the extremities cold and blue; there is dyspnœa accompanied by a feeling of drowsiness; the pulse becomes small, rapid, and intermittent, the pupils are dilated and the urine dark-coloured. Many of these symptoms can be prevented by better ventilation of the factory, but even in those that are best ventilated it is not uncommon for the workmen to complain of nausea and extreme lassitude, for the face to be livid, and the lips and nails cyanosed.

Poisoning may be prevented by having the rooms in dye-works lofty, cool, and well ventilated. Handling of the chromed goods should only be done by men wearing india-rubber gloves. Personal cleanliness is a necessity. The hands should be well washed before meals and no food eaten in the workrooms. Alternation of employment is desirable. The men should not work long in any of the dangerous processes.

In France several cases of fatal poisoning have occurred, especially in young people, in consequence of a particular kind of paste being used to blacken fawn-coloured boots. The symptoms were the same as those above mentioned. On analysis the paste was found to contain as its principal ingredient aniline oil.

What has been said of aniline will almost equally apply to the nitro- and dinitrobenzine compounds that are used in the manufacture of the higher explosives, roburite, etc. This subject has received literary attention at the hands of Drs. E. S. Reynolds and Prosser White. It has also been treated by Simeon Snell, who found failure of eyesight a common symptom due to concentric contraction of the visual field with central colour scotoma and occasionally blurring of the edges of the disc, appearances that as a rule subside on the men giving up their work. Dinitrobenzine is a powerful poison whether inhaled or swallowed, rubbed into the skin, or injected into the circulation. A little over one gramme, when taken by the mouth, will cause death in less than twenty-four hours. The concentrated vapour is particularly dangerous. Poisoning by dinitrobenzine may be acute, subacute, or chronic. Men who breathe the vapour during work often become sleepy and complain of severe headache; the heart's beat becomes irregular, and the men experience a difficulty of breathing; their face is observed to be cyanosed, and the urine they pass exhibits a reddish brown colour free from albumin and sugar, but which on spectroscopic examination gives the spectrum of methæmoglobin with some other pigment not yet definitely named. In acute poisoning death is preceded by coma. At the autopsy the brain is found to be congested, the blood dark and thick, the chambers of the heart are dilated, and the lungs either dark or very pale. There is often exhaled from the body a strong odour not unlike that of bitter almonds. A similar odour is detected in prussic-acid poisoning, but the symptoms caused by prussic acid are more sudden in their development than those of dinitrobenzine-poisoning.

In the chronic cases the skin becomes dirty yellow; the men complain of feeling languid and of being sleepy, and their gait is often ataxic. Occasionally the skin is at places anæsthetic or hyperæsthetic, and the urine is of a

deep maroon colour. When dinitrobenzine kills it is generally by inducing coma. The blood corpuscles disintegrate under its influence, and, as a consequence, oxygen fails to be taken into the system. Although chemical changes occur in the passage of these nitro compounds through the body, it is difficult to say what is the exact nature of these changes.

In order to prevent dinitrobenzine-poisoning the workrooms should be airy and well ventilated. The workmen should wear respirators; when handling the material gloves ought to be worn. Personal cleanliness is also most important, and there should be the minimum hours of labour in such dangerous processes as grinding and the cleaning out of flues. Six hours a day spent in the rooms where the mixing is done and the cartridges are filled, are enough for any person. Since alcohol is known to increase the susceptibility to poisoning, stimulants ought to be avoided by men following this employment. There is no known antidote to dinitrobenzine; but in the acute form of poisoning, inhalation of oxygen, the application of warmth to the surface of the body, careful administration of stimulants, and rest in the recumbent position until immediate danger from syncope has passed away, will be found very helpful.

Risks attending the Manufacture and Use of Explosives

Unless the strictest precautions are observed there is no manufacture more dangerous to life than the manufacture of the so-called high explosives, *e.g.* nitroglycerine and gun-cotton, dynamite, roburite, lyddite, etc. Major Cooper Key, H.M. Inspector of Explosives, states that in ten years there were 44 workmen killed in explosives factories in Britain and 204 injured. No doubt many of the accidents were caused through carelessness, *e.g.* thawing of dynamite over the fire, scraping out of detonators with pins, etc., while in quarries and mines many of the accidents have been caused by driving dynamite into roughly drilled holes, boring out missfires, etc. In the actual manufacture of explosives the workmen run little risk to health. The risk is rather one of sudden mutilation from the severity of an explosion.

Miners often complain of the effects of the fumes after an explosive cartridge has been fired in the pit. They suffer from severe headache, collapse, a sense of nervousness, muscular tremor, alternate flushing and paling of the face, followed by vomiting which often brings relief. When coal-miners are the subjects of chronic pulmonary or bronchial affections their breathing for the time is rendered difficult, and the men are so prostrated that they almost collapse. Since all explosives contain carbon, these when fired either in a sufficiency or in an excess of oxygen form carbonic anhydride (CO_2), a poisonous gas, but when the oyygen is deficient carbon monoxide (CO) is formed, which is still more poisonous. While gunpowder may give off on combustion 3·6 per cent of carbon monoxide, gun-cotton will evolve as much as 28 to 45 per cent of this gas. Haldane has written upon the effects of carbon monoxide upon coal-miners. In his experiments upon animals he found that an atmosphere containing 0·4 per cent of carbon monoxide caused death, and that anything above 0·2 per cent was dangerous. As the gas is, practically speaking, odourless, its presence is not easily recognised. When inhaled it causes dizziness and dyspnœa, attended by palpitation and a sense of drowsiness, or there is experienced a feeling of excitement resembling that caused by alcoholic intoxication. These symptoms are followed by loss of muscular power, mental confusion, and by unconsciousness. Carbon monoxide prevents the absorption of oxygen by the blood owing to the gas itself having entered into stable combinations with the hæmoglobin of the red corpuscles, proof of which is found on examining the blood spectroscopically. The blood is of a bright red colour, shows two bands in the yellow and green of the spectrum, and is not reduced by ammonium sulphide.

All the explosives, with the exception of nitro-glycerine and some of the ammonium nitrate products, evolve, when exploded, carbon monoxide. Nitro-glycerine, on the other hand, gives off carbonic anhydride, sometimes to the extent of 58 per cent. There are in addition nitrous fumes, and while these of themselves are dangerous their effects are exaggerated by the presence of CO and CO_2. Gunpowder gives off on ignition carbonic anhydride and nitrogen, with a small proportion of carbon monoxide and sulphuretted hydrogen. Gun-cotton, tonite, gelignite, and carbonite evolve a fairly large percentage of carbon monoxide, and are therefore dangerous when exploded in places where the ventilation is defective. The headache complained of after the firing of the high explosives is due to dilatation of the cerebral arteries, which is one of the physiological effects of nitrites.

Workers in the Chemical Trades

Although many chemical compounds are used in the arts and manufactures, the term chemical trades is usually restricted to the production of alkali, bleach powder, sulphuric acid, etc. Owing to the fact that the production of a large number of chemical compounds is generally conducted by the wet process, and that these compounds are either crystallised out of or are precipitated from solutions, there is less danger to health in their manufacture than when they are subsequently handled or inhaled in the form of dust. Any possible danger from gas in chemical works is obviated by ventilation. In

the chemical trades the principal goods manufactured are hydrochloric and sulphuric acids, sodium carbonate, caustic soda, caustic potass, and bleaching powder. These are usually all produced in the same factory. In what is known as the Le Blanc process, sulphate of soda, or salt cake, is first formed by acting upon common salt with sulphuric acid. These two compounds are heated together on the bed of a furnace, and raked from time to time until the salt decomposes. Large quantities of hydrochloric acid are given off and removed by means of a blower. As this gas is extremely irritating to the lining membrane of the respiratory passages, the workmen are obliged to wear a flannel muffler tied over their face, or they bite a piece of flannel held between their teeth and breathe through it. As a consequence either of biting the flannel or of the operation of the acid fumes themselves, the teeth rapidly decay. The workmen, too, are exposed to great changes of temperature, and readily catch cold. After removal of the salt cake from the furnace it is mixed with limestone and coal in a black ash furnace; there it is heated and decomposed. This is a process apparently unattended with risk to the health of the operative. The soda having been dissolved, can be converted as required into soda ash, soda crystals, or caustic soda. The liquor containing the caustic soda has to be concentrated by heat. By degrees the cauldron holding the liquor becomes occupied by red-hot caustic soda. This is subsequently baled out into iron drums and allowed to cool, after which it is ready for the market. Considerable danger attends this process. A few drops of water falling into the liquor causes it to spurt. Beyond burns of a serious character having occurred in this process, there is nothing in it that can be said to be absolutely unhealthy to the workmen. It is different, however, with the bleaching powder made from the hydrochloric acid given off from the salt-cake furnace. Chemical manufacturers require the chlorine obtained from the hydrochloric acid, and this is got either by decomposing the acid in large stills by means of manganese mud, the gas being led away by pipes to the bleaching chamber (Weldon process), or the hydrochloric acid gas, mixed with proper proportions of air and steam, is conveyed thither direct from the salt-cake furnace (Deacon process). A. P. Laurie, whose description of the various processes I have followed, tells us, in a paper on "Chemical Trades," that in order to prepare bleaching powder freshly slaked lime must be exposed to chlorine gas. The lime absorbs the gas, and retains it only very slightly, for it is constantly evolving the chlorine.

The packing of the bleach powder is one of the most disagreeable operations a man in a chemical works can do. He is obliged to enter the bleach chamber, which contains free chlorine, and to shovel the powder through holes in the floor into casks. Before he can do this the packer must wrap his face in rolls of flannel, the mouth being covered, but his nostrils free. It is necessary to keep the flannel just sufficiently damp to prevent the gas reaching the lungs. Leather goggles are put on to protect the eyes, while paper leggings are worn to protect the trousers. As the man begins to shovel the bleach into the casks the chlorine gas at once rises, and were the workman not protected in the manner described, it would be impossible for him to remain more than a few seconds in the bleach chamber. As it is, the flannel wrappings cause the breathing to be very difficult and most trying. The bleach-packer when in the chamber is obliged to inhale by the mouth, through the wet flannel, and exhale by the open nostrils.

Chlorine gas is extremely irritating to the respiratory mucous membrane. When inhaled it causes an unpleasant, suffocative feeling. Breathing of the gas is attended by a considerable degree of discomfort, and when pushed too far causes insensibility and death.

In the manufacture of sulphuric acid iron pyrites is burned, and the sulphur dioxide as it escapes is led away into large lead chambers, where it is brought into contact with air, steam, and nitrous fumes, the latter having been obtained by acting upon sodium nitrate with sulphuric acid. Occasionally sulphur dioxide and nitrous fumes escape, and are inhaled by the workman. These gases are irritating to the respiratory mucous membrane.

Attempts are now being made, and on the whole successfully, by chemical manufacturers to recover the sulphur from the black ash left in the vat after the preparation of soda. The ash in question is rich in sulphide of lime. Hitherto this has been looked upon simply as a waste product. Large mounds of it can be seen in the neighbourhood of alkali works. These heaps when acted upon by rain and air give off large quantities of sulphuretted hydrogen. Some of this waste is taken and mixed with water; the mixture is subsequently placed in large closed vessels, and carbonic acid passed through it. Decomposition is effected, sulphuretted hydrogen being given off and carbonate of lime precipitated. The sulphuretted hydrogen is thereafter burned, sulphur and water being formed. The gas itself is not only very unpleasant, it is extremely poisonous. On allowing a dog to breathe an atmosphere containing 0·2 per cent of sulphuretted hydrogen, I have seen the animal fall down apparently dead after 1½ minutes' inhalation of the gas; its breathing was completely arrested, while the heart continued to beat for a short period after death. An animal thus apparently killed by sulphuretted hydrogen might recover if placed in purer air and caused to breathe oxygen by

artificial respiration. Whenever men working in chemical factories, cesspools, or sewers are brought into contact with sulphuretted hydrogen, cylinders of oxygen should be ready in case of emergency, and when men are seen to fall down they should be at once brought out into the open air, and artificial respiration adopted. In addition it may be necessary to give them a hypodermic injection of liquor strychniæ, and to administer stimulants.

Chemical labourers, as a rule, are strong, healthy men to start with. Many of them, unfortunately, become intemperate, and probably, therefore, part of their ill-health may be due to this circumstance. Their work is hard. They are paid good wages, but as they spend their earnings freely on drink, many of them feed badly. The tendency to alcoholic excess is, no doubt, largely due to the nature of their employment. Diseases of the respiratory organs are common in chemical workers, but the death-rate from phthisis is below the normal for all males. How far this circumstance is due to sulphur dioxide, hydrochloric acid, nitrous fumes and chlorine being unfavourable to the multiplication of tubercle bacilli, it is impossible to say. Some medical men hold the opinion that these gases are prejudicial to the development of the bacillus. In chemical works the number of deaths from accidents is unusually large. If we take 56 as the number of deaths for all males, chemical trades stand at 98, whereas shipwrights, who also have a high morality from accidents, number 63.

The Manufacture and Uses of the Bichromate Compounds.—Although the manufacture of the bichromates is a special industry, still the subject may be conveniently dealt with under chemical trades. Workers in bichromate compounds are prone to suffer from erosin of the septum nasi and from ulcers of the skin. Erosin of the nasal septum occurs in those who manufacture sodium and potassium bichromate, but all who handle the salts are liable to ulceration of the skin. In making potassium bichromate, a mixture of chrome ironstone, potash, and lime is roasted together. Afterwards the fused mass is lixiviated with water, sulphuric acid being subsequently added to convert the neutral chromate into bichromate. The chrome ironstone has first to be reduced to a fine powder before it is mixed with the lime and potash. When the mixture has been roasted for three hours it is withdrawn and allowed to cool, after which it is broken up and shovelled into large vats. It is when water and the solution of potassium sulphate are added that steam is given off which carries with it particles of chromate dust. After the addition of sulphuric acid the potassium bichromate in solution is run off into lead tanks, where it crystallises. The crystals have to be subsequently broken up by the workmen, and it is this process which is a source of danger in bichromate factories. The air of the room frequently contains, according to Heise, as much as 6·3 milligrammes of sodium bichromate per cubic metre. Dr. T. M. Legge, H.M. Medical Inspector of Factories, who has given considerable attention to this subject, found the nasal septum of the workmen frequently perforated. Some of the men were more susceptible than others. On an average, perforation of the septum occurred after six to twelve months' work, but others of the men who thus suffered had only worked as many weeks. The site of the ulceration is about a quarter of an inch from the lower and anterior margin of the septum. When perforation occurs it is extremely limited at first, but after the mucous membrane has been destroyed the ulceration progresses rapidly, and extends as far up as the junction of the septum with the ethmoid and vomer. The loss of substance is preceded by symptoms of nasal catarrh and sneezing. Pain is not a prominent symptom. Even when perforation has taken place the general health remains good. There is, however, considerable inconvenience from the formation of plugs of mucus in the nares and from crusting of the discharge. The "chrome holes" or ulcers observed on the skin are sluggish and slow to heal. They are generally caused by a cut or wound of the skin becoming irritated by the bichromate. Men, therefore, who have wounds on the skin should abstain from work for a time. Personal cleanliness, too, is most important. The men when working with bichromate solutions should wear india-rubber gloves. Dust should be removed by fans.

Workers in Arsenic

Arsenic, in the form of arsenite of copper, Scheele's green, and Scheinfurt or Vienna green, has long been used as a colouring agent for wall-papers, for dyeing purposes, in the manufacture of artificial flowers, and the fluid known as "sheep dip" for destroying tick on sheep. Although wall-papers containing arsenic are usually coloured green, yet red, orange, and brown papers may contain the poisonous metal. It is from the dust given off by wall-papers, and arseniuretted hydrogen produced by vegetable moulds, that people have suffered through sleeping in bedrooms the walls of which had been lined by arsenically pigmented papers. The men who make Scheele's green often have pimples on the skin that develop into boils. Others suffer from ulcers of the skin, or complain of headache, nausea, and vomiting. Men who make sheep-dipping fluid, and who are not careful to wear india-rubber gloves, may suffer from paralysis. Arsenical neuritis affects, as a rule, the lower extremities. It is this location of the paralysis and the extreme pain complained of in handling the limb that differentiate the lesion from lead-poisoning. The arms and

hands, however, may also become affected. The arsenic reduction workers in Cornwall suffer from bronchitis and pulmonary diseases in excess of men in the same locality following other occupations. The arsenic powder, too, gets under their clothing and causes eczema. In deference to public opinion, manufacturers have lately discontinued the use of arsenic as a colouring agent for wall-papers, other substitutes having been found. Workers in arsenic cannot pay too much attention to personal cleanliness.

Copper and Brass—Brass-Founders' Ague

The copper ore smelted in this country is known as copper pyrites. As this often contains arsenic, serious symptoms of poisoning have occurred in smelters, probably due to the arsenic. Birmingham is the centre of the brass trade. Brass is an alloy of copper and zinc. In the fusion of these metals, and particularly during the pouring of the same when molten, the workmen have been known to suffer from a peculiar set of symptoms to which the name of "brass-founders' ague" has been given. During the pouring of the molten brass into moulds, dense white clouds of smoke arise and fill the interior of the casting-shop. From these clouds a delicate white powder gently falls, and this, on analysis, has been found to be oxide of zinc. As far back as 1862 Dr. Headlam Greenhow read a paper at a meeting of the Royal Medico-Chirurgical Society on Brass-Founders' Ague. It is now admitted that this name is misleading. Since Greenhow's time the subject has attracted the attention of Hogben, and more recently has been dealt with by Drs. Robert M. Simon and Murray of Birmingham. Opinions are divided as to whether the so-called ague symptoms are due to the copper or the zinc in the brass. It is usually when the operatives are fresh to the work, or when the men have been absent for a time, that they suffer. The symptoms are languor, depression of spirits, and a feeling of chilliness. The individual becomes pale and he has a shivering. His teeth chatter, and his face is seen to be wet with a cold perspiration. There are headache, nausea, and muscular pains. Vomiting occurs, and this is followed by a sense of relief, after which the patient often falls into a comfortable sleep from which he awakes, feeling well, though tired and weak. Both Greenhow and Hogben described a hot and sweating stage as succeeding the one just mentioned. Simon has never met with these. He therefore thinks that the term ague should be discarded altogether. In brass-workers the teeth are often green, and the gums blackened at their edges. The white hair of the older workmen is usually coloured green. As a rule the symptoms detailed above are transitory. In addition to the so-called ague many of the men suffer from bronchitis and fibroid phthisis. Brass-casters, too, are often the subjects of nervous disorders not unlike paralysis agitans. Dyspeptic symptoms are common. There are loss of appetite and gastro-intestinal catarrh; complaint of a metallic taste in the mouth, vomiting, and either constipation or diarrhœa. The men often become nervous and hypochondriacal, or they complain of headache and obscure muscular pains. A similar train of symptoms is known to occur in copper-poisoning. Stephenson says it is impossible to say whether the symptoms in brass-workers are due to zinc or copper. Greenhow attributes them to deflagrating zinc, while Hogben blames the copper. It is difficult to say which of the two metals is the cause of the symptoms. Zinc is not toxic to any extent by itself, and copper, although usually considered poisonous, is much less so than hitherto believed. Metallic copper is not at all dangerous, and copper oxide is only so in very large doses. It is just possible that the fumes given off from the molten brass are poisonous owing to some peculiar and unexplained condition in existence at the time, and therefore due to the combination of the two metals. Simon holds that the ague-like symptoms are probably caused by this admixture, while the more chronic complaints he attributes to the copper.

Milk has been proved by experience to be the best preventive of brass-poisoning, and by many it is also regarded as an antidote. The workmen resort to it on their own account. As a rule these men do not call in medical assistance for the ague. The workrooms ought to be well ventilated, so that the fumes of the molten metal may escape. It is during heavy, foggy weather, when it is difficult to clear the air of the workshops, that symptoms among brass-workers are most prone to occur.

The Textile Trades

Cotton

In the manufacture of cotton the operatives incur no special risks other than those due to working in ill-ventilated rooms, and where machinery is running at high speed. For trade purposes it was found necessary to introduce moisture into the air of the workrooms in cotton factories, and as this has been carried on frequently in excess of manufacturing requirements, the operatives have suffered in their health. It is in the weaving-sheds where this artificial humidity is especially needed. Raw cotton as seen in the bales is often dirty and dusty. It has therefore to be cleaned and combed. In the process of *carding* or combing of the raw material a considerable amount of dust is given off, while in the *spinning* rooms, owing to the outside air being excluded so as to keep the yarn clean, and in consequence, too, of the rapid rate at which the machinery is run,

not only does the air become impure, but the temperature rises as high as 90° to 100° F. As no moisture is introduced into these rooms the air becomes extremely dry. For the *weaving* of cotton, on the other hand, moisture is said to be necessary. Weaving-sheds are therefore usually built in damp places: they are low-roofed, their height on an average being from eleven to fifteen feet from the flagged floor to the ceiling; the light enters by the roof. The health of the weavers is endangered by impurities in the air, by the products of respiration and the combustion of gas, by cotton-dust and emanations from the soil. The sanitary conveniences for the work-people are often built too close to the walls of the factory. To these defects must be added excessive moisture of the air in the shed, and a high temperature, especially in summer. Within the last forty years, and largely in consequence of the scarcity of cotton during the American War, manufacturers have "sized" the cotton, *i.e.* have passed it through liquid containing starch, china clay, tallow, and such deliquescent salts as magnesium chloride, to which small quantities of zinc chloride have been added to prevent the growth of mildew. Sizing of the cotton, along with excessive humidity of the air, has been regarded as a cause not only of discomfort, but of ill-health. During the weaving of the sized cloth a considerable amount of dust is given off. Steam is therefore infused into the weaving-shed with the object of enabling more of the admixture to be woven into cloth and of softening the stiff, oversized threads, thus rendering them soft and pliable and less likely to break. The clothes of the women and young persons who work in the weaving-sheds are frequently so damped by the moisture that on leaving the factory, Dr. Wheatley, formerly M.O.H. Blackburn, says they readily get chilled, become the subjects of rheumatism, and, what is worse, of pulmonary disease.

The size itself adds considerably to the dust in the air of the weaving-shed. The amount of dust is determined by the quantity of the size used and the quality of the cotton. Fifty per cent of the dust on the floor and rafters of weaving-sheds was found by Dr. Dupré to be insoluble mineral matter (china clay), 30 per cent insoluble organic matter (cotton), 15 per cent soluble organic matter (starch), and 5 per cent soluble mineral matter (chloride and sulphate of magnesium).

Since steaming in cotton manufacture must be allowed, the humidity of the air in weaving-sheds is regulated by an order from the Home Secretary, which states that the water used for steam must be pure; it also regulates the length and calibre of the pipe that conveys the steam, and it requires that the amount of carbonic acid in the air of the shed must not exceed nine volumes per 10,000 of air. The moisture of the air is tested by a hygrometer. It is difficult to say what is the effect upon health when operatives are obliged to breathe an excessively moist atmosphere for ten hours a day. There is no doubt, however, as to its debilitating effects, and it is probably in consequence of this that tubercular diseases and rheumatism are so frequently met with in cotton-weavers. Ventilation has, to some extent, improved matters. Although it is admitted that ·9 part of CO_2 per 1000 of air is not a high standard of purity, yet in order to obtain this it is necessary to introduce as much as 2000 cubic feet of air per head per hour.

While the death-rate of cotton operatives from phthisis is little above that of all males throughout the country, there is a high mortality from pneumonia and bronchitis, from rheumatism and heart disease. Formerly the deaths of cotton operatives in Lancashire from phthisis were 25 per cent of the total deaths (1880-1882), but in the years 1890-1892 these had fallen to 19·6. During the same periods bronchitis and pneumonia showed an increase from 25 to 29·7 per cent.

Flax and Linen

Belfast is the seat of the linen trade. Although the manufacture of linen from flax is a very old industry, it has only been carried on in Ireland for a little over two hundred years.

In the carding- and spinning-rooms of the factory there is often a great amount of dust, and as the atmosphere is extremely moist and overheated, the workers, especially the half-time doffers, become liable to bronchitis. During the summer months the spinners, in consequence of being on their feet all day and moving about, often without boots, on a wet floor, suffer from œdema of the ankles, onychia, and eczema. Dr. Henry S. Purdon of Belfast found the hands of spinners frequently the seat of eczema rimosum. On the hands of Belgian flax-dressers Dr. Glibert, Medical Inspector of Factories, Brussels, observed a few small round ulcers. Young doffers, *i.e.* the boys and girls who remove the filled bobbins from the spinning-frame, and who replace these by empty bobbins, usually suffer from "mill fever" after they have worked for a few days, an illness which lasts from two to three days, and disappears without treatment. The symptoms of "mill fever" are nausea, vomiting, headache, thirst, and feverishness.

Tragacanth.—A gummy exudation from *Astragalus gummifer* and other species of Astragalus. Its chief constituents are *Tragacanthin*, a gum, and a little starch. *Preparations*—1. Glycerinum Tragacanthæ. 2. Mucilago Tragacanthæ. *Dose*—1-4ʒ. 3. Pulvis Tragacanthæ Compositus. *Dose*—20-60 grs. Tragacanth is one of the constituents of Unna's Jelly. The chief use of Tragacanth is to sus-

pend insoluble bodies such as resins and oils and insoluble powders. For such purposes the mucilage is to be preferred. The Glycerinum is a useful pill excipient.

Tragus.—A small cartilage of the external ear (Gr. τράγος, a goat) projecting over the meatus auditorius externus. *See* EAR, EXAMINATION OF THE (*Pinna*).

Training. — Education by practical manœuvres which gradually gives the power of doing easily complicated processes; muscular exercise, special diet and regimen as in preparing for sports; also, the early education of the blind or mentally defective in the ordinary acts of life, walking, dressing, feeding themselves, and the like. *See* BLINDNESS (*Home-Training*); NURSING, TRAINING FOR; PHYSIOLOGY, DIETETICS (*Energy Requirements*).

Trance.—A form of abnormal sleep, due commonly to hysteria or hypnotism, and so profound that it is very difficult to arouse the patient from it. *See* CATALEPSY; HYPNOTISM; HYSTERIA, SLEEP, NORMAL AND MORBID; UNCONSCIOUSNESS.

Trans-.—In compound words *trans-* (Lat. *trans*, across) means through or across, e.g. *transfix* (to pierce through and through), *transforation* (the act of boring or perforating, as in craniotomy or basilysis), etc.

Transference. — Thought-reading or mind-reading; suggestion in hypnotism. *See* THOUGHT-READING.

Transfusion.

USES OF 101
METHODS—
(*a*) *Immediate* 102
(*b*) *Mediate* 102
(*c*) *Transfusion of Blood with Phosphate of Soda Solution* . . 102
(*d*) *Injection of Saline Solution* . 102

See also BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Treatment*); ECLAMPSIA (*Treatment*); LABOUR, POST-PARTUM HÆMORRHAGE (*Treatment*).

THE transfusion of blood is an operation of some antiquity. History relates that so long ago as in 1492, it was practised in the case of Pope Innocent VIII.

It has been performed from time to time during the last century, and comparatively recently was often employed in severe cases of post-partum or other hæmorrhage.

In such cases of sudden hæmorrhage the operation is not called for, and should never be performed. In the infusion of saline fluids we have a simpler and safer proceeding, which satisfactorily fulfils the indication in a sudden and profuse bleeding of filling the emptied heart and vessels, and maintaining the intravascular pressure at a point at which the heart is able to carry on the circulation, until new blood has time to be formed.

There are, however, certain conditions other than sudden hæmorrhage in which blood-transfusion may be, and has on many occasions proved itself to be, beneficial. In pernicious anæmia, a condition little amenable to other treatment, sufficient cases have been successfully treated by blood-transfusion to encourage further work in this direction; while in certain cases of persistent recurrent hæmorrhage, which were operated upon by the writer, transfusion of blood put a stop to the obstinate bleedings, when all other forms of hæmostatic treatment, local and general, had failed. In these cases the operations which were originally undertaken for the relief of the anæmia resulting from persistent small hæmorrhages, had the unexpected, but undeniable, effect of curing, not only the anæmia, but also the bleeding which had caused the anæmia.

The exact value of blood-transfusion in pernicious anæmia, recurrent hæmorrhages, and other diseased conditions, such as septicæmia, must be considered to be still *sub judice*; and it is well to remember that, in judging of such a method, an ounce of unmistakable clinical success is of more value than a ton of adverse laboratory speculation.

The arguments brought against blood-transfusion are, firstly, the immediate danger of the operation from the introduction of air, clots, or septic material, and the formation of thrombi, especially in the portal and pulmonary capillaries; and, secondly, the supposed uselessness of the procedure, because of the breaking up of the corpuscles of the blood which has been injected.

With regard to the first objection it may be said that the introduction of air, clots, or septic organisms is due to faulty operative methods, and is avoidable; while the danger from thrombosis, apart from the introduction of palpable clots, is somewhat problematical. With regard to the second objection, it may be the case that the corpuscles break up, and that this event may be followed by a certain amount of fever and hæmoglobinuria: fortunately these occurrences do not appear to be dangerous, or necessarily to interfere with the good effects of the operation. Nothing can be more certain than that the corpuscular improvement noted in several cases of pernicious anæmia has been out of all proportion to the small amount of blood introduced; this improvement, which is often progressive, is evidently due to a stimulation of the blood-forming organs by the new blood thrown in; but in what particular part of the injected blood this stimulant property lies is doubtful; possibly it is in the serum and not in the corpuscles at all.

Methods of Transfusion of Blood.—(1) *Immediate Transfusion.*—In this method pure blood is passed directly from the blood-giver to the patient by means of some form of syringe, the instruments of Roussel and Aveling being those most widely known. Immediate transfusion need not be described; it need only be mentioned to be condemned. The risk of introducing clots is very great, while the difficulty of preventing the admission of air and sepsis is considerable.

(2) *Mediate Transfusion.*—Here the blood is defibrinated before injection. The blood being caught in a sterilised vessel standing in hot water (at a temperature of about 105° to 110° F.) is thoroughly defibrinated by whipping, and is then strained through fine muslin into a second warmed vessel; thence it is drawn up into a clean warmed glass syringe, and slowly injected through a short glass and rubber nozzle into the patient's vein. In this method one disadvantage is, that a large quantity of blood is required, a good deal being wasted in the whipping process.

(3) *Transfusion of Blood with Phosphate of Soda Solution.*—As this method is by far the safest and most satisfactory, it will be described a little more fully.

The health of the blood-giver, who should not be beyond young adult life, must be beyond suspicion.

In patients who have suffered from anæmia for some time, the veins are often much diminished in calibre, so much so that, for example, the median basilic vein may scarcely admit an ordinary probe.

In such cases it may be necessary to select some larger vein, such as the saphena, in order to introduce even a fine glass nozzle.

The parts concerned, both in blood-giver and in patient, must be carefully sterilised, and the operation performed aseptically.

A suitable vein having been selected in the patient, it is exposed and two catgut ligatures passed beneath it. The distal ligature is then definitively tied, but the ends are not cut off yet, as they are useful to lift the vein and steady it when opening into its lumen subsequently. The blood-giver is next bled to the extent of from 4 to 6 ounces, in such a way that the blood may fall neatly into a vessel containing the phosphate of soda solution.

This solution is of the strength of 1 to 20. One part of such solution must be mixed with three parts of blood in order to delay coagulation. If the blood appears unusually rich, a rather larger percentage of solution may be advisable.

Two ounces of phosphate solution should be warmed to 105° F., and poured into a flat graduated glass vessel, which should be floating in another basin containing hot boric solution.

The blood-giver is bled direct into the vessel containing the phosphate solution, and the mixture is gently stirred with a glass rod. When the necessary amount of blood (about 6 ounces) has been obtained, an assistant attends to the blood-giver's arm, while the operator now opens the patient's vein, which he had previously exposed, and inserts a small glass nozzle, 1½ inches long, having 1 inch of india-rubber tubing attached to its end. This nozzle must be filled with phosphate solution, and clamped before introduction into the vein, or else air may be admitted. The nozzle is fixed temporarily in place by the first half of a "surgeon's knot," made on the proximal piece of catgut originally passed under the vein. A large glass syringe is now filled with the blood and phosphate solution, and this must be kept warm by wrapping the syringe round with lint wrung out of hot boric lotion. The mixture is then injected into the patient, and great care must be taken (1) to inject very slowly—ten minutes may be taken to empty the 8-ounce syringe; (2) to stop immediately there are any signs of distress, such as dyspnœa or discomfort about the heart: (the pulse should be continually watched).

When the whole of the 8 ounces, or as much as the patient can comfortably take, has been injected, the nozzle is withdrawn, the second half of the knot tied, a couple of stitches put in, and the wound dressed in the usual way.

In cases of pernicious anæmia this operation may have to be repeated several times at intervals varying from two to six weeks, according to the patient's progress. It has been frequently noted that improvement, as tested by frequent enumeration of the corpuscles, continues progressively for some weeks and then halts. It is advisable to operate again when this takes place, the patient in favourable cases being left on a better level as regards his blood after each transfusion, until, after several injections, a normal condition of matters may be reached.

Transfusion of blood into the arteries has been used by Hueter, the object being to avoid the introduction of clots into the heart. As this danger can be avoided by the use of the phosphate of soda method, and as gangrene of the part supplied by the artery employed for the purpose may occur from embolism, the method need not be further discussed.

(4) *Injection of Saline Fluids.*—This procedure has been used with great success after severe hæmorrhage, and in cases of profound shock. It is recommended to be used before or after severe abdominal operations, and has also occasionally been employed in septicæmia, diabetic coma, and post-operative suppression of urine. It is perfectly useless in pernicious anæmia, except as a temporary stimulant.

There are several methods of introducing saline fluid into the circulation—

(*a*) By injection into the rectum.

(*b*) By injection into the loose subcutaneous tissue, *e.g.* of the axillary region.

(*c*) By injection into the veins.

(*d*) By injection into the arteries.

The last of these methods seems to possess no advantages, and some obvious disadvantages, while in regard to the first and second, one must remember that absorption through the vessels of the rectum or subcutaneous tissue can only take place when the circulation is being fairly carried on in the part, whereas intravenous infusion of salines has sometimes a marvellous effect in patients who are almost pulseless.

The saline fluid most commonly used for infusion is a solution of 1 drachm of common salt in a pint of sterile water at a temperature of about 105° F. Some prefer a solution containing 50 grains of sodium chloride, 3 grains of potassium chloride, 25 grains of carbonate, and 25 grains of sulphate of sodium, with 2 or 3 grains of phosphate of soda, in a pint of water. It is very doubtful if this has any advantage over the simpler salt solution. The fluid is introduced with a glass syringe or funnel arrangement through a glass and rubber nozzle into the patient's vein (special instruments for the purpose have been devised, for example, by Spencer and Collin). The quantity introduced is from ½ a pint to 3 or 4 pints, depending on the condition of the patient and the amount of blood lost. The infusion must be discontinued should the patient show any distress, and, in any case, as soon as the normal pulse tension is regained. The operation must be done very slowly and cautiously, and with aseptic precautions. In cases of hæmorrhage it should only be used when the bleeding has been stopped. The procedure may require to be repeated. It is apt to be followed by a considerable, but transient, rise of temperature. The infusion of milk or of the blood of the lower animals has been tried. These methods are dangerous, and have been discontinued.

Transillumination.—The passage of light through an organ or through the walls of a cavity, *e.g.* of the maxillary sinus as a means of diagnosis. *See* Larynx, Examination of (*Transillumination*); Nose, Accessory Sinuses, Inflammation of (*Acute Inflammation of Maxillary Sinus, Transillumination*); Nose, Acessory Sinuses, Inflammation of (*Chronic Suppuration in Frontal Sinus, Transillumination*).

Transitional Epithelium. *See* Physiology, Tissues (*Epithelium, Squamous*).

Transition Organism.—The intermediate stage in development when the new organism is neither an embryo nor a fœtus (six weeks to two months), the neofœtus. *See* Embryology (*Neofœtal Period*).

Transmigration.—The act of passing from one place to another; *e.g.* the wandering of white blood corpuscles from the blood stream into the tissues, or the passage of an ovum from one ovary into the opposite tube *viâ* the uterine cavity (*internal* transmigration) or the peritoneal sac (*external* transmigration.)

Transmission. — The communication of a thing (*e.g.* a disease) from one person or place to another; also, the handing on by heredity of characters from parent to child. *See* Epidemiology; Heredity (*Transmissibility of Acquired Characters*).

Transplantation.—The carrying of a part from one region or area of the body to another; grafting (*e.g.* of a piece of the cornea or skin).

Transport. *See* First Aid (*Means of Transport*).

Transposition.—A change in position, especially that in which the organs normally present on one side of the body are found on the opposite, or in which the viscera of the abdomen are displaced upwards into the thorax; heterotaxy or situs inversus viscerum. *See* Heart, Congenital Malformations of the (*Transposition of Aorta and Pulmonary Artery*); Teratology (*Heterotaxy*).

Transudation. — The passage of a liquid through a membrane, *e.g.* a vessel-wall, the liquid which has so passed being named a *transudate*. *See* Dropsy; Inflammation.

Transvaal. *See* Therapeutics, Health Resorts (*South Africa*).

Transversalis.—The name given to the muscle and artery and fascia of the anterior abdominal wall, the neck, or other part, which has a transverse direction.

Transverse.—Removing crosswise, *e.g.* a *transverse presentation*, a shoulder-case or cross-birth. *See* Labour, Diagnosis and Mechanism (*Transverse Lies*).

Trap. *See* Sewage and Drainage (*Traps*).

Trapezium.—One of the bones of the carpus; also, a cross band of fibres near the lower border of the pons Varolii.

Trapezius.—A superficial muscle of the back of the neck and thorax. *See* Spinal Accessory Nerve (*Functions and Effects of Lesions of*).

Trapezoid.—A carpal bone, second bone of the second row articulating with the metacarpal bone of the index finger.

Trapp's Coefficient or Formula.—A means of estimating the amount of solid matter in the urine, consisting in multiplying the last two figures of the specific gravity by 2 or 2·33 (Hoeser's coefficient) and so getting the number of grammes of solids per 1000 c.c. of urine.

Traube-Hering's Curves.—Rhythmical undulations seen in a sphygmographic tracing soon after respiration has stopped (*e.g.* from curara and section of the vagi); they represent rhythmical variations in blood-pressure.

Traube-Rosenstein Theory.—The theory that eclampsia is due to toxic cerebral œdema causing anæmia of the brain. *See* ECLAMPSIA.

Traube's Corpuscles. — Phantom corpuscles or erythrocytes which have the form of pale yellowish rings.

Traube's Plugs. *See* DITTRICH'S PLUGS.

Traube's Space.—A semilunar space on the left side of the thorax where a tympanitic note is got on percussion due to air in the stomach; the effusion in pleurisy may lead to its obliteration.

Traube's Theory. *See* RESPIRATION (*Cheyne-Stokes, Explanation of*).

Trauma.—A wound or injury of any kind, from which come such terms as *traumatic* (relating to or caused by a wound), *traumatism* (the state produced by trauma, or trauma itself), and *traumatology* (the science of wounds). *See* CATARACT (*Traumatic*); JOINTS, DISEASES OF (*Arthritis Deformans or Traumatic Arthritis*); NERVES, PERIPHERAL (*Injuries, Traumatic Neuritis*); SPINE, SURGICAL AFFECTIONS OF (*Railway Spine or Traumatic Neurosis*); SYRINGOMYELIA (*Etiology, Trauma*); WOUNDS (*Drainage, Traumatic Fever*), etc.

Traumaticin.—A saturated solution of gutta-percha in chloroform, used for closing superficial wounds.

Traumatides.—The consequences of the pruritic state. *See* PRURITUS (*Consequences*).

Traumatol.—An antiseptic and dusting powder obtained by the action of iodine on cresol, and being, chemically, iodocresol (C_7H_7IO); it is insoluble in water and alcohol but soluble in chloroform.

Traumatopnœa.—Breathing through the wound as in cases of penetrating wounds of the pleura. *See* PLEURA, SURGICAL AFFECTIONS OF THE (*Traumatopnœa*).

Tread.—The germinating point or cicatricula of an impregnated egg.

Treatment.—The management of cases of disease or injury with the purpose of procuring alleviation of suffering or complete removal of the morbid state; it may be medicinal means, by operation, by diet, by exercise, by electricity, etc. *See under* the various *diseases* and under such general headings as *hydropathy, therapeutics, prescribing*, etc.

Trefriw. *See* BALNEOLOGY (*Great Britain, Wales, Chalybeate Waters*); MINERAL WATERS (*Chalybeate*).

Trematodes. *See* PARASITES (*Helminths, Trematodes or Flukes*); LIVER, LIVER PARASITES (*Trematodes, Distomum*).

Trembles.—A disease of cattle, occurring in certain parts of the United States of America, believed to be the cause of *milk-sickness* in the human subject (*Osler*).

Trembling Palsy.—Paralysis Agitans. *See* PARALYSIS (*Paralysis Agitans*); TREMOR (*Pathological Causes*).

Tremens.—*See* DELIRIUM TREMENS.

Tremor. *See also* ALCOHOLISM (*Variations*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Resulting Conditions, Intentional Tremor*); BRAIN, TUMOURS OF (*Symptoms, Tremor*); CHILDREN, CLINICAL EXAMINATION OF (*Nervous System, Tremor*); GENERAL PARALYSIS (*Symptoms, Tremor of Tongue*); HYSTERIA (*Motor Disorders, Hysterical Tremblings*); MALINGERING (*General Considerations*); PARALYSIS (*Diagnosis, Paralysis Agitans*); THYROID GLAND, MEDICAL (*Exophthalmic Goitre, Symptoms, Tremors*); TRADES, DANGEROUS (*Mercurial Poisoning, Symptoms*); URÆMIA (*Symptoms*).

THE name tremor has been somewhat loosely given to various forms of rhythmical muscular contraction, which may be widely dissociated not only in their clinical character, but, as far as we can judge, in their physiological or pathological origin. It is customary, for instance, to talk of the tremor of fatigue, the tremor of Graves' disease, or the tremor of disseminate sclerosis. Tremor is not, therefore, a disease, nor, in all cases, a sign of disease, and owing to its widely different character under different circumstances it is not capable of a good pathological or clinical definition. Speaking generally, the term may be said to embrace all the oscillations of a part or of the whole of the body which are rapidly repeated more or less continuously, and more or less regularly without being under the complete control of the will. Such oscillations may recur from two to ten or more times a second. They are all liable to be modified by the will and by the emotions, and in most cases cease entirely under the influence of sleep or of general anæsthetics.

Physiology.—In order to understand the prevailing view of tremor it is necessary to turn for a moment to the character of voluntary muscular contractions and of muscle and nerve tonus. It is impossible to produce artificially a muscular contraction similar to that presented by a willed movement without the application of several rapidly-repeated stimuli, and there are many other reasons for supposing that even a single voluntary movement of a single muscle, such as the blink of an eyelid, is the result of several intermittent discharges of nervous energy. Moreover, physiological experiments tend to show that fatigue produces a diminution in the rapidity with which these discharges follow one another. If these assumptions are correct, voluntary muscular contractions are *naturally* rhythmical in character, and the close association of muscular tonus with muscular activity would lead us to believe that tonus itself is the result of intermittent impulses flowing in a continuous stream, under normal conditions, from living nerve cells, and capable of modification by various influences acting upon these cells. These physiological considerations are not calculated in the present state of our knowledge to explain the tremor of disease, but they enable us to put it in line with the natural activity of nerve and muscle, and to show that there is probably a very close relationship between tremor, hypertonicity, and spasm, a relationship which may perhaps depend on variations in the amplitude and the rapidity of the so-called nervous discharges

Clinical.—It has been customary to divide all tremors into two classes and to classify them under the terms "passive tremor" and "intention tremor," but this classification is unscientific and leads to many fallacies in practice. We find, for instance, that the tremor of disseminate sclerosis, although generally brought into notice by the attempt to perform some voluntary action, is often present while the patient is at rest, and that many passive tremors, such as that of paralysis agitans, may be remarkably exaggerated by voluntary exertion. If it were possible we should prefer to classify tremors on a scientific pathological basis, but our knowledge of their pathology is too scanty to permit of this course, and we shall therefore attempt a scheme of classification dependent on the conditions under which tremor is liable to occur. This method has the advantage of bringing together types of tremor which are similar not only in their clinical characters, but very probably also in their origin.

Psychological		1. Emotion.
		2. Hysteria.
Physiological		3. Heredity.
		4. Senility.
		5. Acute fatigue.
		6. Chronic fatigue.
		7. Cold.
Pathological	(A)	8. Toxæmia.
		9. Metallic poisoning.
		10. Chronic narcotism.
	(B)	11. Neurasthenia.
		12. Rheumatoid arthritis.
		13. Paralysis agitans.
		14. Exophthalmic goitre.
	(C)	15. Disseminate sclerosis.
		16. Lesions about the mesencephalon.
		17. Spastic conditions generally.

Psychological.—The tremors of emotion are too well known and too variable to admit of detailed description, but it may be noticed that they are more often excited by the unpleasant conditions of terror and anger than by more pleasurable states of mind. The tremors of hysteria are generally irregular, inconstant, often confined to one limb or part, and always worse under observation. They may be fine or coarse, but are more often coarse and moderately slow than fine and rapid.

Physiological.—The hereditary and senile forms of tremor, like those of grey hair and arterio-sclerosis, are closely associated one with the other, and appear to be the visible signs of some premature tissue decay to which the family or the individual is especially prone. The hereditary form generally appears after twenty years of age, and resembles the senile form in being rather fine in character and in affecting most frequently the muscles of the upper extremity and of the face and neck.

All forms of severe exertion may cause a temporary state of tremulousness, which must be familiar to every one, and which is especially noticeable in the attempt to carry out a fine movement, such as that of writing. On the other hand, the constant daily use of certain muscles, perhaps in constrained positions, is a prolific source of tremor in connection with the different forms of occupation neurosis. Miners' nystagmus and certain cases of writers' cramp are good examples of this, and the latter condition serves to show how closely spasm and tremor are associated not only in organic but in functional disorders of the neuro-muscular system. Exposure to a rapid fall of temperature is liable to set up momentarily a form of tremor especially affecting the muscles of the head and trunk, of which the most common type is known by the name of "teeth-chattering."

Pathological.—(A) Many specific fevers both in the acute stage and in the debility of convalescence may give rise to tremors of the extremities and of the tongue and lips. It is impossible to say how far the rise of temperature or how far the circulating toxins are responsible for this result, but it is probable that both have a deleterious influence on certain nervous structures, of which the tremor is the outward expression.

Tremor is often an essential element of a rigor, and is not unfrequently associated with pain and an elevation of temperature in cases of renal and other forms of colic.

A morbid entity consisting of severe paroxysmal attacks of tremor accompanied by loss of consciousness and some fever has been described, but little is known of this rare condition except that the prognosis appears to be favourable.

Syphilis in its more remote influence on the nervous system is sometimes productive of tremor, and the early diagnosis of general paralysis of the insane is often rendered easy by the very characteristic tremor of the lips and tongue, which may be very noticeable, and which doubtless largely contributes to the peculiar articulation observed in these patients.

Lead, mercury, arsenic, copper, and zinc are responsible for most cases of tremor due to metallic poisoning, and the symptom is most commonly present in the upper extremities and sometimes in the muscles of the face. The oscillations are usually fine and rapid, but in some mercurial cases coarse movements affecting the whole body are met with.

As is well known, most narcotics are primarily stimulants, but the tremor which is produced by their habitual abuse must be accounted a symptom of the secondary narcotism, and it is interesting to note that the tremor of a chronic alcoholic subject or of a morphomaniac may be temporarily controlled by a dose of alcohol or of morphia, the tremor reappearing as the stimulative effect of the dose wears off. Alcohol, morphia, and tobacco are the best-known members of this group of poisons, but a fine tremor of the extremities is also common in patients suffering from bromidism.

(B) The four diseases, neurasthenia, rheumatoid arthritis, paralysis agitans, and Graves' disease, are purposely placed together because they appear in more ways than one to be related, and to belong to a group of which the pathology is still obscure. The tremor of some neurasthenics is extremely suggestive of Graves' disease, and some cases of rheumatoid arthritis resemble paralysis agitans in having either a hemiplegic or diplegic distribution of tremor, which, combined with the rigidity caused by joint troubles, gives the patients a strong resemblance to cases of Parkinson's disease. Moreover, all four diseases present trophic disorders of skin, nails, etc., which can hardly fail to attract attention as being of practically the same kind. The tremulousness is, in the first instance, always limited to the extremities in these diseases. The tremor of paralysis agitans deserves a little more attention in that it serves as a good general example of the phenomenon. The movements are repeated from three to seven times a second, and their size and rapidity vary with the size of the muscle affected. In some cases the most prominent movement is that of the thumb against the first finger (cigarette-rolling), and the oscillations may be fine and limited in extent. Another common rhythmic action is that of alternate pronation and supination of the wrist, and then both the intermittence and size of the movement are greater. Only in more advanced stages of the disease does the tremor attack the proximal parts of the limbs and the muscles of the head and trunk, which are generally the seat of great rigidity.

(C) The diseases of the last group are alike in presenting well-known anatomical lesions of the central nervous system and in being characterised by spasticity and tremor of varying degree and severity. The tremor in each case is called into existence or exaggerated by efforts of voluntary activity, and is of a coarse and not very co-ordinate type. A spontaneous ankle clonus occurring in an extremely spastic patient is a good specimen of this kind of tremor, which can hardly be placed in the same category as, and deserves a different name from, the finer movements seen in exophthalmic goitre or chronic alcoholism. The rate of the oscillations in disseminate sclerosis varies from four to eight per second, but the excursions of the affected limb are not of constant size.

Certain cases of hemiplegia and of diplegia present a form of intention tremor which cannot be distinguished from that of disseminate sclerosis, but we are still ignorant of the anatomical basis for this phenomenon; it can only be said that disease in the mesencephalic region of the brain appears to be most frequently associated with it.

Diagnosis.—A knowledge of the conditions under which tremor occurs is of great practical value, but the diagnosis of a disease can rarely be made from the character of the tremor alone in any particular case. It is true that the tremor of paralysis agitans is very characteristic in many patients, but it is usually associated with such an unmistakable picture in other respects that even its absence would not discount the diagnosis.

All other forms of fine tremor are not easily differentiated on their own merits, and the coarse intention movements of disseminate sclerosis may not infrequently be seen in cases of hemiplegia, diplegia, or cerebral tumour. The accompanying signs and symptoms must, therefore, be always carefully considered before arriving at any definite conclusion.

Pathology.—Enough has already been said to show that our knowledge of the morbid process concerned in the production of tremor is too insufficient to allow of any dogmatic statements to be made on this point, but it must be granted that in many cases the phenomenon would seem to result from the action of poisons on some part of the motor neurons, and probably on the cellular part of those structures. It may be

inferred, too, from a consideration of the diseases in which some forms of tremor occur, that the same nervous elements are influenced by the altered metabolism associated with degenerative processes as in fatigue and senility, or with modified conditions of internal secretion, as for instance in Graves' disease, and possibly, according to some authors, in paralysis agitans. It is not safe, however, to do more than suggest the possibility of this connection.

Treatment.—In most cases the treatment of tremor is the treatment of the disease of which it is one of the physical signs; in others, such as paralysis agitans, where it is often a source of great distress to the patient, attempts directed to its alleviation are seldom very successful. Injections of hydrobromate of hyoscine are sometimes helpful in reducing the severity of the condition, but most other sedatives are quite ineffectual. Galvanism may at times be used with advantage, and the administration of thyroid gland in small doses, gradually increased, is worthy of trial.

Fibrillary Tremor.—This condition should not, except for its name, be mentioned in this connection. The name has been applied to waves of contraction which can be seen passing over the individual fibres of a muscle in an early state of degeneration in cases of neuritis or of progressive muscular atrophy, and which do not result in movements of the parts affected. They are usually made more noticeable by the exposure of the overlying skin to cold air, as in stripping the patient, but are unimportant except for purposes of diagnosis.

Trendelenburg Posture. — The dorsal decubitus with the trunk raised at an angle of about 45° and the legs and feet hanging over the lower end of the table; the weight of the body thus rests on the shoulders and the back of the knee joints; it is carried out by raising the lower end of the bed or operating-table by means of two stools (under the feet of the table) or by a special mechanism; and it is useful in all operations where it is desirable to bring the pelvic organs into view; *e.g.* hysterectomies, myomectomies, oophorectomies, etc. *See* Ovaries, Diseases of (*Ovariotomy*).

Trepan.—A trephine (Gr. τρυπάω, I bore or pierce through) or instrument for cutting a circular piece of bone out of the cranial vault.

Trephine.—A trepan (q.v. *supra*), the operation in which the circular piece of bone is cut out of the cranial vault being termed *trephining*. *See* Brain, Surgery of (*Trephining*); Labour, Obstetric Operations (*Embryotomy*); Spine, Surgical Affections of (*Fracture of the Spine, Treatment by Laminectomy*).

Triacid.—An acid or alcohol containing three atoms of hydrogen which can be replaced by a base.

Triacid Stain.—In histology this name is given to Ehrlich's triple stain which contains one basic and two acid dyes, viz. methyl-green, orange-green, and acid fuchsin; the tissues which have an affinity for acid dyes are stained by this mixture the colour of one of the acid constituents, the basic structures take the colour of the basic dye, and those having an equal affinity for both have the colour of the neutral principle. (*Da Costa.*)

Triakaidekaphobia.—An insane fear of the number thirteen (Gr. τριακαίδεκα, thirteen).

Triangle.—The name given in descriptive anatomy to various regions of the body which have this three-sided shape; e.g. *Scarpa's triangle* of the thigh, the *triangles of the neck*, *Hesselbach's triangle* (above Poupart's ligament), the *triangle of Petit*, etc. *See* Hernia (*Inguinal, Direct*).

Triangular. — Having a three-sided shape, *e.g.* the triangular ligament of the pelvic floor (*see* Generation, Female Organs of, *Pelvic Floor*), a triangular bandage (*see* First Aid, *Improvised Dressings*), and the triangularis sterni muscle.

Tri-.—In compound words tri- as a prefix means three or relating to three; e.g. *tribasic* (a compound having three atoms of hydrogen replaceable by basic atoms), *tricornis* (having three horns), etc.

Tricephalus.—The extraordinarily rare teratological type in which a double fœtus carries three heads; a triple monster. A case of this kind was reported by Reina and Galvagni in 1834; there were three heads set upon two united trunks, supported by two lower limbs, there were three upper limbs and a single set of male genitals; all the heads had to be perforated during labour, and two of them amputated before the uterus could be emptied.

Triceps.—Having three heads; the name given to the muscle of the upper arm which extends the forearm. *See* Muscles, Traumatic Affections of (*Rupture of Triceps*); Tabes Dorsalis (*Symptoms, Abolition of Triceps-Jerk*); Tendon-Jerks (*Triceps-Jerk*).

Trichauxis.—Hypertrichiasis or excessive hairiness. *See* Hirsuties.

Trichiasis.—A morbid state of the eyelids in which the eyelashes, being abnormally placed, irritate the ocular conjunctiva and cornea. *See* Conjunctiva, Diseases of (*Trachoma*); Eyelids, Affections of (*Defects in Position, Trichiasis*).

Trichina.—A genus of the Nematode worms, of which *Trichina spiralis* is one of the species. *See* PARASITES (*Nematodes, Trichotrachelidæ*): *see also* FILARIASIS (*Filaria Bancrofti or Trichina Cystica*); FOOD (*Characteristics of Unsoundness, Trichina Spiralis*); MUSCLES, DISEASES OF THE (*Trichiniasis*); TOXICOLOGY (*Animal Foods, Trichiniasis*).

Trichiniasis or Trichinosis.—The morbid condition due to the presence of the *Trichina spiralis*. *See* PARASITES (*Nematodes*); TRICHINA and *Cross References*.

Tricho-.—In compound words *tricho-* (Gr. θρίξ, a hair) means relating to the hair or resembling a hair; e.g. *trichomatosis* (a matted condition of the hair, plica), *trichomonas* (a flagellate infusorium which lives in the vaginal mucus (*T. vaginalis*) or in the intestines (*cercomonas hominis*).

Trichocephalus.—A parasitic Nematode worm belonging to the Trichotrachelidæ; the morbid state due to its presence is named *trichocephaliasis*. *See* PARASITES (*Nematodes*).

Trichoglossia.—Hairy tongue, a morbid state in which the enlarged papillæ give the tongue a hairy appearance.

Trichophyton.—A parasitic organism or fungus found on the hair, *e.g.* the *Trichophyton tonsurans* of ringworm. *See* ALOPECIA (*Varieties*); SKIN, PARASITES (*Tinea Tonsurans*); SUPPURATION (*Etiology, Micro-organisms in Pus*).

Trichophytosis.—The morbid state due to the presence of the trichophyton (*q.v.*).

Trichorrhexis Nodosa.—A brittle condition of the hair which shows irregular thickenings or "nodes" on the shafts. *See* SKIN, BACTERIOLOGY OF (*Trichorrhexis Nodosa*).

Trichotrachelidæ. *See* PARASITES (*Nematodes*).

Trichromic Vision. *See* COLOUR VISION (*Theories of Colour Perception*).

Trichterbrust.—The funnel chest or thorax due to depression of the lower part of the sternum, seen in tuberculosis, adenoids, etc., or as a congenital malformation; sternal cyphosis.

Tricornis.—Having three horns or processes, *e.g.* the lateral ventricles of the brain.

Tricresol.—A mixture of the three cresols (ortho-, meta-, and paracresol), specially purified; is an efficient antiseptic.

Tricrotic.—Possessing three wavelets or crests in each beat of the pulse as seen in a sphygmographic tracing. *See* PHYSIOLOGY, CIRCULATION (*Form of the Wave*). The pulse which has this character is said to show *tricrotism*.

Tricuspid.—Having three cusps or segments, *e.g.* the right auriculo-ventricular valves of the heart. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Valvular Affections, Tricuspid Valves*).

Triencephalus.—Saint-Hilaire's term for the monstrosity more commonly known as aprosopus, in which the *three* parts, mouth, nose, and eyes, are all absent.

Triethylamine.—A ptomaine ($C_6H_{15}N$) obtained from putrid haddock.

Trifacial or Trigeminal.—The name given to the fifth cranial nerve on account of its three branches (ophthalmic, superior maxillary, and inferior maxillary). *See* FIFTH NERVE, APPLICATIONS OF; NERVES, NEURALGIA (*Symptoms*). The term *trigeminal* is also applied to the pulse in which the full systole is followed by two premature systoles. *See* PULSE (*Bigeminal Pulse and its Modifications*).

Trigger Finger.—A morbid condition in which extension or flexion of a finger is at first impeded and then takes place with a jerk.

Trigone.—A triangular space, *e.g.* the *trigone of the bladder*, which lies behind the internal orifice of the urethra and between the orifices of the ureters and has a smooth surface.

Trigonocephalus.—A teratological type in which the head of the fœtus has a triangular shape. (Gr. τρι-, thrice; and γανία, a corner or angle).

Trillat's Autoclave.—An apparatus for carrying out gaseous disinfection; formic aldehyde may thus be employed for fumigation, formalin being heated with calcium chloride.

Trilocular.—Having three chambers or loculi, *e.g.* the trilocular heart which results when the interauricular septum is incompletely closed.

Trimethylamine.—A ptomaine obtained from herring-brine and other decomposing substances. *See* SNAKE-BITES AND POISONOUS FISHES (*Decomposing Fish*).

Trinitrin.—Glonoin oil, Nobel's blasting oil, or nitroglycerine. *See* NITRITES (*Nitroglycerine*).

Trocephalus.—A teratological type, being a variety of otocephaly in which the cranium is reduced to the occipital bone, the petrous temporals, and a rudimentary parietal; there is no mouth, no eye, and no nasal proboscis, the external ears are closely approximated or united, and the brain is reduced to the medulla with or without the pons.

Trional and Tetronal.—Diethyl-sulfone-methyl-ethyl-methane. It is a hypnotic and is used in the same class of cases as sulphonal. Its action is somewhat quicker than that of sulphonal. *Dose*—10-30 grs. TETRONAL is closely allied to trional and is similar in action. *Dose*—10-20 grs. Both are best given in cachet and followed by a drink of hot fluid. Like sulphonal they may produce hæmatoporphyrinuria.

Triorchidy.—The anomalous state in which three instead of two testicles are present; there is usually some doubt whether the supernumerary gland is really testicular in nature.

Trioxypurin.—Uric acid. *See* URIC ACID (*Chemical Characters*).

Tripara.—A woman who has passed through or is passing through her third confinement; one who has been pregnant thrice.

Tripe. *See* INVALID FEEDING (*Meats, Tripe, Methods of Preparing*).

Triphalangy.—The structural anomaly in which a digit which has normally two phalanges has three (*e.g.* a thumb or great toe with three phalanges); hyperphalangy is not exactly synonymous, for it includes the cases in which a digit has more than three phalanges (*e.g.* the index finger with four phalanges).

Triphenamine.—A mixture of salicylate and acetate of phenocoll with phenocoll itself; it is used in rheumatism.

Triphenin.—A homologue of phenacetin, used in the same maladies and in similar doses.

Tripier's Amputation.—A method of amputating the foot, resembling Chopart's but differing in that part of the os calcis (the part below the sustentaculum tali) is removed whereas by Chopart's method the whole os calcis is left.

Triple.—Threefold, as triple phosphates (phosphates of ammonium and magnesium), triple monsters (the very rare teratological type in which three fœtuses are fused together), etc.

Triplets.—Three children born at one time by the same mother; the occurrence is rare, about once in seven thousand pregnancies; they may be uniovular, binovular, or triovular; one of the fœtuses is not uncommonly an acardiac. *See* PREGNANCY, MULTIPLE.

Triplopia.—A form of disturbed vision in which three images of a single body are seen.

Tripod Fœtus.—A double monster in which one fœtus is represented only by a limb attached to the pelvis of the autosite; the result is an *ischiomelus* or a pygomelus; Dos Santos was the best-known instance of this teratological type, and was called the "human tripod." *See* TERATOLOGY (*Parasitic Twins*).

Triradiate.—Radiating in three directions, *e.g.* the pelvis in marked cases of osteomalacia or malacosteon.

Trismus.—Lockjaw, especially the form which occurs in the new-born infant (trismus neonatorum) from infection of the umbilicus. *See* NEW-BORN INFANT (*Tetanus Neonatorum*); SPASM (*From Irritation of Sensory Nerves, Hysterical*); TEETH (*Alveolar Abscess, Sequelæ*).

Tristitia.—Melancholy as distinguished from tristemania or melancholia.

Triticum.—*Triticum repens* or dog-grass is used as a diuretic and bladder-sedative in cystitis in the form of the *extractum tritici liquidum* (dose, 1 to 6 fl. dr.); it is official in the U.S. Pharmacopœia but not in the British. *See also* AGROPYRUM.

Trituration.—The process of reduction to powder by rubbing; a substance so reduced.

Trocar or Trochar. *See* ASPIRATOR, USES OF.

Trochanter.—A process or projection (Gr. τροχός, a wheel or anything that runs round), especially the two projections (trochanter major and trochanter minor) on the upper end of the shaft of the femur below the neck. *See* HIP-JOINT, DISEASES OF (*Diagnosis of Hip-Disease*).

Trochiscus or Troche.—A lozenge, especially one containing some drug, such as tannic acid (Trochiscus Acidi Tannici) or catechu (T. catechu) or morphine (T. Morphinæ). *See* PRESCRIBING (*By Mucous Membranes, Trochisci and Pastilles*).

Trochlea.—A grooved articular surface (*e.g.* the *trochlea* of the lower end of the femur or of the upper surface of the astragalus) or a fibrous pulley (*e.g.* the *trochlea* of the upper margin of the orbit) (Gr. τροχῖλία, a pulley).

Trochlear or Trochlearis.—Of the nature of a pulley or belonging to the trochlea of the orbit or astragalus; *e.g.* the trochlear muscle, or nerve, or the musculus trochlearis. Trochlear paralysis is paralysis of the fourth cranial nerve and of the superior oblique muscle of the eyeball.

Trochocephaly.—An anomalous shape of the head, a round-head (Gr. τροχός, a round ball or cake).

Trommer's Test. *See* URINE, PATHOLOGICAL CHANGES IN (*Glucose, Tests for*).

Tromomania.—Delirium tremens (Gr. τρομέω, I tremble).

Tropacocaine or Tropococaine.—A local anæsthetic, used in the form of the hydrochloride ($C_8H_{14}NO.C_6H_5CO.HCl$) for intraspinal injection (lumbar anæsthesia), and as a solution in dental surgery; it is said to be less dangerous than cocaine.

Trophic.—Belonging to or influencing nourishment (Gr. τροπφή, food). *See* AINHUM (*Etiology*, *Trophic Lesion*); ATROPHY (*Physiology of Trophic Influence*); BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF (*Symptoms, Trophic*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Conditions Resulting from*); HEMIPLEGIA (*Trophic Changes*); HYSTERIA (*Trophic Disorders*); NERVES, PERIPHERAL (*Injuries, Symptoms, Trophic Changes*); RAYNAUD'S DISEASE; SYRINGOMYELIA (*Symptoms, Trophic Changes*).

Trophoblast.—The epithelium covering the segmenting ovum and developed probably from its epiblast, which serves as the means by which implantation of the new organism on the mucous membrane of the uterus takes place; it is believed that the trophoblast invades the uterine mucosa and that the maternal blood converts the trophoblast cells into syncytium. *See* EMBRYOLOGY, HUMAN (*Second Week*).

Trophoneurosis.—Atrophy of a part due to defective action of the nerves which control its nutrition. *See* RAYNAUD'S DISEASE; ATROPHY; etc.

Tropical.—Belonging to the tropical regions of the earth; *e.g.* tropical liver, phagedæna, and ringworm. *See* TROPICS, UNCLASSED FEVERS OF; LIVER, TROPICAL AFFECTIONS OF; SUNSTROKE; etc.

Tropics, The Unclassed Fevers of the.

INTRODUCTION 110
CLASSIFICATION 110
CONTINUED FEVERS OF SHORT DURATION
Nakra 112
"Low" Fever 113
CONTINUED FEVERS OF LONGER DURATION—
Bastard Typhoid 113
Double Continued Fever . . . 113
Non-Malarial "Remittent" Fever . 114
RECURRENT FEVERS OF LONG INTERVAL . 115
FEVERS OF MIXED ORIGIN—
Typho-Malarial 115
Kala-Azar 115
Hæmoglobinuric Fever . . . 116

See also LIVER, TROPICAL AFFECTIONS OF.

THE enormous extent of the field which hot climates offer for the clinical and pathological study of febrile diseases may be estimated by a glance at the annual returns of the Government of India. In 1899, which was an unusually healthy year, no fewer than 4,072,750 deaths, exclusive of those caused by the exanthemata, were registered under that heading. In other years the number has reached four and a half millions (1892) in a population of 250,000,000; and remembering the very imperfect registration of mortality in that country, where it is probably no exaggeration to say that a fourth of the deaths are not registered at all, and that in the native army and jail population, from which we obtain exact returns, the case mortality from these diseases (including hepatitis, pneumonia, and other inflammatory diseases, which are all massed together with "fevers" in those of the general population) is only 10 per 1000, one can form some conception of the amount of sickness which these figures represent and of the field which they offer for study. Out of a total registered mortality of 29·5 per 1000, 19·5 are entered under this head, and it may be said that in India—and there is no reason to think that India is exceptional to other hot countries in that respect—of every three cases which present themselves for treatment two are suffering from fever of some kind.

Fevers have, accordingly, always loomed large in writings descriptive of tropical diseases, and the history of tropical medicine is naturally largely that of the disentanglement of this mass of fevers and the gradual differentiation of clinical types.

Putting on one side malarial fevers of the intermittent and "remittent" forms, typhoid, Malta, relapsing, filarial fevers, and the exanthemata, there remain a large number of cases which refuse to range themselves under any of these headings. These may be subdivided into the following clinical types:—1. Continued fevers of short duration; 2. Continued fevers of longer duration and more or less definite course; 3. Periodic fevers of long interval; 4. Fevers of uncertain and probably mixed origin.

CONTINUED FEVERS OF SHORT DURATION

Writing in 1857, J. B. Scriven (*Indian Annals of Medical Science*, vol. iv., 1857) was able to recognise four essential kinds of fever in Bengal, exclusive of the exanthemata, namely,—1. Malarious fever, divided into continued, remittent, and intermittent; 2. Febricula or ephemeral fever; 3. Insolation or sun fever; 4. Typhoid fever. This classification took firm hold of the profession, and received the sanction of such great teachers as Maclean and Chevers. It was simple and comprehensive, but the lumping together of continued, remittent, and intermittent fevers under the heading of "Malaria" has greatly retarded the progress of tropical pathology. The difficulty of grouping together fevers offering such diverse clinical pictures was not unperceived, but the early defervescence of those of a short continued type

was believed to be due to treatment. Writing in 1886, Chevers (*Diseases of India*, p. 216) says:—"Assuredly he who in Bengal proper treats all cases of ordinary fever (excluding jungle fever, enteric, and the exanthemata) with quinine, as soon as perspiration shows itself after the first paroxysm, will find that febricula is a very ephemeral disease indeed, and that his experience of common continued fever is extremely small. I am confident that these three diseases, or, to speak more accurately with Dr. Morehead, these three degrees of the same malady, are in Lower Bengal the most usual types in which intermittent fever first attacks the robust, full-blooded, and perhaps free-living European who arrives there during the hot weather." The fallacy incorporated in these remarks is that ephemeral fever, febricula, and simple continued fevers, are fevers of a single paroxysm of a few hours' or days' duration, and that there is no tendency to recurrence of the paroxysm whether they are treated with quinine or not. If they show any tendency to a repetition of the paroxysm this must be taken as evidence that they are of a different type, and due to malarial infection, when quinine in full doses becomes the appropriate treatment; but I have treated many hundreds of such cases without the exhibition of a single grain of quinine, and without their showing any tendency to the manifestation of the phenomena of the life-cycle of the malarial parasite.

This is the commonest type of fever, not only in Lower Bengal, but also in Burma, and it is probably so equally in other parts of India. That these fevers do not take the first place in the returns of the Government of India is due to two circumstances: first, because cases of fever of such short duration are not necessarily admitted to hospital; and, secondly, because there is a strong tendency, especially in the native army and jails—such is the result of the teaching of which I complain—to return all cases of mild fever of short duration as "ague." These returns indeed do not show the existence of ephemeral fever or febricula at all, the commonest forms of fever with which the practitioner in civil life has to deal. Thus in the Alipore jail in 1899 the admission-rate for "simple continued fever" (probably including all the milder expressions of single paroxysm fevers) was 373·7 per 1000 of population, against 74 per 1000 for "intermittent" fever; whereas in the Presidency jail, a mile distant, the returns would indicate 2·5 and 299·3 per 1000 respectively. The difference between the returns of these neighbouring institutions is indicative of the personal element in classification, and it must be admitted that such returns are valueless as an index to the relative prevalence of these mild fevers of a single paroxysm, and true malarial fevers. This can only be arrived at by a systematic examination of the blood.

These three fevers, or rather these three degrees of the same fever, differ not only in duration, but in intensity. When the duration is only a few hours, and the intensity is slight, it is called "ephemeral" fever; when it lasts some forty-eight hours it is spoken of as "febricula"; and when its continuance is longer, from three to eight or ten days, when the temperatures usually vary from 102° F. in the morning to 103° to 104° in the evening, it rises to the dignity of "simple continued fever." Quinine in full doses has no appreciable effect either on their duration or course. They subside spontaneously by lysis, and in the warm, humid climate of Lower Bengal are often accompanied by much visible perspiration throughout their course, which is absent in the hot, dry atmosphere of other parts of India where evaporation from the surface is active. The occurrence of such perspiration does not, however, as in true malarial fevers, mark the advent of any terminal "sweating stage."

When the intensity is greater, and the temperatures reach 107° to 109°, other phenomena, characteristic of hyperpyrexia, intervene, of which a flushed face, rapid breathing and pulse, intensely hot, generally dry skin, injection of the conjunctiva, delirium, convulsions, and coma are the most constant. This is the "thermic fever," "heat apoplexy," or "insolation" of systematic writers. It differs from the other three only in intensity, and the prodromal symptoms, which have been insisted upon as an indication of some specific infection—the disinclination for exertion, the headache, anorexia, nausea, and hot, dry skin, etc., which precede the distinctive hyperpyrexia, are precisely those of the milder forms, and are preliminary to the final, often sudden, giving way of the thermic control, of which the post-mortem appearances, the pulmonary and cerebral congestion, and heart-failure are the natural expression. There is no essential difference between the phenomena of "febricula" and "heat apoplexy." It is merely one of degree.

With regard to the causation of this class of fevers the influence of climatic conditions cannot be denied. They require for their prevalence a high maximum daily temperature and an unclouded sky—solar traumatism being evidently the most potent factor—or an atmosphere laden with moisture. To demonstrate the effect of these conditions it is only necessary to "plot out" the admission-rate per 1000 for each month of the year for "simple continued fever," in two such different climates as those of Lucknow and Rangoon (making every allowance for errors in nomenclature).

The months of highest maximum temperature in Lucknow are March, April, May, and June. These are also the months of highest admission-rate for "simple continued fever." In June, with the advent of cloudy weather preceding

the burst of the monsoon rains in July, the admissions are fewer than in May, when there is nothing to veil the fierce heat of the sun. With the arrival of the rains there is a drop both in the admissions and in the atmospheric temperature, which never again rises above that of the body. In Rangoon, on the other hand, the hottest months are February, March, and April; and January, February, and March give the greatest number of admissions. In that part of Burma the monsoon breaks in May, and the rains are preceded and accompanied by cloud-laden skies, and during their continuance admissions for "simple continued fever" disappear from the returns. With the cessation of the rains in September admissions again take place in October.

An atmospheric maximum in the shade of 100° F. or more seems to be most conducive to the prevalence of this type of fever, and I have elsewhere pointed out (*Transactions of the Medical Society of London*, 1902) the difficulties which present themselves to the maintenance of normal temperature under conditions of high atmospheric temperature and humidity. This has also been shown experimentally in artificially heated chambers, the introduction of moisture immediately causing a rise of body temperature, which may then exceed physiological limits by many degrees; and I have also shown (*Indian Annals of Medical Science*, vol. xxxii. of 1874) that slight exertion in the conditions which prevail in India during the hot, moist months is capable of raising the body temperature to 99·9° F. The experience of troops on the march, unsuitably clothed and carrying weights, is to the same effect, and many men fall out under such circumstances with short febrile attacks. These phenomena show that the immediate cause of these fevers is probably failure of the control which is normally exerted by the nervous system in maintaining normal temperature when thermolysis is next to impossible, the atmospheric temperature being higher than that of the body, and evaporation from the surface hindered by great atmospheric humidity and heavy clothing. It is under such conditions that "heat-apoplexy," as distinguished from "sunstroke," so constantly occurs, the attack frequently occurring in the evening after sundown. Chills are also often noticed as the immediate cause of the symptoms, especially in individuals whose nervous system has been exhausted by long exposure to heat or impaired by the free use of alcohol. Alcoholics are indeed the most frequent subjects of these climatic fevers, especially heat-apoplexy. Europeans are much more liable to them than natives, and I do not think that long residence gives any immunity except such as may come from greater caution, the result of the teaching of experience.

Such are the conditions under which these fevers are most frequently seen, but it by no means follows that they constitute the whole case. With regard to the fourth member of the group, which Sambon prefers to call "siriasis," notwithstanding the etymological incongruity of the name, the capricious distribution of the disease, its varying prevalence in the same place from year to year, its apparent epidemicity at times, and its more or less definite course, have led him to argue that it is probably a specific fever due to some infecting germ, and similar arguments might be used with equal force to the other members of the group. The strongest evidence in this direction is, however, the fact that individuals, and especially children, who have come to reside in a temperate climate after a long residence in the tropics, where they may not have had any obvious fever, often exhibit a strong tendency to the occurrence of fever of this type for some years. Even those who are the subjects of the malarial cachexia contracted in the tropics, in whom the spleen often remains enlarged for many months after their return to Europe, suffer more often from a "febricula," an elevation of temperature for a few hours, on the smallest provocation, than from fever of an intermittent character. Each of these attacks is accompanied by a fresh enlargement of the spleen, and their liability to them continues until the spleen, from the effects of treatment, notably with arsenic, has assumed its natural size.

I know of no observations on the blood in these cases except those of Maynard and myself. In February and March 1895, the least malarious season in Calcutta, I found the malarial parasite present in less than 10 per cent of the cases of fever admitted to the European General Hospital. Maynard, working a few months later, found it in 28 per cent of admissions. From September to November, the most malarious season, they were detected in 75 per cent of all cases admitted to hospital. The rapid recovery of typical cases of this kind without quinine, the refusal of the more persistent cases to yield to that drug, and the absence of symptoms referable to the life-cycle of the parasite, lead to the opinion that its presence is accidental, and that it plays only a secondary part in the causation of fevers of this kind; and until extended and exhaustive bacterial research has been brought to bear on this group of fevers, the propriety of admitting them to the class of specific fevers must remain doubtful.

Nasha or Nakra.—Nasha or nakra (*nak* = the nose) is a continued fever of from three to five days' duration, accompanied by tumefaction of the nasal mucous membrane. It is also a fever of the hot months, being rarely seen except between April and August. It is especially prevalent in Lower Bengal, and appears to

affect natives only, males more than females, and children never. It is treated by local blood-letting by puncture of the swollen mucosa. It appears to cause considerable distress, but seldom gives rise to any anxiety. It is very doubtful whether it deserves to have a place as a specific fever, the nasal symptoms, which are diagnostic, being regarded by many merely as a local catarrh grafted on an ordinary febricula. An interesting discussion on this fever will be found in the *Transactions of the First Indian Medical Congress*, 1895.

"Low" Fever.—"Low" fever is a condition which seems also to be due to a climatic embarrassment of the heat-regulating centres, and differs from the other members of this group by its indefinite duration, which may cover a period of weeks and months without the development of any specific symptoms. It is characterised by a persistent low elevation of temperature, beginning insidiously with slight general malaise and anorexia. The temperature ranges from 99° in the morning to 101° or 101·5° in the evening, and it may continue for many weeks unaffected by quinine or arsenic pushed to extreme doses, or by any medicinal treatment, but generally yields at once when the patient is removed to more favourable climatic conditions. A case which has resisted all treatment for two months will get well immediately on a change to the "hills," or to sea, or even from one part of the same town to another. Cases have been known to persist for eight months, the patient continuing to follow his usual avocations until gradual loss of strength forces him to take the necessary change. It is not contagious, no other member of the family being attacked, though the patient mixes freely with them. If it were due to a specific organism, it would run a more definite course, and "change" would not have the immediate effect which follows it. This fever is not of great frequency, but it is sufficiently differentiated clinically to merit special bacteriological investigation.

Continued Fevers of Longer Duration and more Definite Course

Bastard Typhoid.—Instead of resolving in eight or ten days, certain cases of what may at first be regarded as "simple continued fever" run on to twenty-one or twenty-eight days. Their course is that of mild typhoid, but they do not manifest any of the clinical symptoms which are diagnostic of that disease. There are no spots, no pea-soup or other diarrhœa, no gurgling in the right iliac fossa, no delirium, and no marked tendency to adynamia. The spleen is not perceptibly enlarged, or if it is, this in a malarious country is of little diagnostic value. The temperature curve is that of mild typhoid. These cases are met with chiefly in the cold and early hot seasons, the seasons of typhoid prevalence, and they occur most frequently in individuals who have only been a short time in the tropics, as happens also with true typhoid. Their course is uninfluenced by quinine, they show no sign of periodicity, and you look in vain for the malarial parasite. They are met with oftenest in large cities, and there with such frequency that they have received local names, as "Calcutta fever" in Calcutta, "Bombay fever" in Bombay, etc., and for that reason I have distinguished them (*Brit. Med. Jour.*, 24th September 1898) as "urban continued" or "bastard typhoid" fever. There is very little doubt that they are really mild cases of typhoid fever, and the diagnosis could easily be established by the Widal test, as this fever occurs chiefly in Europeans. I am not aware that this has yet been done in India, but similar cases in South Africa have given a positive reaction in 75 per cent of those examined in this way. In one case of this kind under my observation there was sharp hæmorrhage from the bowel, but in the absence of diagnostic clinical symptoms of typhoid, practitioners in tropical towns have hesitated to include them under that name, having in their minds the possibilities of other bacteriological infections by other members of the coli group.[1] It is not improbable as I have suggested elsewhere (*Transactions of the Clinical Society for* 1901, vol. xxxiv. p. 127), that we are dealing here with the true "bed-rock" of typhoid fever, and that these cases are really due to a pure infection by the bacillus of Eberth, the characteristic phenomena of the clinical picture presented by typical typhoid being due to "mixed infection" from the admission to the blood of other organisms, and that it is these, strengthening and modifying the action of the bacillus typhosus, that give rise to the diagnostic features of typhoid, which are absent in the cases I am discussing. The experimental work of Sanarelli and Rémy add great weight to this view. They found that the addition of the B. coli communis to a culture of B. typhosus increased its virulence, and at the same time materially altered its cultural characters. The varying features presented in different epidemics of typhoid, the comparative frequency of thrombosis and muco-colitis, and the profuseness of the eruption, in South Africa for instance, are also in favour of such a theory. In view of these considerations it might be well to retain the name of "bastard typhoid" to designate cases which run the course of typhoid without its characteristic symptomatic and pathological phenomena.

Double Continued Fever.—Manson (*Tropical Diseases*, p. 247) mentions a form of continued fever which he encountered in South China, both in Amoy and Hong Kong, "characterised by an initial pyrexial stage of from ten days' to

[1] *Vide* articles on "Paratyphoid Fever," *American Journal of Medical Sciences*, Aug. 1902.

a fortnight's duration, followed by a stage of from three to seven days' relative or absolute apyrexia, which, in its turn, was succeeded by another spell of about ten days' duration of smart fever, and then by convalescence. Both in the primary and in the terminal fever the evening temperature may rise to 104° or 104·5°." There are no special symptoms, nor any special complications, as far as has been observed—the double fever being constant and characteristic; and the same succession of events may occur almost, in one instance quite, simultaneously in two patients living in the same house. Relapses of "simple continued fever," such as I have described in the first section of this paper, are of the rarest occurrence, and we may therefore assume that in the cases mentioned by Manson we are dealing with a specific infection of an altogether different character. I have not met with instances of this fever in India.

Non-Malarial "Remittent" Fever. — There, however, occurs in the tropics a fairly frequent form of continued fever which presents a very close clinical resemblance to the malignant type of malarial infection, with which it has been long, and is still confounded, though distinctions force themselves on the observation of attentive practitioners. The first point of distinction is that the spleen does not enlarge in these cases, or only in a very minor degree; and, second, quinine given in heroic doses does not in the smallest degree affect the course of the fever, but only adds materially to the distress of the patient; thirdly, the most careful and repeated search for the malarial parasite fails to find it. It is a continued fever of great severity running a definite course of about six weeks, and showing occasionally a tendency to relapse after an apyrexial period of about a week, the relapse being of the same type, but usually shorter and milder. It is more common in natives than Europeans, and children frequently suffer, while it is seldom seen after thirty years of age. It is called "remittent" because of its resemblance clinically to the "remittent" forms of malarial fever, but it is really a continued fever, and evidently due to some specific infection.

The attack often begins with distinct intermissions, as happens also with typhoid fever in the tropics, probably from the accidental presence of malarial parasites in the blood (Laveran), but after the first two or three days, notwithstanding large dosage with quinine, the fever becomes continued, and rapidly reaches continuous high temperature, 104° and 105° being the rule after the fourth day, with a remission of not more than 1·5° in many cases, though in others it amounts to 2° or 2·5° at some part of the twenty-four hours. With this continuance of high temperature, head symptoms are early and severe, with delirium and coma. The size of the spleen remains little altered, but the liver is early affected, becoming enlarged and congested, and a bilious diarrhœa, perhaps with slight jaundice, is the rule. From the eighteenth to the twenty-fourth day congestion of the backs of both lungs will generally be detected, and death is not uncommon about this period. In more favourable cases the symptoms gradually subside, and in another fortnight the attack is at an end. The firm belief that these cases are a form of malarial fever leads to treatment by repeated full doses of quinine, but without any benefit, and the distress which its indiscriminate use in these attacks causes has done a great deal to bring about the dread with which natives regard that drug, a dread which often prevents its effective use in other cases in which its beneficial results would be so conspicuous.

The question as to the true nature of the infection in these cases of non-malarial "remittent" has to some extent been answered by the discovery first made by Professor Wright of Netley and Surgeon-Captain Smith, that many convalescents from India give a positive reaction to the serum sedimentation test for Malta fever (*Brit. Med. Jour.* of 10th April 1897), and this reaction has since then been found to occur in India in many cases which formerly would have been regarded as malarial (Bamber, *Ind. Med. Gaz.*, Nov. 1900). I am not aware, however, that the *bacillus Melitensis* itself has yet been obtained from any fatal case of this fever, and looking at the wide divergence which the clinical symptoms exhibit from those of the typical course of Malta fever, and the absence of the sequelæ which are so characteristic of that disease, and the uncertainty as to the signification of the results of the serum test, I would, in the absence of detailed cases, join with Manson (*Lancet* of 23rd August 1902, p. 544) in advising caution in accepting such evidence as conclusive as to the prevalence of Malta fever in India.

Similar remarks apply with still greater force to the recent discovery of the presence of Widal's reaction in the blood of natives of India suffering from obscure attacks of continued fever, which has been advanced to prove that the belief that natives of India are comparatively immune from enteric fever must be abandoned, and that many attacks of this nature are in reality due to infection by the bacillus typhosus. Surgeon-Captain Freyer was the first to sound a warning note as to the doubtful diagnostic value of Widal's reaction in the examination of native blood. In dilutions of "about 1 in 12" he found—without mentioning a time limit—a positive reaction in the blood of sixteen out of eighteen specimens examined in healthy natives over two years of age (*Brit. Med. Jour.*, 7th August 1897, p. 329). Lamb of Bombay, applying the sedimentation test, failed to confirm Freyer's results (*Ind. Med. Gaz.*, April 1901, p. 123); but out

of eleven specimens taken from natives of India resident in London, in good health, and with no history of a recent attack of fever of any kind, a positive result by the microscopic method was obtained in seven instances, in dilutions of 1 in 50. Three of those whose blood failed to give any reaction were over forty years of age, and only one of eight below thirty failed altogether to show the presence of agglutinins. In four of these a well-marked reaction, which would have been sufficient in Europeans to establish the bacteriological diagnosis of enteric fever, occurred within half an hour: in the others it was more belated. It is clear, therefore, that conclusions based on the serum test in natives in India must be accepted with great caution, and though enteric fever may be of more frequent occurrence in some parts of India, as in Bombay and Poona (Lamb, *l.c.*, *Ind. Med. Gaz.*, February 1902), and in Madras (Elliot, *Ind. Med Gaz.*, March 1901, p. 115), especially though not exclusively in native Christians, Goorkhas, Burmans, and other natives who have abandoned a strict non-alcoholic and non-animal dietary, the belief in a comparative immunity from enteric fever enjoyed by natives on the whole, which is founded on the evidence of post-mortem examinations in the jails and large hospitals of India, remains unshaken (*vide Ind. Med. Gaz.* of May 1893). It argues rather a narrow view of the possibilities of bacteriology—a science still in its infancy—to limit our conceptions to those infections which are already known. It was this limitation which so long led clinicians to confound Malta fever with malarial and enteric fevers in spite of its well-marked and distinctive clinical symptoms.

Recurrent Fevers of Long Interval

The malarial parasites are organisms which have life-cycles of twenty-four, forty-eight, and seventy-two hours, giving rise to quotidian, tertian, and quartan ague respectively, and their life-history is incompatible with a regular recurrence of fever at longer intervals, and it is in our present state of knowledge impossible to ascribe to these organisms those fevers which are marked by a regular recurrence at intervals of a week, a fortnight, or a month. The regular recurrence of "relapsing fever" after a week of apyrexia has been found to be due to the *spirillum Obermeieri*; and "elephantoid fever" (an uncouth name which should now be abandoned in favour of "filarial fever"), with its regular recurrence, in typical cases, at intervals of a month, with its rigors, high temperature, and lymphangitis in those parts destined to be the seat of elephantiasis, is unquestionably caused by the presence of the filaria Bancrofti; but the cause of a fever, with a fortnight's interval without any specific phenomena except enlargement of the spleen, is as yet unexplained. These cases are of sufficient frequency to mark a distinct fever of specific causation. In the writer's experience the patients have mostly resided in the valley of the Brahmaputra, and have been the subjects of a fever coming with the greatest punctuality every fourteenth day. If the first attack occurs on a Thursday the subsequent attacks are on every alternate Thursday for two or three months, the febrile paroxysm lasting twenty-four to thirty-six hours. In the interval the patient is free from fever, but after several attacks the spleen will be found to be considerably enlarged, and ultimately all the phenomena of the malarial cachexia manifest themselves. We know of no modification of the known malarial parasites capable of causing a fever of this character, and the punctual regularity of the recurrence precludes the idea of accidental infection by them. It suggests infection by some organism with a life-cycle of fourteen days, possibly a hæmamœba of a yet unnoted kind. Quinine in full doses has, in my experience, invariably broken the sequence of events.

Fevers of Mixed Origin

Typho-Malarial Fever. — In the tropics the combination of malarial infection with that of typhoid serves to obscure the early symptoms of enteric fever, giving rise in many cases to a violent and sudden access, instead of the gradual step-like rise of temperature which is so characteristic of it in temperate climates, and in such cases convalescence is often delayed by several days of intermittent fever, when the course of the typhoid symptoms appears to have ended. At these initial and terminal stages the malarial parasite may be detected in the blood. It was in such cases of "congestive" or "adynamic remittent" fever that the older writers remarked the presence of ulceration or congestion of the small intestine in post-mortem examination (Annesley, *Diseases of India and Warm Climates*, 1828, and Twining, *Diseases of Bengal*, 1835). Otherwise the ordinary course of typhoid fever is little modified by this combination with malaria, and I do not think that it is entitled to a separate place in nomenclature. In many cases diagnosed as typhoid fever in the tropics, the post-mortem appearances are by no means characteristic (*vide Annual Reports of the Government of India*, and my *Report on the Fevers of Burma*, 1876), but this argues a "mixed infection" with some other bacillus, rather than with a hæmamœba.

Kala-Azar. — This is a very fatal malady, which for the past fifteen years has been devastating portions of Assam, especially in the district of Goalpara, a low-lying malarious district between the Brahmaputra and the Garo Hills. It has been investigated by Giles, Rogers, and Ronald Ross. It is characterised in its advanced stages by extreme anæmia, great and

progressive debility, intercurrent attacks of fever, enlargement of the spleen and liver, darkening of the complexion, and dropsy. Except during the intercurrent attacks of fever, the temperature is markedly subnormal, as in other anæmias. Two views are taken of its essential nature. Giles, finding the ova of the ankylostoma duodenalis present in the fæces of practically every case he examined, regarded kala-azar as essentially an ankylostomiasis, and the concomitant signs of malaria as accidental and only constant because of the highly malarious surroundings of those affected. Rogers and Ross take the opposite view, and look on the malarial infection as essential, and the ankylostomiasis as accidental. They found the malarial parasite present in the blood in all early cases, and that at that period the disease was undistinguishable from ordinary malaria, except, according to Rogers, that the parasite was of unusual virulence. There is no doubt that the ankylostoma is exceedingly prevalent in other parts of India where kala-azar is unknown; but there are few districts which combine so great a prevalence of this parasite with so virulent a form of malaria as exists in the parts of Assam decimated by kala-azar, and pending further study of this disease it may with fair reason be attributed to this combination. This is the view expressed by Giles himself (*B.M.J.*, 26th March 1893). The best results of treatment are obtained by the early use of quinine and thymol.

Hæmoglobinuric Fever.—This is the "Blackwater fever" of tropical Africa. It has also been reported in the hotter parts of America, the West India Islands, the Eastern Archipelago, in the south of Japan, in Assam, and in other districts of India. It is also met with in southern Europe, Greece, Italy, and Sardinia, in Algeria, and in the Koomati Poort district of South Africa. It is certainly not prevalent generally in India, and it is by no means clear from the reports received that the observers have always been careful to distinguish between hæmoglobinuria and hæmaturia. It is hardly possible that a fever with so marked peculiarities could escape notice, and the returns of the large hospitals of India are silent with regard to it. It may be that a recent introduction of the infective germ has taken place, just as is believed by many to have occurred in tropical Africa itself.

It is characterised by pyrexia, which is ushered in by rigor, bilious vomiting, with icterus, hæmoglobinuria, and diminution in the quantity of urine. It is extremely fatal, the deaths being in the proportion of 75 per cent of attacks. The pathognomonic symptom is strongly suggestive that the infective organism is related to the *Pirosoma bigeminum* of Texas cattle fever, in which hæmoglobinuria is also the constant symptom, but as yet all attempts to detect this or any other specific organism in man have failed. (*See also* "BLACKWATER FEVER," vol. i. p. 376).

Its association with malaria is undoubted. All the endemic areas in which it is met with are noted for malaria of a severe and virulent type. This is true of those districts of India where it has been reported, and also of Koomati Poort, the last addition to the list. It usually occurs in individuals who, being residents in these areas, have had several previous attacks of ordinary malarial fever, and it is unusual in the first year of residence, being most frequent in the third year. It may, however, occur without any previous attack of malaria; of this many instances have now been recorded. At the beginning of the attack parasites undistinguishable from those of a mixed malarial infection will be found in the blood, belonging generally, but not necessarily, to the malignant, crescent-forming type. On the appearance of hæmoglobin in the urine these generally disappear from the blood, although quinine may not have been given, and this may terminate a chronic malarial infection.

Two explanations would seem to be possible of these phenomena. First, that the occurrence of hæmoglobinuria is due to the addition of a distinct specific infection, of less frequency than, and inimical to, the malarial parasite, destroying it along with its containing red-blood corpuscles; or, secondly, it may be due to a special virulence of the toxin formed by the malarial parasites, which not only destroys the corpuscles which they inhabit, but is also fatal to their own existence. In the present state of our knowledge the latter is the more likely hypothesis, which would, however, be immediately set aside by the discovery of a specific infective organism.

Koch's idea that the cause of the hæmoglobinuria is the quinine given to cure the malarial infection cannot be entertained; in the first place, because it may occur, with a fatal result, in cases in which the drug has not been given, and in the second place, because quinine may be given, and is constantly given in heroic doses, in districts where hæmoglobinuric fever is not endemic without its ever inducing hæmoglobinuria. In my own experience in Lower Bengal, sixty, and even ninety grains of quinine in twenty-four hours was no unusual dosage in malarial attacks, and I only saw the occurrence of hæmoglobinuria in one instance, which was one in which quinine was not being pushed.

Natives of an endemic area seem to acquire a relative immunity from hæmoglobinuric fever which is not enjoyed by immigrants of the same race.

The teaching of Koch has led some to suspend the use of quinine on the occurrence of hæmoglobinuria; others regard it as an indication for increased doses. The treatment, how-

ever, which seems to have the most marked beneficial effect is the subcutaneous injection of large quantities of normal saline solution. This treatment is said to have resulted in the recovery of fifty-five consecutive cases so treated. Its effect is probably a mechanical one, that of clearing the renal tubuli blocked with hæmoglobin infarcts, and increasing the blood-pressure (Gouzien, *XIII^E Congrès Internat. de Méd., Sous-section, Méd. Coloniale*, p. 155).

Tropidonotus Natrix. *See* SNAKE-BITE IN THE BRITISH ISLES (*Species of Serpent*).

Tropometer. — An instrument for measuring the rotations of the eyeball or the amount of torsion of long bones (Gr. τροπή, a turn).

Trough Closet. — A form of urinal for public use, in which the urine accumulates in a trough containing water which is periodically flushed by an automatic apparatus. *See* SEWAGE AND DRAINAGE (*Closets*, Fig. 8).

Trousseau's Disease.—Vertigo due to stomach disorder.

Trousseau's Marks.—The "taches cérébrales" seen in tuberculous meningitis and some other maladies of the nervous system. *See* TACHE and *Cross References*.

Trousseau's Symptom.—The fact that in tetany, so long as the attack is not over, one may reproduce the paroxysms by pressing the parts affected so as to interfere with the blood-supply or to compress the nerve trunks. *See* TETANY (*Motor Symptoms*).

True.—Real, not spurious, as in such expressions as *true pelvis* (the pelvis below the plane of the brim), *true skin* (the corium), *true rib* (one attached directly to the sternum anteriorly), and *true corpus luteum*, etc. *True image* is the image formed upon the normal eye in diplopia, that on the affected eye being called the false. *See* PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Binocular Vision*).

Trusses. *See* HERNIA (*Treatment, Trusses*).

Trypanosoma. *See* SLEEPING SICKNESS (*Cause*); PARASITES (*Mastigophora*).

Trypanosomiasis.—The diseased condition due to the presence of the trypanosoma (any species) in the blood. *See* SLEEPING SICKNESS.

Trypsin.—The proteolytic enzyme or ferment of the pancreas; it has been lauded as a remedy for malignant disease, but its beneficial effect has not been fully established; indeed it has rather seemed as if it had more effect upon tubercle than upon cancer (in some recently reported observations). *See* PHYSIOLOGY, FOOD AND DIGESTION (*Pancreatic Secretion*); ENZYMES (*Trypsin*); PANCREAS, PHYSIOLOGY OF (*Enzymes*); SNAKE-BITES (*In Cobra Poisoning*); TEMPERATURE (*Causes of Fever, Subcutaneous Injection of Trypsin*).

Trypsinogen. — The antecedent of trypsin. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Pancreatic Secretion*).

Tryptophane. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Pancreatic Secretion, Action*).

Tscherning's Theory. *See* ACCOMMODATION (*Theories*); IRIS AND CILIARY BODIES (*Physiology of, Accommodation*).

Tsetse Fly. *See* PARASITES (*Trypanosoma Brucei*); SLEEPING SICKNESS.

Tubal.—Belonging to a tube, especially to the tubules of the kidney and to the Fallopian or Eustachian tubes; *e.g.* tubal pregnancy, tubal disease, etc. *See* ECTOPIC GESTATION (*Tubal Pregnancy*); MOLE, MOLAR PREGNANCY (*Tubal Mole*); PERITONEUM, ACUTE PERITONITIS (*Diagnosis from Ruptured Tubal Pregnancy*); PREGNANCY, AFFECTIONS OF GENERATIVE ORGANS (*Retroversion of Gravid Uterus, Diagnosis*).

Tube.—A hollow, cylindrical structure, such as the Eustachian or Fallopian tube in anatomy, or the glass, indiarubber, or metal tubes used for drainage, for forced feeding, tracheotomy, etc.

Tube-Casts. *See* URINE, PATHOLOGICAL CHANGES IN (*Urinary Sediments, Casts*).

Tuber.—A projection or rounded swelling (plural, *tubera*) such as the *tuber cinereum*, the *tubera ischii*, and the *tubera mentalia* of anatomy.

Tubercle.—A small tuber or projection, such as the deltoid tubercle on the clavicle, the tubercles of Montgomery in the areola round the nipple of the breast, and the subcutaneous tubercles of rheumatism; also the specific lesion produced by the tubercle bacillus. *See* TUBERCULOSIS.

Tubercula.—The plural of *tuberculum*, a tubercle (*q.v.*).

Tubercular.—Having the shape or appearance of a tubercle, or, more frequently, being of the nature of tuberculosis, and characterised by the presence of the tubercle bacillus. *See* TURERCULOSIS; MENINGITIS, TUBERCULOUS; etc.

Tuberculide.—A cutaneous disease regarded as due to the toxinic products of the tubercle bacillus.

Tuberculin.—Koch'slymph. *See* TUBERCULOSIS (*Chemistry and Chemical Products of the Tubercle Bacillus*).

Tuberculocidin. *See* TUBERCULOSIS (*Chemistry of the Tubercle Bacillus and its Products*).

Tuberculoderma. *See* TUBERCULIDE.

Tuberculosis.

THE TUBERCLE BACILLUS—
STAINING METHODS 119
BIOLOGICAL CHARACTERS . . . 120
Human, Bovine, Avian . . 120
RELATION TO STREPTOTHRICES . . 121
DIAGNOSIS OF TUBERCLE BACILLUS . 121
SEPARATION AND CULTIVATION . 123
TUBERCULIN, ETC. 124
TUBERCULIN AS A DIAGNOSTIC AGENT . 125
MORBID ANATOMY—
Lesions produced by the Tubercle Bacillus 126
MIXED INFECTION 138
TUBERCULOSIS IN MAN AND THE LOWER ANIMALS 140
Avian Tuberculosis 140
Piscine Tuberculosis 141
Relation of Human and Bovine Tuberculosis 141

See also ALCOLOL (*Indications*); ALOPECIA (*Varieties and Etiology*); ANÆMIA (*Diagnosis, Causal Factors*); APPENDIX VERMIFORMIS (*Tuberculosis of Appendix*); ASCITES (*Recognition of Cause*); ASPHYXIA (*Artificial Respiration, Inflation, Dangers of*); BLADDER, INJURIES AND DISEASES OF (*Tuberculous*); BONE, DISEASES OF (*Tuberculous*); BRONCHI, BRONCHITIS (*Diagnosis*); BRONCHI, BRONCHIECTASIS (*Diagnosis*); BURSÆ, INJURIES AND DISEASES OF (*Tubercular Bursitis*); CHLOROSIS (*Diagnosis*); CHOROID, DISEASES OF (*Tuberculous Disease*); CONJUNCTIVA, DISEASES OF (*Tuberculosis*); DIATHESIS (*Tuberculous*); EAR, MIDDLE, CHRONIC SUPPURATION (*Tuberculous Disease*); EXPECTORATION (*Tuberculosis*); FÆCES (*Microscopical and Bacteriological Examination of*); HÆMOPTYSIS (*Etiology*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Morbid Processes, Tubercular Lesions in Inflammation*); IMMUNITY (*Protective Inoculation, Tuberculin, Serum Therapeutics*); IRIS AND CILIARY BODIES (*Tuberculous Irido-Cyclitis*); JOINTS, DISEASES OF (*Tuberculous*); KIDNEY, SURGICAL AFFECTIONS OF (*Renal Tuberculosis*); LUNG, TUBERCULOSIS OF; MALARIA (*Diagnosis*); MAMMARY GLAND, DISEASES OF (*Tuberculosis*); MEDIASTINUM (*Growths, Diagnosis*); MELÆNA (*Diagnosis*); MENINGITIS, TUBERCULOUS; MENSTRUATION AND ITS DISORDERS (*Amenorrhœa, Primary, Causes of*); MICRO-ORGANISMS (*Streptothrices*); MILK (*Pathological, Control*); MOUTH, INJURIES AND DISEASES OF THE JAW (*Tuberculous*); MUSCLES, DISEASES OF (*Tuberculous Myositis*); NERVES, PERIPHERAL (*Neuritis, Causes*); NERVES, MULTIPLE PERIPHERAL NEURITIS (*General Etiology*); NOSE, EXAMINATION OF (*Secretion, Bacteriological Examination*); NOSE, CHRONIC INFECTIVE DISEASES; NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Etiology*); NURSERY HYGIENE (*Mouth and Teeth*); ŒSOPHAGUS (*Rare Affections*); ORBIT, DISEASES OF THE (*Inflammatory*); OSTEO-ARTHROPATHIES (*Pulmonary*); OVARIES, DISEASES OF THE (*Tubercle*); PALATE (*Diseases, Infective Granulomata*); PANCREAS, DISEASES OF (*Tubercle*); PERICARDIUM, DISEASES OF (*Pericarditis, Etiology*); PERITONEUM (*New Growths, Tubercular Peritonitis*); PERITONEUM, TUBERCULOUS PERITONITIS; PHARYNX, CHRONIC INFECTIVE DISEASES (*Tuberculosis*); PLEURA, DISEASES OF (*Bacteriology of Pleural Effusions*); PNEUMONIA, CLINICAL (*Etiology, Tuberculosis*); PNEUMONIA, CLINICAL (*Complications and Diagnosis*); PREGNANCY, DISEASES OF PLACENTA AND CORD (*Tuberculosis*); PREGNANCY, INTRAUTERINE DISEASES OF FŒTUS (*Tuberculosis*); PROSTATE GLAND (*Tuberculosis*); PUERPERIUM, PATHOLOGY (*Septicæmia, Diagnosis*); PULSE (*In Tubercular Meningites*); SCROTUM AND TESTICLE, DISEASES OF (*Tuberculosis of Testicle*); SEPTICÆMIA (*Diagnosis*); SHOULDER, DISEASES OF (*Tuberculous Disease*); SKIN, TUBERCULOSIS OF; SPLEEN, MEDICAL AFFECTIONS; STOMACH AND DUODENUM, DISEASES OF (*General Etiology*); TEETH (*Bacteriology of Mouth*); THERAPEUTICS, HEALTH RESORTS; TONSILS, DISEASES OF (*Tubercle*); TRADES, DANGEROUS (*Cotton*); TYPHOID FEVER (*Complications, Diagnosis*); ULCERS AND ULCERATION (*Classification*); UNDULANT FEVER (*Diagnosis*); URINE, BACTERIA IN (*Tubercle*); X-RAYS (*Uses*).

DEFINITION.—Tuberculosis is a chronic infective disease caused by the tubercle bacillus.

Man and all domesticated animals may suffer from it. Wild animals, particularly the carnivora, are insusceptible. While this is so, it is probable that all animals may be infected by tuberculosis if placed in suitable environment and with suitable doses of the organism.

It has been stated that 7 per cent of mankind die from tuberculosis, and that from 30 to 50 per cent of cows in Britain are tuberculous.

ETIOLOGY.—In the causation of the disease one must bear in mind two factors, the soil and the germ. In the absorbing discussions which are taking place concerning the tubercle bacillus and its modes of entrance into the body, we are apt to give insufficient weight to the personal factor. It is now generally concluded that the bacillus is only capable of taking root in individuals whose nutrition and power of resistance are greatly lowered. In fact, tuberculosis may be looked on as the sequel of another debilitating condition due to accident or disease.

As, however, the tubercle bacillus bulks so largely in our consideration of the disease, we shall first consider it morphologically and biologically, returning later to the personal factor in the disease, when considering the portals of

infection and channels of spread in the body and the relationships of human to bovine tuberculosis.

HISTORICAL.—1. Tuberculous lesions were first supposed to be evidences of a weak form of inflammation, occurring in a weak or scrofulous subject, no specific cause being assigned.

2. It was noticed that if the disease began at one spot, generalised tuberculosis might follow.

3. Niemeyer and Virchow considered that the formation of the tubercle nodule was not a specific process, but that casation occurred because the inflammation was of a low degree; that the blood-supply was cut off by pressure of inflammatory products; that in the caseated material a ferment developed, and when the caseous material softened and broke into a blood-vessel, the ferment entered the blood and caused general tuberculosis.

4. Klencke in 1843 produced tuberculosis in rabbits by intravenous injection of tuberculous material, but the real significance of this was not recognised at the time, and by demonstrating the inoculability of tuberculosis in the lower animals, Villemin has the credit of being the first to show that the cause of the disease is a specific one. He took pieces of tuberculous material and successfully inoculated various animals, proving that the disease was infective (1865).

5. Koch and Baumgarten demonstrated microscopically the organism which causes the disease, and Koch succeeded, after many failures, in cultivating it on artificial medium. Koch's announcement of his discovery was made public on 24th March 1882. His demonstration of the relationship of the bacillus to the disease was so complete that the facts were at once accepted by scientists throughout the world. He laid down with the greatest precision that there was but one form of the tubercle bacillus; that *Perlsucht* in cattle, pulmonary tuberculosis in man, and tuberculosis in domestic animals, were all caused by one and the same micro-organism. Since the bacillus became available in pure culture, experiments on animals have more and more decisively demonstrated its infective nature, and somewhat modified Koch's first conclusions.

The proofs of the relationship of the bacillus to the disease may be stated as follows:—

1. The bacillus can be readily demonstrated in tuberculous lesions in man and in the lower animals.

2. It can be separated from such lesions and obtained in pure culture.

3. Inoculation of these pure cultures, and of subcultures from them, reproduces in susceptible animals the same disease, with macroscopic and microscopic lesions corresponding to those found in the natural disease.

4. From these experimental lesions the bacillus can be recovered in pure culture.

5. The chemical substances produced by the bacillus in cultivation and in the body are related.

THE TUBERCLE BACILLUS (*Bac. tuberculosis*, Koch; Syn. *Mycobacterium tuberculosis*).—The bacillus as ordinarily found in man is a short narrow rod of the length of one-quarter to one-half the diameter of a red blood corpuscle (1·5 to 3·5 μ). The breadth (*circ.* 0·2 μ) is pretty constant, provided the same method of staining be used. The ends are rounded or pointed. The rods are not as a rule absolutely straight; they are commonly slightly curved or bent at an angle; sometimes they are slightly sinuous or spiral, and occasionally adjacent rods lie at an obtuse angle with each other. Frequently there is an appearance of "beading," so that the rod presents deeply stained thicker parts with intervening clear spots. These were formerly supposed to be of the nature of spores, but it is more probable that they are not, one of the most cogent arguments against this interpretation being that the tubercle bacillus can be killed by a temperature far below that necessary to destroy spores. Some authorities suppose that the appearance may be an "artefact," produced in process of preparation of the microscopic film, but it occurs too constantly for this. Bulloch looks upon the clear spaces as degenerative in character.

It must be remembered that many competent observers consider that spores are present in certain stages of the life-history of the bacillus.

Occasionally the bacillus may fail to take the stain—for example, in some miliary lesions in the lung, lupus, and tuberculous disease of bone—but on inoculation of such tissue the disease may be reproduced.

The numbers of well-formed bacilli present in a lesion or discharge may, roughly speaking, be taken as evidence of the activity of the process.

The *bovine* bacillus is shorter than the human, seldom more than one-quarter the diameter of a human red blood corpuscle (2 μ) and averaging less. They are thick and straight. They stain evenly and deeply, and "beading" is usually absent. *Avian* tubercle bacilli are similar microscopically to the mammalian type, but present marked difference in culture and on experimental inoculation, so that many consider that they are of different species.

STAINING METHODS.—The methods of staining the tubercle bacillus depend on the difficulty with which it receives the stain, and the tenacity with which it holds the stain when it has entered.

Ehrlich has undoubtedly the credit for first pointing out these peculiar properties of the bacillus. It was found out at a very early stage that ordinary watery solutions of anilin dyes penetrated tubercle bacilli with extreme slowness and imperfectly. Ehrlich showed that by combining a basic anilin stain (gentian violet) with anilin water, its penetrating power was increased,

and he also showed that when once stained the organism was able to resist the decolorising power of acids, even in strong solution, *e.g.* 33 per cent nitric acid or 25 per cent sulphuric acid, and even after exposure to these strengths for so long a period as a quarter of an hour. The stain now generally employed is *basic* fuchsin (rosanilin hydrochlorate).

The property of resistance to the decolorising action of acid, or "acid-fastness," was supposed by Ehrlich to be produced by some substance surrounding the bacillus, forming a covering permeable for anilin water, alkalies, carbolic acid, etc., but impermeable for acids. It is not possessed by very young cultures, as shown by Klein and later by Marmorek. Borrel, by protracted action of warm xylol on tubercle bacilli, extracted a substance resistant to acids and alcohol after treatment with stain. The tubercle bacilli had lost these qualities, but could still produce tubercles.

Unna and Klebs, de Schweinitz and Dorset, supposed that acid fastness depended on the presence of fat in the bacillus, and there is no doubt that fatty acids, at any rate, are important constituents of the bacillus. Aronson (1898) has, however, shown that the body which gives the acid-fast character is of the nature of a wax, and this view has been confirmed by Bulloch and M'Leod.

These observers removed the substance by solvents of wax, and found that fat, fatty acids, and wax constituted 43·7 per cent by weight of the dried tubercle bacilli.

The changes which go on during the staining procedure may now be explained. When the fuchsin-stained bacilli are immersed, for example, in sulphuric acid, the mono-acid rosanilin salts are converted into the soluble brownish tri-acid compounds which diffuse out of bacteria, which are not acid-fast. In the case of tubercle bacilli this change takes longer than in the case of other bacteria, not setting in, apparently, until some change in the permeability of the bacillus has been induced by the acid. The subsequent washing in water leads to a decomposition of the tri-acid compound with re-formation of mono-acid fuchsin, and the bacilli again appear red, as the fuchsin does not diffuse out (Bulloch).

A similar resistance is also manifested towards organic acids such as acetic, formic, oxalic, tartaric, lactic, and picric acids.

Recently other organisms, morphologically very similar to the tubercle bacillus, have been discovered which are also "acid-fast," and in some of these a distinction is made by their being less resistant to decolorisation by alcohol than the tubercle bacillus. In the case of others, however, *no known staining method differentiates from tubercle*, and diagnosis from biological characteristics must be had recourse to.

With a view to demonstrating the bacilli in sputum, for example, it is well to select that first expelled in the morning. Pour it out on a flat dish, and pick out any small yellow caseous points, or, failing these, the purulent-looking parts. With a sterilised platinum loop place the selected material on an absolutely clean cover-glass (No. 1 thickness preferably), and smear in a thin layer over it; or better, superimpose another cover-glass, press gently so as to secure a thin film, and *slide* apart. It is well to place the cover-glass between clear filter paper before pressing, so that any excess of sputum soaks into the paper, which should be at once destroyed by burning.

For subsequent procedure refer to article on "Post-mortem Methods," vol. viii. p. 216.

In the Pathological Department of the Royal Infirmary of Edinburgh, in the routine method of examining tuberculous sputum and discharges, the periods of exposure to alcohol and acid are one minute and two minutes respectively.

Biological Characters of the Tubercle Bacillus.—As the bacilli of human, bovine, and avian tuberculosis in cultivation show certain fairly constant differences, which justify their classification as distinct varieties or races, they must be described separately; nevertheless, at a later stage data will be brought forward which indicate, contrary to Koch's rather arbitrary dictum, that there is a close relationship between these varieties—at least, between the two first, and that the apparent differences most probably depend on modifications of one original organism.

Human Bacillus.—On inspissated blood serum at a temperature of 35° to 37° C., the colonies appear from the tenth to the fourteenth day as discrete, dry, scaly growths, greyish in colour. These gradually extend and coalesce so as to form a thin, wrinkled layer on the surface of the medium, reaching full development in six or seven weeks. In subcultures the growth tends to become more voluminous, and occasionally becomes brownish in colour in old cultures. The bacillus is non-motile. It is aerobic and facultative anaerobic. While its optimum temperature for growth is 35° to 37° C. (98° to 101° F.), it can develop from 28° to 42° C. Its thermal death-point is about 70° C.

It is still doubtful whether it has a saprophytic stage of existence, and this question will be discussed later in considering the relationship of the tubercle bacillus to the streptothrices. Up to the present time it has been looked on as —in natural surroundings—strictly parasitic, developing only in the tissues of tuberculous animals.

It grows well on inspissated blood serum, and this medium is used in separating it in pure culture. Subcultures grow well on peptone agar to which 5 to 8 per cent of pure glycerine has been added, also on veal broth containing 5 per cent glycerine (Nocard and Roux), or on

potato smeared with glycerine in sealed tubes (Powlowski), and even on other vegetables and vegetable infusions to which glycerine has been added. On these media, however, the bacillus rapidly loses its virulence.

Other media will be referred to when treating of the separation of the tubercle bacillus from lesions or fluids.

Adult cultures of the *Bovine* bacillus are somewhat similar to those of the human organism, though for several generations they are very thin and delicate, growing less luxuriantly than the human. They cannot as a rule be induced to develop on glycerine agar. On blood serum, cultures approximate in character very closely to those of the human bacillus.

Avian Tubercle Bacillus.—While the bacilli of avian tuberculosis correspond very closely to those of the mammalian type in their microscopic appearance and staining reactions, they differ considerably in culture and in results of experimental inoculation.

The growth on the surface of blood serum and glycerine agar is much more voluminous, forming soft, whitish, moist masses. The organism grows not only when glycerine is present, but on ordinary agar-agar and beef-broth. It will not grow upon potato. Development takes place at a temperature (43° C.) at which mammalian tubercle ceases to grow. On experimental inoculation, guinea-pigs usually resist infection, and intravenous injection even of large quantities of the avian bacilli in the case of dogs fails to produce any effect.

Nevertheless, Nocard has proved that mammalian bacilli may be modified so as to acquire all the characters of the avian bacilli, so that the apparent difference evidently depends on environment. Again, tuberculin prepared from avian bacilli has the same action as mammalian tuberculin.

Relation of the Tubercle Bacillus to Streptothrix Forms. — While the tubercle bacillus is of greatest importance in the form most commonly met with—that of the rodlet—later researches have shown, apparently on good grounds, that this form may be but an early form of an organism which develops into a more highly constituted filamentous fungus. Some authors have gone so far as to place its adult form in or near the streptothriceæ or actinomycetes.

Metschnikoff found the organism as short filaments in sputum and in the splenic pulp of tuberculous birds. Fischel and Burns found similar forms, and Burns considered them to be the saprophytic form of an organism whose parasitic form was a rodlet. Metschnikoff, in 1888, proposed to call the saprophytic form Sclerothrix Kochii.

Coppen Jones (1895) found similar appearances in sputum and in old cultures, and demonstrated dichotomous branching and clubbing. In cultures the filaments were found on the surface of the growth. They contained certain deeply staining bodies, which possessed some of the properties of spores, and he considered that these were of the nature of chlamydospores.

Many observers have confirmed these observations. Babes and Levaditi found similar aberrant forms, in rabbits injected with cultures under the dura mater, at the end of four weeks.

Friedrich, Nösske and Schültze concluded from experiments that such forms developed in lesions caused by injection of virulent cultures.

It must, however, be remembered that other bacteriologists, for example, Bostroem and Fischer, looked upon clubbed forms as resulting from a degenerative change in the sheath, whilst others, Lubarsch and Schultz, look upon them as malformations from interrupted growth.

Abbot and Gildersleeve (1902) state that other acid-resisting bacilli also present these characteristics, and conclude that as these also produce on experimental inoculation nodular growths with many features in common with the lesions of tubercle and actinomyces, all these organisms ought to be classed together.

In the present state of our knowledge, as Bulloch remarks, "however seductive the theory may be that the tubercle bacillus, so called, is really a filamentous fungus with a relatively complicated structure, it is perhaps wiser to wait for more conclusive evidence, as the issues at stake are so vital for the doctrine of the origin and transmission of tuberculosis."

Nevertheless, sufficient evidence has been brought forward to emphasise the growing tendency to associate the tubercle bacillus with the streptothriceæ.

Diagnosis of the Tubercle Bacillus.—The difficulties in the way of diagnosing tubercle bacilli from other acid-fast organisms has already been indicated. Bacilli morphologically similar to those of tubercle are found in many non-tuberculous lesions in man. Though as a rule pseudo-tubercle bacilli are less resistant to acids and alcohol than the genuine organism, this is not true of all varieties.

A. Fraenkel stated in 1898 that he had repeatedly found acid-resisting bacilli in the sputum obtained from cases of non-tuberculous gangrene of the lung, and his observations have since been confirmed by Pappenheim and Lydia Rabinowitsch. Lubarsch (*B. M. J. Ep.*, 14th June 1902) shows that these pseudo-tubercle bacilli may occur in a variety of conditions. In a case of gastric cancer, with pleural and pulmonary metastases and purulent bronchitis, accompanied by rapid emaciation, he found acid-resisting bacilli which were innocuous to guinea-pigs. He found similar bacilli in a bronchiectatic cavity; in an abscess near the hip-joint in a child one year old; in small cystic

swellings, containing cheesy material, on the forearm of an adult who as a child had had tuberculosis. The swellings had the microscopic characters of sebaceous cysts. In the last case the explanation that the bacilli were dead or attenuated is not altogether outwith the realms of possibility. He concludes that the only reliable test is injection of suspected material into susceptible animals.

This does not solve the difficulty, however, as other observers have shown that acid-fast organisms—not tubercle—can produce experimental lesions identical with those of tubercle, so that we are thrown back again on cultural characters to control experimental results.

Möller observed the presence of acid-fast organisms in the mouth and sputum of non-tuberculous patients. Marzinowski found similar bacilli in the tonsil. Dietrich, in 1899, found acid-fast bacilli, resembling but distinct from those of tubercle, in a suppurating cyst of the ovary which had ruptured into the intestine. The chief complication in the diagnosis of tuberculosis of the urinary tract in man is the presence of the *smegma bacillus* in the urine. This cannot be distinguished by its morphological characters alone from the tubercle bacillus. The tubercle bacillus is exceedingly pleomorphic, and may simulate the usually shorter and thicker smegma bacillus; and, on the contrary, the latter may occur as thin granular rods, indistinguishable from the typical organism of tuberculosis. As a rule, however, the smegma bacillus is not so resistant to decolorisation by alcohol as the tubercle bacillus, and so can be distinguished from it. It is harmless when inoculated into animals. The smegma bacillus was first discovered by Tavel and Alvarez, and fully described by Kruse.

Pappenheim recommends the following method of differentiating the tubercle bacillus from the smegma bacillus:—

1. Stain in the ordinary way with carbol-fuchsin.

2. Decolorise without washing in water, by immersing 3-5 times in a solution composed of absolute alcohol 100 parts, corallin 1, methylene blue to saturation, glycerine 20 parts.

3. Wash, dry, and mount in balsam.

The tubercle bacilli appear red, the smegma bacilli blue.

The bacillus of *Leprosy* is morphologically similar to that of tuberculosis, but it may be distinguished by being found in "lepra" cells, in the nodules in the disease, or lying in the sheaths of the nerves in the anæsthetic form. Moreover, they stain with ordinary aqueous aniline dyes, and at room temperature, whereas this fails with tubercle. When stained by Ziehl Neelsen's method they decolorise more readily.

G. van Houtum claims to have isolated the specific bacillus of leprosy (*Jl. of Path. and Bact.*, Sept. 1902).

Lustgarten's *Syphilis* bacillus also resembles the tubercle bacillus, but it still possesses a doubtful individuality, being possibly simply the smegma bacillus. In any case it has not been found in sufficient numbers, or with sufficient constancy in syphilitic lesions, to merit further attention here. The tubercle bacillus also stains by the complicated methods recommended by Lustgarten, and Baumgarten suggests that the few cases in which Lustgarten's bacillus has been found may be cases of mixed infection of tuberculosis and syphilis.

In certain domestic animals, and in foods derived from them, numerous acid-fast bacilli have been discovered. They are extremely common in milk. Many of these, however, can be decolorised with alcohol. Others, however, resemble tubercle bacilli in their properties and characters so closely that diagnosis is very difficult.

Acid-fast organisms, not tubercle bacilli, have been found by Murray Cowie in films made from scrapings from the udders and teats of healthy cows.

Similar organisms are frequently found in butter and cheese. The chief butter bacillus is one isolated by Petri and Rabinowitsch (*Mycobacterium butyri*). It is very like the tubercle bacillus, but now and then is thicker. In its protoplasm deeply coloured granules are sometimes visible, as in that bacillus. It grows at room temperature, and at incubator temperature there is visible growth after twenty-seven hours. Inoculation into guinea-pigs gives rise to nodular swellings resembling to some extent tuberculous infiltrations, but none of the animals inoculated reacted to tuberculin.

In this connection it has been reported that true tubercle bacilli, when mixed with melted butter and injected into the peritoneal cavity, do not produce a generalised tuberculosis, but simply a localised fibrous thickening of the peritoneum.

Korn claims to have isolated another bacillus from butter similar to tubercle, but differing in some respects from the Petri-Rabinowitsch bacillus.

Möller isolated acid-fast bacilli from cow-dung presenting certain characters distinguishing from tubercle bacilli and from other acid-fast organisms.

The same investigator created great interest by the announcement of the discovery on certain grasses of acid-fast bacilli closely resembling those of tubercle. One was isolated from timothy grass (*Phleum pratense*), and hence he named it the Timothy grass bacillus, or Grass bacillus I. It has since been found on other grasses, occurring widely in temperate climates. It resembles tubercle both in staining properties and in culture, and experimental inoculation gives rise to nodular swellings closely simulating those produced by tubercle.

He also isolated another bacillus from grasses, Grass bacillus II. (*Mycobact. graminis*), resembling both the tubercle bacillus and Grass bacillus I., but presenting certain differences. In culture it resembles rather that of avian tuberculosis, and the form of the bacillus in culture was extremely variable. Inoculation of animals led to fatal results, with changes closely resembling those found in experimental tuberculosis.

Möller considers that acid-fast bacilli found in butter and milk are probably simply varieties of the Grass bacillus.

Acid-fast bacilli have also been discovered in sewage.

Other acid-fast bacilli have been described; in fact, American authors lately tabulate nearly thirty of them, and some of them claim one after another of the characteristics formerly accredited solely to the tubercle bacillus, until one alone remains, viz. the tardy development of the tubercle bacillus in culture.

Instead of depending now as formerly on the tinctorial properties, one must now consider the origin of the material examined, and the history of the case from which suspected sputum or other fluid is taken. For example, in most cases of suspected pulmonary tuberculosis in man one can still give help, and sometimes even a definite diagnosis. The difficulty comes in when one has to examine urine, food-stuffs, or material which may contain tubercle bacilli, but the relationship of which to a possible infection from diseased man or animals cannot be proved.

Specific Diagnostic Tests for Tuberculosis.—During 1907 and 1908 several new diagnostic tests for tuberculosis have been introduced, and despite the comparatively short time they have been in use their utility is already very generally admitted. The tests are (1) the oculo-reaction of Calmette; (2) the cutaneous reaction of Pirquet; (3) the subcutaneous reaction; and (4) Moro's salve reaction. The subcutaneous reaction is a localised area of swelling and redness which follows the injection of 1 mgrm. of old tuberculin under the skin of a tuberculous individual; the salve reaction is a dermatitis following inunction with an ointment consisting of 5 c.c. old tuberculin and 5 grams anhydrous lanolin. Neither of these has been employed to any extent in this country, and they need no further mention. Pirquet's test is probably the most delicate; a drop of 25 per cent solution of old tuberculin is placed on the skin, and a punctate scarification of the epidermis is made through it, as in ordinary vaccination; a control vaccination through saline solution is also performed. A positive reaction is shown by the development within six or eight hours of a small papule, which speedily becomes surrounded with a small erythematous zone. If there is any latent or obsolete tuberculous deposit in the body a positive reaction is very probable; hence it is not surprising that from 80 to 90 per cent of clinically healthy adults react. Its very delicacy robs the test of some of its value so far as adults are concerned; it is most reliable as an evidence of clinical tuberculosis in children. The oculo-reaction is performed by instilling one drop of a ·5 per cent solution of tuberculin into the conjunctival sac. In the course of six or eight hours a positive result is shown by the appearance of some mild inflammation of the caruncle, followed in a few hours by more or less general conjunctivitis. The chief drawback to the oculo-reaction is the occasional occurrence of more serious inflammation of the eye, and one or two cases of permanent impairment of vision have been reported. In most of these the eye has been the seat of previous disease, and it is of the greatest importance to assure oneself, before applying the test, that the organ is healthy. It is also advisable to irrigate the conjunctiva with sterile saline solution before instilling the tuberculin. While, therefore, the test is not entirely free from risk, it should be remembered that it has now been used in many thousands of cases, and that the number of accidents is comparatively small. A number of them have been due to the use of too strong a solution of tuberculin; the solution should not be stronger than ·5 to 1 per cent. As a general rule it may be said that a marked, early ocular, or cutaneous reaction suggests a mild infection; a slight, delayed reaction indicates a more severe one. The test is usually negative in tubercular meningitis, general tuberculosis, and advanced cases generally.

Separation and Cultivation of the Tubercle Bacillus.—Koch, after failing with other media, succeeded in cultivating the bacilli on the surface of stiffened blood serum, and it is an evidence of the greatness and completeness of his work that his methods remain still, after twenty years, those most generally used for the separation of the tubercle bacillus and its cultivation outside the body of man.

To obtain cultures, a guinea-pig with early acute experimental tuberculosis is killed, portions of tuberculous tissue or glands are taken with aseptic precautions and implanted on the surface of solidified blood serum. The growth appears in ten to fourteen days as a dry scale, which gradually spreads over the surface, reaching full development in six or seven weeks. Some workers recommend that the piece of tissue should not at once be smeared over the surface of the serum, but kept for two or three weeks at 37°-38° C., and that after this time it should be broken up against the wall of the test-tube, and the juice from it smeared over the surface. In first cultures as a rule the growth is not plentiful, but it becomes more voluminous and luxuriant in subcultures.

Subcultures may be made on blood-serum or on glycerine agar, a medium which, curiously enough, cannot be used for separating the bacillus direct from lesions.

Glycerine beef-broth, glycerinated potato, *i.e.* slices of potato over which 5 per cent solution of glycerine in water has been smeared, and other glycerinated media, have been used for continuing the subcultures.

Hesse (*Zeitsch. f. Hyg. und Infectionskrankh.*, Bd. xxxi.) has recommended a medium containing a soluble albumin preparation known in commerce as "Nährstoff Heyden," made up as follows: Nährstoff Heyden, 5 gm.; common salt, 5 gm.; glycerine, 30 gm.; agar-agar, 10 gm.; normal soda solution, 5 gm.; distilled water, 1000 c.c. This is at once seen to correspond to glycerine agar, with Nahrstoff Heyden substituted for beef-broth. Menzi (*id.*, Bd. xxxix.), Jochmann, and others, have also worked extensively with this medium, and come to the conclusion that it is a very good one for separating the bacillus, particularly from sputum. If tubercle bacilli are plentiful in the sediment from urine, they can also be readily separated. Microscopic growths are obtained in one to three days. It is, however, inferior to glycerine agar as a medium for subculturing the organism.

Kühne, Proskauer, and Beck found that luxuriant growths could be obtained in proteid-free media. The two last have shown that the bacillus will grow in a medium consisting of magnesium citrate, ammonium sulphate, glycerine and mannite, or even in one containing only commercial ammonium carbonate, 0·35 per cent; acid potassium phosphate, 0·15 per cent; magnesium sulphate, 0·25 per cent; and glycerine, 1·5 per cent. The glycerine, however, cannot in any medium be replaced by bodies allied to it chemically.

De Schweinitz and Dorset, from chemical analysis of cultures, drew attention to the large amount of phosphoric acid present in the ash, and to the fact that this was the only acid present. They also stated that an acid reaction of the culture medium appeared to be favourable to the growth of the tubercle bacillus, and subsequently reported that addition of acid phosphate of potassium and the omission of sodium chloride gave a medium upon which development took place very rapidly (cp. Proskauer and Beck's medium). The medium they now used is composed as follows (*American Medicine*, 19th July 1902, p. 93):—"Chopped meat, one part; distilled water, two parts, heated at 45° to 60° C. for three hours, strained, boiled, filtered, and then 1 per cent peptone and ½ per cent acid potassium phosphate added. This solution is neutralised with sodium hydrate, boiled for one hour, 7 per cent glycerine added, and filtered; acidity equal to about 10 c.c. N/10 sodium hydrate required to neutralise 100 c.c. beef-broth, phenolphthalein being used as indicator." They report that this medium is eminently satisfactory, not only in securing a rapidity of growth, but also apparently in keeping up the virulence of the organism.

Chemistry and Chemical Products.—Little need be detailed here of the actual chemical composition of the tubercle bacillus. In addition to the fat, fatty acids, and wax already referred to, it contains a nucleic acid which is credited by Behring with possessing to a high degree the specific properties of Koch's tuberculin. The paper of de Schweinitz and Dorset, just referred to, may be consulted on this point.

It is much more important to consider the chemical substances produced by it to which the pathogenetic actions are due.

The most important of these are tuberculin and other bodies separated from crude tuberculin, some highly toxic, some believed to be directly curative.

Tuberculin was separated by R. Koch. It is prepared by growing the bacilli for six to eight weeks in a slightly alkaline veal-broth containing 1 per cent of peptone and 4 to 5 per cent of glycerine.

The whole is then evaporated to a tenth of its bulk on a water-bath, which kills the bacilli, and this fluid constitutes crude tuberculin. It contains 40 to 50 per cent of glycerine, which prevents the growth of putrefactive organisms.

What would appear to be a specific action is shown by the fact that injection of it produces in tuberculous animals a considerable rise of temperature, whereas after injection into healthy animals the reaction is not so great. It was introduced by Koch as a curative agent, and accepted at the moment by the world as a "gift from the gods." Koch's theory of its action, founded upon experimental results in guinea-pigs, was that the tubercle bacillus secretes a substance having a necrotic action on the tissues, that when tuberculin is injected into a tuberculous patient, the proportion round a tuberculous focus becomes suddenly increased, great reaction takes place with necrosis and separation of the material containing dead and living tubercle bacilli. Virchow showed that from this very process its employment was attended with the greatest danger, as it led in many cases, by the freeing of bacilli formerly imprisoned in fibrous nodules, to dissemination of the disease. It is still used to produce this effect in cases of lupus, but the resulting ulcer takes long to heal up.

Tuberculin is chiefly used now in the diagnosis of the disease in cattle, only a small percentage of cases failing to react, and it is also used, chiefly on the Continent, with great precautions in the diagnosis of early tuberculosis in man. In this country, however, there is still a strong prejudice against its employment in man.

Hunter stated (*Brit. Med. Jour.* 1891) that tuberculin consists chiefly of (1) albumoses, (2)

alkaloidal substances, (3) extractives, mucin, inorganic salts, etc. He prepared two modifications of tuberculin, one of which, containing most of the albumoses, produced less fever than the other.

Whether the albumoses found are the toxic bodies in tuberculin is doubtful. They may simply, according to Kühne, be carriers of the real toxine. This view he corroborated by growing the bacilli in a proteid-free medium, and found that an albuminate produced had the same effect on animals as tuberculin.

Curative.—The opinion gained ground that the substance in tuberculin which causes the reaction resides in the fluid, whereas the supposed minimising and curative agent seems to reside in the bodies of the bacilli themselves. Koch's further researches were directed towards separating these, and attempting to employ the immunising substance in strengthening the tissues to resist the disease. These intracellular toxines seem to be very intimately bound up with the protoplasm, and to separate them he used the following procedures:—Young virulent cultures were dried *in vacuo*, and thoroughly rubbed up in an agate mortar, treated with sterilised distilled water, and centrifugalised for a long time. The clear fluid resulting was decanted, and formed Koch's "Tuberculin O." This was found to produce a reaction like the original tuberculin.

The deposit remaining was again dried and treated in a similar way, and the clear fluid decanted, the process being repeated until no deposit remained. All the fluids were then put together and formed Koch's "Tuberculin R" (T.R.) He stated that this contains only the substances present in the bacilli which are insoluble in glycerine. When this is injected into animals in small, repeated, and gradually increasing doses, he claims that it produces immunity against the original tuberculin, against Tuberculin O, and against living and virulent bacilli. He and others have applied it in man, and though they claim to have obtained success in treatment of tuberculosis in man, a great deal of healthy scepticism prevails, particularly in this country. Siill the hope is gaining ground that in this direction a cure for tuberculosis may yet be obtained.

Working on similar lines, Klebs has introduced two extracts — *tuberculocidin* and *antiphthisin*. The former is made from tuberculin by removing so far as possible the substances which cause reaction. The latter is made from the fluid culture without the use of boiling heat to concentrate. This procedure, it is thought, preserves the immunising agent.

Von Ruck has also introduced two preparations.

The first, *tuberculinum purificatum*, is made from the culture fluid with the bacilli, by extraction *in vacuo* at 120°-130° F. for six weeks to two months, the object being to extract more of the bacterial proteids, and so to contain more of the immunising principles Koch found in Tuberculin R.

The second is a watery extract made at 120°-130° F. of dry tubercle bacilli freed from fat by action of sulphuric ether. Denison gives lengthy statistics which would go to prove that in therapeutic action these excel all other preparations.

Tuberculin as a Diagnostic Agent in Man.—Koch in 1890 expressed himself as follows:—"I believe I do not go too far when I assume that the medicament will in future be an indispensable aid to diagnosis. It will enable one to diagnose doubtful cases of incipient phthisis even when one fails to obtain certain knowledge of the nature of the disease by finding bacilli or elastic fibres in the sputum, or by physical examination."

At the London Congress on Tuberculosis, 1901, he detailed his method of using tuberculin and his results so far.

After ascertaining that the temperature is not above normal for one, or better, two days, he gives an injection of one-tenth to one milligram under the skin of the back in the forenoon, the amount given depending on the strength of the patient. If no reaction takes place double the dose is given on the third day, *i.e.* one full day intervening. If there is a slight rise of temperature—only a quarter of a degree centigrade, for example—the dose is not increased, but repeated as soon as the temperature has gone down to normal. It often happens that, though the same dose has been given, the second reaction is stronger than the first. This is quite specially characteristic, and may be regarded as an infallible sign of the presence of tuberculosis.

If the first small doses produce no reaction, he gives five, and, finally, even ten milligrams. He is accustomed to give this last quantity twice, and only when no reaction occurs under these conditions does he feel justified in assuming that the case is not one of fresh or progressive tuberculosis demanding specific treatment. By the end of 1900 he had personally observed 3000 cases. He expressly stated that he had in no case seen any untoward effect when the tuberculin was used in this way.

His paper was followed by a lively discussion. In general the use of tuberculin as a diagnostic agent was supported by all the continental and American members of congress. While some British physicians, *e.g.* M'Call Anderson and Eric France, spoke strongly in favour of it, others, notably Dr. C. Theodore Williams, condemned it. He thought that in initial cases without fever its employment is not advantageous, and considered it unreasonable, after all our efforts to build up the tissue and textures of the human body, to use a means for breaking

them down. Dr. Edward Squire thought that the use of tuberculin even for diagnostic purposes was unjustifiable in tuberculosis of the lungs, though it was undoubtedly useful in surface or surgical tuberculosis.

The result of the discussion seemed to be the establishment of Koch's views, but nevertheless the agent is still far too dangerous to be employed as a routine means of diagnosis by practitioners.

Agglutination of the Tubercle Bacillus.—Koch has lately published a paper in the *Deutsche medicinische Wochenschrift*, November 28, 1901, in which he details the employment of this property of the bacillus in diagnosis. Arloing and Courmont, by using cultures on potato, in which the bacilli are fairly well separated from each other, obtained positive results as regards agglutination with the blood serum or serous exudation from tuberculous men and animals. Koch also obtained the reaction with ordinary cultures by special methods, but as it is obtained in other diseases as well, he concludes that a marked distinction cannot be drawn between the cases. He found, however, that by employment of small doses of tuberculin he could raise the agglutinating power, and concluded that this corresponds with the formation of an antitoxin, from the fact that the general condition of the patient showed improvement from the time that the agglutinating property appeared in the serum. He suggests this as a method of treatment in cases where no progress is being made under other methods. Others have also suggested the use of tuberculin therapeutically in alliance with other procedures, *e.g.* sanatorium treatment.

Morbid Anatomy.—*Lesions produced by the Tubercle Bacillus.*—In order to understand the variation in the character of the lesions seen in man, we must premise variations both in the infective agent and in the soil on which it is implanted.

Thus the bacilli may vary in virulence and the individual may vary in power of resistance.

Virulent bacilli may be expected to produce actively developing lesions, tending early to caseate and break down. Bacilli of slight virulence will rather produce chronic changes with tendency to formation of fibrous tissue enclosing the bacilli, and there is less liability to caseate and break down.

In children, in young adults, and in weaklings the active form is more often met with. As age advances resistance becomes greater, and the lesions partake more of a chronic character. Hence, one must not expect—all things being apparently equal so far as the infective agent is concerned—to find exactly similar changes in all subjects.

Relation of the Bacilli to the Cells.—In man the bacilli, as a rule, may be said to be extracellular, *i.e.* except as regards the giant cells. Sims Woodhead points out, however, that in certain cases where recovery appears to be going on, bacilli may be found in epithelioid cells. He states that in animals the bacilli are found comparatively frequently in cells, particularly in the epithelial cells of the acini and small milk ducts of the tuberculous udders of cows. Benda has also noticed these appearances in the cells lining canals in other organs as well as in the lacteal ducts of udders, and looks on them as evidence of the spread of tuberculosis along the epithelial canals, a mode of spread which he considers very common, if not the most common.

Sims Woodhead further notes the interesting point that this accumulation of bacilli in the cells, particularly in those of the cat, has a peculiar significance from the fact that the tissues of that animal are exceedingly resistant to the attacks of tubercle bacilli, and only when this resistance is overcome by the invasion of large numbers of bacilli, does the animal succumb to the disease.

The "Grey" Tubercle.—The histological basis of all tuberculous lesions is the "grey" or "miliary" tubercle. This consists essentially of a cluster of large oval cells each of which possesses a large and distinct nucleus and a considerable amount of protoplasm.

These cells—termed endothelioid or epithelioid—are derived by proliferation of the connective tissue cells of the part infected, and also by proliferation of endothelial cells of lymph channels, or capillaries near. Metschnikoff considers that they represent a form of white blood corpuscle, amœboid, and phagocytic, which have been attracted to the spot in virtue of positive chemotaxis, and are engaged in attacking the invading organisms. These epithelioid cells lie in the meshes of a stroma derived from the original connective tissue of the part. As one passes to the periphery of the grey tubercle, smaller cells—lymphocytes—are seen scattered among the endothelioid cells, and they become more numerous until at the edge of the nodule they form a more or less dense layer surrounding it. Frequently fibrin is found amongst the cells, particularly in lower animals.

These tubercles become non-vascular, and are then nourished by the fluids which transude from the blood-vessels, the vessels which formerly penetrated the area becoming blocked by endothelial proliferation or by thrombosis.

Such is the appearance of the rapidly forming acute miliary tubercle.

When development is less rapid, and also—according to Hamilton — when infection has taken place by way of lymphatics, *giant cells* are commonly seen.

These usually develop towards the centre of the tubercle, and surrounding them are arranged the endothelioid cells and lymphocytes. Some look on giant cells as evidence of degeneration,

but many pathologists consider that they are active products of tissue reaction, and have defensive and perhaps reparative functions.

The giant cell of tubercle is large, rounded, or oval, with a crenated edge, from the prominences of which processes pass to join the supporting network or reticulum of the tubercle. It is multinucleated. The nuclei are flattened, with their long axes arranged radially, and are placed near the periphery of the cell, or clustered at one pole, this appearance to some extent depending on the plane of section. Occasionally the nuclei lie irregularly in the cell—for example, in small young giant cells. If bacilli are present they may be demonstrated near and between the nuclei.

The protoplasm takes ground stains better at the periphery than in the centre, indicating the presence of some necrotic action at the centre.

Origin of Giant Cells. — Giant cells may develop in several ways. 1. By excessive proliferation of a single endothelioid cell; the stimulus being so strong that the nuclei divide far more rapidly than the protoplasm, and tend to gravitate to the surface of the cell, seeking nutrition. This is the most generally accepted explanation.

2. By coalescence of a number of endothelioid cells, those in the centre becoming necrotic.

Occasionally appearances are met which support this explanation, and there seems to be no reason why it should not hold good in some instances.

3. The appearance is due to the proliferation of the endothelial cells of a blocked capillary, belonging either to the lymphatic or blood vascular system.

4. Gaule and J. Arnold have shown that in tuberculous disease of organs containing epithelium, the epithelial cells begin to proliferate and may yield epithelioid and giant cells (Thoma).

The striking appearance of the grey tubercle containing the giant cell has led to this being looked on as characterising tuberculous formations, but as similar giant cells are met with in other chronic inflammatory changes, they must not be looked on as absolutely essential components of the miliary tubercle. Even when the various components of the tubercle are arranged in the characteristic manner described, one can look upon this picture as sufficient to stamp the disease as tubercle only, if, in addition, the presence of the typical tubercle bacillus is assured.

For example, experimental inoculation of "butter" bacilli in guinea-pigs produces similar morbid processes, differing only in the presence of polymorphonuclear leucocytes and fibrin, and the absence of giant cells, though lymphocytes and endothelial cells abound. Moeller's bacilli also produce a generalised condition in guinea-pigs, macroscopically and microscopically resembling ordinary tuberculosis.

Yellow or Caseating Tubercle. — Round the original "grey" tubercle others soon form, and all of these being non-vascular the central part of the enlarged nodule tends to undergo the peculiar necrotic change — caseation — so commonly met with in tuberculosis. In "caseation," or "cheesy transformation," death of the cells takes place comparatively slowly, so that in addition to coagulative necrosis there is time for some fatty degeneration to occur. This change converts the central part of the tubercle into a structureless, homogeneous, or finely granular material, in which none of the individual components can be distinguished. The cell breaks down, though particles of nuclear chromatin may long persist and still take hæmatein or anilin stains. This is the yellow or caseating tubercle.

This morbid change may extend by formation of grey tubercles around until a large area of the tissue or organ becomes caseous. The caseous part tends usually to undergo liquefaction, producing a tuberculous "cold" abscess, or, in an organ such as the lung, bringing about excavation, the central softened material escaping by way of a bronchus.

It is believed (Prudden) that liquefaction presupposes a "mixed infection," the chemical products of the tubercle bacillus being able to cause caseation, whereas other organisms, such as micrococcus tetragenus, streptococcus, etc., are required to bring about the further change.

The destructive process does not by any means always take place. Frequently the diseased area becomes enclosed by formation of fibrous tissue walling it in, rendering it quiescent and innocuous. Under favourable circumstances the caseous area becomes dried up, and forms a soft whitish mass in the midst of pigmented scar tissue, or calcareous deposit may take place in it. In these collections fragments of tubercle bacilli may, with some difficulty, be demonstrated. In experimental tuberculosis in refractory animals the bacilli may become enclosed in concentric layers of a homogeneous substance in which calcareous deposit occurs. This drying up with fibrosis or calcification is nature's method of cure, and is frequently referred to as evidence of the curability of tuberculosis scars containing evidence of old tuberculosis being met with in from 20 to 50 per cent of all persons dying from other diseases.

Chronic Tuberculosis. — Acute lesions may become chronic from encapsulation by fibrous tissue, or lesions may be chronic from the outset. In cases where the bacilli are of moderate virulence, or where the individual attacked has great power of resistance, infection results in formation of connective tissue rather than in caseation. Giant cells are usually found, it being permissible to regard these as indicative

of a degree of chronicity in tuberculosis as in other diseases, such as syphilis. Such changes develop very slowly, and after a time the central part may undergo very slow caseation.

Lesions of the various Organs and Tissues. Tuberculosis of the Lungs

Acute Miliary Tuberculosis.—This is the result usually of the breaking down of a primary focus in the lung, or in the bronchial or root glands, and escape of infective material containing virulent tubercle bacilli into the blood stream, in which they may be detected during life. The immediate result is that bacilli are fairly uniformly scattered through the lung, and produce grey tubercles in the walls of the vesicles. On section these are seen to have no special relation to bronchi or interlobular septa, but are scattered over the whole of the cut surface, and appear as well under the pleura. Frequently they tend to be aggregated at the apex. As a rule the patient dies before the tubercles can break down. In most cases the tuberculosis is generalised throughout the body, particularly in children, and the membranes of the brain, liver, spleen, and, to a less extent, the kidneys, contain numerous grey nodules. The pericardium and heart are rarely affected. Microscopically the tubercles show relationship to the circulation by their connection with blood-vessels, and in small vessels may form a projection in the intima. The latter appearance is not uncommon. Acute miliary tuberculosis can only arise when a tubercle involves and breaks through the wall of a still previous vessel of some size. In about half of the cases the pulmonary vein is involved (Coats).

Lymphatic Miliary Tubercle.—The bacilli may also spread from a primary focus by way of lymphatics; and the nodules are then seen in clusters in the adventitia of the bronchi and vessels, and in lines along the septa. They are not so uniformly scattered as in the case of the vascular tuberculosis, and are very frequently localised in the neighbourhood of the primary focus. For example, a band of tubercles may extend into the lung from a tuberculous gland, or if the primary focus is in the lung they extend from that focus towards the root. Moreover, the tuberculosis is not generalised throughout the body. Bearing in mind these characters, it is not extremely difficult to distinguish between these two forms of miliary tuberculosis of the lungs.

Hamilton considers that the lymphatic tubercles invariably possess giant cells, and that these characterise it, but, on the contrary, their absence is by no means characteristic of vascular miliary tubercle.

Broncho-Pneumonic Tuberculosis.—Tuberculosis most frequently attacks the lungs by way of the air passages. Out of 257 cases of pulmonary tuberculosis in children investigated by the writer, 173 showed the broncho-pneumonic type, and in 26 others this type was combined with miliary tubercle, probably chiefly lymphatic in spread.

The condition commences as an inflammation of the smaller bronchi—bronchiolitis—which assumes a tuberculous character, or is of that nature from the outset. It may also be secondary to a lymphatic invasion of its walls, this producing a lesion which gradually spreads towards the lumen, causing a tuberculous bronchitis, or it may arise from the bursting of a caseous focus into the lumen of the bronchus.

In this form of the disease one must remember certain factors which have an important bearing on its origin and progress, in children particularly.

1. The smaller bronchioles possess no cartilage to strengthen their walls. They do possess, however, a complete encircling band of unstriped muscle, which is of great assistance to the short ciliated columnar epithelium lining their lumina in keeping them free and open. In inflammation this muscularis mucosæ becomes infiltrated with fluid and leucocytes, weakening it and interfering with its contractility.

2. Following certain diseases of childhood, *e.g.* measles and whooping-cough, bronchitis and other catarrhal conditions are apt to occur, and these may be looked upon as not improbably predisposing causes.

3. In bronchitis the epithelium tends to become detached in large pieces; and

4. As a result of paralysis of the muscularis referred to above, the cast-off cells and exudation remain *in situ*. When the condition spreads to the infundibular passages, as they do not possess ciliated epithelium or muscularis mucosa, the exudation cannot be expelled.

5. If this bronchiolitis be not, as it probably usually is, primarily tuberculous, we have, consequently, a suitable nidus for the implantation and development of the tubercle bacillus. Soon the manifestations dependent on its action appear. Caseation commences in the plug, and spreads to the walls of the bronchus. Meanwhile a marked inflammatory reaction is spreading from the wall of the bronchiole out along the septa between the vesicles and in the vesicles themselves. In the former proliferation of the cells and of the endothelium of the capillaries occurs, producing great thickening of the septa. The vesicles become packed with large catarrhal cells and leucocytes, with a varying amount of fibrin. As the condition progresses a coherent mass tends to form, attached to the wall of the vesicle nearest the centre of the nodule. As the walls are gradually becoming non-vascular the process of caseation gradually spreads outwards until a homogeneous, firm nodule has formed, in which, microscopically, the outline of bronchus and vesicles can with difficulty be made out.

The acute broncho-pneumonic nodules may possess no giant cells, but the later process spreading along the lymphatics (Hamilton) shows a giant-celled lesion.

In older children and in adults this caseated area tends to break down to form a cavity containing tuberculous pus and necrotic tissue, which becomes evacuated through the bronchus.

In *children* broncho-pneumonic nodules, all of recent date and varying in size up to a diameter of a quarter of an inch, may be scattered throughout the whole organ, the lung between being congested and vesicular. These nodules can readily be felt on passing the hand into the pleural cavity on opening the thorax. Those near the surface are roughly pyramidal in shape, with their base to the pleural surface. The nodules may be more numerous near the apex, but softening and excavation is not so commonly met with as in the adult.

Occasionally in children large areas of a lobe or the whole of a lobe become consolidated and caseous, presenting a more or less uniform greyish or yellowish colour. The term *caseous pneumonia* is sometimes applied to this condition.

In the *adult* the characters are different. The disease usually at first attacks the lung a short distance below the apex. This localisation near the apex is probably due to that being the least expansive part of the lung. The air stagnates, and tubercle bacilli are more likely to grow and develop in such circumstances (Coats). From the apex it extends downwards through the lung, so that the oldest lesions are to be found near the apex or in the lower lobes near their upper part, and the more recent farther down; the extreme base may be vesicular and healthy to the unaided eye.

The process, moreover, goes farther, so that softening of the caseated areas occurs, and the pus is discharged through the bronchi, forming the expectoration of pulmonary tuberculosis. This process results in "excavation" or "cavitation." The softened material tends to become disseminated throughout the bronchial tree, giving rise to fresh broncho-pneumonic nodules in the same lung; or later, may in a similar manner cause infection of the sound lung. In addition the process sets free bacilli, which spread along the lymphatics, so that lymphatic miliary tubercles frequently coexist along with the broncho-pneumonic. These can usually be distinguished by careful inspection.

Once more, lymphatic spread may predominate, but in such a case the primary lesion is usually less acute.

Briefly, the "picture" of a spreading broncho-pneumonic tuberculosis—the common pulmonary phthisis—is as follows:—

Extreme base — congested, vesicular, free from tubercles, or with a few small irregular clusters of grey tubercles, either early broncho-pneumonic or lymphatic.

Above this these have coalesced to form greyish masses, which in their centres are practically non-vascular.

Still higher they have become uniform, firm, non-vascular, yellow and caseating in the centre, grey at the periphery.

Higher still softening is beginning in the centre.

At the apex one or more recent cavities of varying size are seen, their walls irregular, rough, and lined with necrotic caseous material, the cavity communicating with a bronchus, the surface of which may be—usually is—congested and ulcerated. If the cavity be large ridges are seen on the walls, or bridges cross it, representing the more resistant septa, bronchi, or blood-vessels.

If the process take a favourable course a layer of vascular granulation tissue forms on the wall. The caseous material becomes cast off, and the irregularities smoothed down.

Later still the granulation tissue becomes fully developed to form fibrous tissue, and we now find a healed cavity with smooth, thin, fibrous walls. In course of time this tends to contract, and the walls to become dense and pigmented.

The *expectoration* in the early stages is composed of mucus with more or less numerous leucocytes, and large catarrhal epithelial cells with one or more nuclei. Later, these undergo fatty degeneration, and the sputum also contains elastic tissue. Bacilli are present in all stages.

Pleurisy.—Reaction also takes place in most cases in the *pleura* over extensively diseased areas of the lungs, and is usually simply an extension of the disease. The pleurisy may be fibrinous, sero-fibrinous, purulent, or hæmorrhagic. It may show grey tubercles or caseous points, most easily detected on the inter-lobar or diaphragmatic pleura, or these may not be evident to the naked eye, though demonstrable under the microscope. In chronic stages the pleura becomes greatly thickened and its layers adherent. It may occur without tuberculosis of the lung, spreading from the peritoneum or from a caseous mediastinal gland. Tuberculosis, in man, is rarely primary in the pleura.

Chronic Pulmonary Tuberculosis. — The acute forms may become chronic, or the changes may be chronic from the outset. In the latter case the bacilli are less virulent, the dose is smaller, or perhaps, most important of all, the individual possesses greater resisting power.

The tuberculous process is always tending to heal by encapsulation with fibrous tissue. The outer layers of the tubercle become progressively fibrous, and the process consequently becomes quiescent. No special description of chronic phthisis or "fibroid phthisis" is necessary. The process is essentially the same as that described in the acute disease, but it is

characterised by advance by way of the lymphatics into the septa, rather than by invasion of the vesicles. The cases are long protracted, and the process of the disease is slow. In the lung there is abundant cicatrisation, so that the fibrous or fibroid changes are very evident, and may result in cure. The part of the lung affected is very firm, dense, pigmented. It usually contains small cavities with purulent contents, or at the centre of the mass a dry cheesy focus may be present. The interstitial tissue at the margins becomes greatly thickened and the vesicles contracted; their endothelium becomes cubical, so that in a well-marked case they present the appearance of narrow gland tubules. Giant cells are always to be seen at one part or other.

Round the fibroid contracted patches emphysema usually develops, and by the retraction of the thickened fibrous septa attached to the walls of bronchi these undergo irregular dilatation—bronchiectasis.

Larynx, Trachea, and Bronchi. — Tuberculosis in larynx and trachea is rarely primary. It usually follows a destructive tuberculosis of the lungs, the sputum passing over the surface being the infective agent. In the case of the smaller bronchi the tubercles may extend along the wall in the lymphatics, develop into the yellow caseous tubercles, and break down into the lumen of the bronchus.

Wherever occurring, the final result is ulceration. The single ulcer is more or less rounded, and sometimes shows distinct tubercles at its edge. In the trachea the early ulcers may coalesce so as to produce a peculiar superficial worm-eaten appearance, which is somewhat characteristic. This appearance is not constant, and the ulcers may remain few in number, tending to extend at their edges, which are thickened, and also deeply into the wall, the general characters resembling those of tuberculous ulcers elsewhere.

In connection with the larynx tuberculosis usually begins below the epiglottis, differing from syphilitic ulceration, which usually starts on the upper surface or edges of the epiglottis and works downwards into the larynx.

The most common situation of tuberculous ulcers of the larynx is at the posterior ends of the vocal cords, when they extend deeply at the bases of the arytenoid cartilages, their depth being great in proportion to their superficial area. The cartilages may be separated and destroyed. From this situation the ulceration may extend along the aryteno-epiglottic folds, or forwards and downwards in the larynx and trachea.

Heart and Pericardium. — The heart is rarely affected by tuberculosis.

It is most commonly met with in the form of caseous nodules in the heart muscle, the septum ventriculorum being frequently selected, or the outer wall of the ventricles. It accompanies tuberculosis elsewhere.

Tuberculosis of the endocardium is sometimes seen affecting the mural endocardium, but more frequently as a valvular condition with the usual features of an acute endocarditis. It is rare, however, judging from the experience of Edinburgh pathologists.

Tuberculosis of the pericardium is more common, but not so frequently met with in this country as American and German statistics would lead us to expect. Acute miliary tuberculosis may accompany a generalised tuberculosis, occurring in 7 cases out of 355 cases of tuberculosis examined in the Royal Hospital for Sick Children in Edinburgh. It may also extend from a softened mediastinal gland, or a tuberculous pleurisy or peritonitis. In older subjects it is characterised by great general or nodular thickening, by abundant fibrinous or hæmorrhagic exudation, beneath which small tubercles are seen.

It is remarkable, nevertheless, how often the pericardium escapes, even though surrounded by tuberculous disease of lungs, pleura, peritoneum, or glands.

When it does occur, and the patient survives long enough, the pericardium becomes greatly thickened and frequently adherent, encasing the heart in a dense thick fibrous capsule, in the deep layers of which caseous tubercles are evident.

Lymphatic Glands.—These glands are affected very commonly following the lungs as favourite points of attack by the tubercle bacillus.

Frequently no other lesion can be made out, and softened tuberculous glands frequently become the starting-point of a generalised tuberculosis.

The changes macroscopically and microscopically are similar to those seen elsewhere. The bacilli are caught in the sinuses, or brought thither enclosed in leucocytes. If active they set up reaction, producing the grey tubercle, so that the enlarged gland may show small grey points. These must be distinguished from the uniformly mottled appearance obtained in other conditions.

Part or the whole of the gland caseates and may break down, discharging its virulent contents on an epithelial or endothelial surface. The condition may retrogress at any stage, and heal by fibrosis, or if caseation has occurred the dead tissue may become encapsuled and invaded by fibrous tissue, and it may dry up and become the seat of calcareous deposit.

Tuberculosis of lymphatic glands is of supreme importance, from the point of view of origin and spread of the disease.

It may now be granted that the bacilli frequently enter the system by way of the tonsils and pharyngeal tonsillar structures, being carried inwards by leucocytes. They are fre-

quently destroyed, but if the resistance is feeble they pass onwards and are caught in the glands. Similarly they may enter through decayed teeth, or through a moist catarrhal skin surface, and in none of these cases may there be any evident surface manifestation of tuberculosis.

They extend along the cervical chain of glands, sometimes missing one or a series, as they appear to be able to "jump" a gland, its sinuses probably being sufficiently wide to allow of their passage.

If the condition is not cured by surgical interference it may still extend downwards, and a caseous tuberculosis result in the bronchial and mediastinal glands from which the lungs may become affected, and an apparently primary respiratory tuberculosis be really the result of a tonsillar absorption from food. Benda and others disbelieve in tonsillar or cutaneous infections.

Bronchial and Root Glands.—In children the writer found that out of 183 cases where these glands were affected, in 123 the glands on the right side were more extensively affected than those on the left. Similar observations have been made independently by Dr. Batten (*Barth. Hosp. Rep.*, vol. xxxi.).

Again, a tuberculosis of bronchial glands may extend by way of anterior or posterior mediastinal glands—the latter more commonly—to the cœliac glands, and thence to the retroperitoneal and mesenteric glands. It may also progress in the opposite direction from mesenteric glands to bronchial.

Mesenteric Glands.—These are very commonly affected in children—far more commonly in this country than shown by any post-mortem statistics, because frequently a cure is brought about by suitable treatment, or even without treatment. Wherefore care must be taken not to base our ideas of the prevalence of abdominal tuberculosis upon post-mortem findings only.

The mesenteric glands are frequently found affected alone in children. In such a case the bacilli are supposed to be few in number, and insufficient to cause a local lesion in the intestine. They are supposed to be carried by leucocytes through Peyer's patches, and become blocked in the gland. As successive arrivals take place, by summation of their effect they are at last able to cause caseation.

If the bacilli are in greater numbers in the intestine they develop in and cause primarily an ulceration of Peyer's patches, then secondarily tuberculosis of the glands in connection with the affected patches.

While these lesions may occur from a primary ingestion tuberculosis, they more frequently follow destructive changes in the lungs, from which virulent sputum is swallowed.

Glands in connection with the extremities are seldom affected in man.

Alimentary Tract.—The lower part of the pharynx, the œsophagus, stomach, duodenum, and jejunum enjoy a comparative immunity from tuberculosis, and are usually affected only in advanced cases. There are many theories to account for this, such as the rapid passage of the contents of these parts, and the acidity of the gastric juice. In addition to these factors, however, one may suppose that the antiseptic power of the hydrochloric acid of the gastric juice and of the bile may have some effect in protecting the upper part of the tract, these influences being lost as the ileum is reached. Again, though decomposition of contents takes place normally in the large intestine only, in the presence of the lowered vitality of susceptible cases this may occur higher up in the ileum, and pave the way for infection by the tubercle bacillus. In addition the lymphoid Peyer's patches, like the similar structures in the pharynx—the tonsils and pharyngeal tonsils—tend to attract or separate out the bacilli.

The principal lesion found is the ulcer of the Peyer's patch, *i.e.* in the lower part of the ileum, and chiefly in the lowest Peyer's patches.

Here, as in other tissues, the grey tubercle is the first manifestation of infection. Several develop in a Peyer's patch, grow, and coalesce. The resulting ulcer is necessarily irregular and its outline is sinuous. The whole Peyer's patch need not be affected. Spread is chiefly by the lymphatic vessels, and as these on the whole take the nearest way to their glands, *i.e.* round the lumen of the bowel towards the mesentery, the long axis of the ulcer tends to be transverse. In advanced cases the ulcer may encircle the bowel.

Seen from the peritoneal aspect the ulcers are recognised as dark areas, mottled with minute whitish spots or streaked with whitish lines corresponding to the tubercles which are spreading in the subserous lymphatics.

On opening the bowel the ulcer is seen to have raised, irregular edges, the thickening gradually lessening towards the mucous membrane. The base is rough, showing caseous points, and in the ulcer and its edges tubercles may be detected.

Microscopically, all around the ulcer tubercles are seen in the submucous, muscular and subserous coats. The last-named is practically always thickened, thus guarding against perforation.

In advanced cases the mesentary is thickened and shortened, and the glands contained are enlarged and caseous.

Frequently the surfaces of peritoneum become adherent, and the whole contents of the abdomen matted together. In the adhesions, caseous foci and glands are readily made out.

Occasionally only the lowest Peyer's patch and a line of glands leading upwards from the region of the ileo-cæcal valve are affected. In all cases the disease is commonly most advanced

in this situation, and by fibrosis may lead to stricture of the bowel.

In adults, tuberculous strictures occur rarely in the small intestine, resembling closely, to the unaided eye, malignant growths.

The large intestine may show ulceration similar to that found in the ileum, but it may escape even in cases where the ileum is extensively diseased.

Fistula in ano is sometimes tuberculous in nature, and if so, resists attempts at cure.

Liver.—Tuberculosis of the liver is always secondary, and may be miliary or caseous, in both cases being usually accompanied by a generalised tuberculosis.

Small grey tubercles are most numerous immediately underneath the capsule; but though they are apparently not so numerous, to the naked eye, on the cut surface, when sections are examined microscopically, minute tubercles may be found in enormous numbers, in all stages of development. The liver presents one of the best opportunities for examining the microscopic structure of early tubercles. They are best seen near the portal spaces, apparently developing in connection with these. Some observers are of opinion that the epithelioid cells of the grey tubercle may develop from the liver cells themselves. Benda considers that spread occurs chiefly along the bile-ducts.

The yellow tubercle breaks down early, and whether caseous or forming an abscess, is always bile-stained like any other necrotic area in the liver.

Spleen.—Acute miliary tubercle and caseous tubercle are met with.

In the former case, occurring along with generalised miliary tuberculosis, the tubercles, which usually occur in the neighbourhood of arteries, may be distinguished from enlarged Malpighian bodies, which have a similar seat, by their projection on the freshly cut surface, the retraction of the splenic pulp pushing them upwards.

In caseous or chronic tubercle the cut surface is covered with large yellowish masses which may be softening at their centres. They are prominent, the splenic tissue retracting on section, and have to be distinguished from the nodules of lymphadenoma. This form occurs mostly in children in whom the abdominal glands are tuberculous (Coats).

Supra-Renals.—In Addison's disease the prevailing anatomical feature is enlargement and tuberculous caseation of the supra-renals.

Genito-Urinary Tuberculosis is usually associated with disease elsewhere.

Chronic tuberculosis of the *kidneys* is more common in males than females, and this fact has been taken by some observers (Weigert, Coats) to indicate that in most cases it originates in the testicle, or rather epididymis, and spreads thence upwards. Supposing that this were the case, one would expect that simultaneous affection of the kidneys would be common, whereas the disease is usually more advanced in one kidney, or may be confined to one.

Benda states that the disease starts in the duct-like structures in the kidney—the collecting tubules—and he has demonstrated bacilli in the cells of these parts.

Most pathologists consider that the bacilli are conducted to the kidneys in the blood stream, and that grey tubercles and then yellow caseous tubercles are formed in the usual way. In acute generalised miliary tuberculosis the kidney is usually less affected—to the naked eye—than spleen or liver.

In advanced cases, whichever be the manner in which the infective agent is brought, the result is the same. All stages of tuberculosis are seen, from the grey miliary tubercles to the softened caseous tubercle, discharging into the pelvis of the kidney. The process may go on until the whole kidney is converted into a multilocular cyst, lined by caseous material, the fibrous cyst wall showing little kidney substance. This condition is known as chronic tuberculosis of the kidney, renal phthisis, or scrofulous kidney. It is rare to find the perinephric tissue affected.

From the kidney substance the condition spreads by direct continuity to the pelvis and ureter. The accumulated caseous products may block the ureter at its lower end, so that the renal pelvis with the cavities opening into it and the ureter become distended—hydronephrosis being superadded.

The wall of the ureter becomes greatly thickened, and lined by caseous material.

When the disease reaches the urinary bladder its spreads over the mucous membrane, and in time may reach the other ureter and extend up it by way of the lymphatics, contrary to peristalsis and current.

The mucous membrane of the pelvis of this kidney is first affected, and the disease possibly spreads up the collecting tubules, gradually extending outwards. One kidney with its ureter may, however, remain for long free from disease, and carries on the functions of both.

Extension may continue downwards, so that tubercles are found along the urethra, and even in the glans the writer has found early tubercles beneath the mucous membrane. It attacks prostate, producing caseous tubercles, extends to the seminal vesicles and vasa deferentia, finally producing tuberculosis of the epididymis and testis.

Although Benda and his co-workers look upon primary tuberculosis of the epididymis as extremely rare, there seems no doubt that the disease may originate here, the bacilli being carried thither in the blood stream, and it may also commence in the vesiculæ seminales. From these situations it spreads upwards to bladder,

ureters, and kidneys, in this case affecting the kidneys simultaneously or nearly so.

As a result of the destructive ulceration bacilli appear in the urine. They are usually shorter and thicker than the common varieties, and tend to lie in clumps. They usually stain uniformly.

Epididymis and Testis.—The disease in the majority of instances begins in the epididymis, and it usually becomes extensively caseous and softened before the testis is attacked, the tubercles in the latter radiating inwards from the mediastinum testis. In most cases inflammation of the tunica vaginalis coexists. Rarely calcareous masses are met with in the epididymis which appear to represent old healed tuberculous foci.

Uterus and Appendages.—Tuberculous endometritis is rare. It follows advanced tuberculosis elsewhere. In this condition the lining membrane is pale, thickened, and succulent, showing microscopically the usual evidences of the nature of the disease.

Tuberculous salpingitis is less rare, and tubercles may spread through the wall to the serous surface.

Tuberculosis of the ovary in some cases affords the only evident origin of a tuberculous peritonitis.

Tuberculous Peritonitis. — Acute miliary tuberculosis of the peritoneum may be localised to the bases of tuberculous ulcers of the intestine, or may be generalised throughout the whole peritoneum.

Generalisation seldom takes place by perforation of an ulcer, but usually from softened caseous lymphatic glands, more especially those of the mesentery. Occasionally it follows disease of the vertebræ, and in some cases it extends from the vasa deferentia, vesiculæ seminales, or ovary, in these cases being usually localised.

Sometimes chronic fibroid tubercles occur over the whole or part of the surface as glistening, flattened patches, which may be one-tenth of an inch or more in diameter. These may be the only visible manifestation of tuberculosis in the body, as in one case at least examined by the writer in which the tuberculosis was alimentary in origin, and the condition is possibly analogous to the "Perlsucht" or "duckweed" type found in cattle.

The more acute form may result in an acute peritonitis with fibrinous exudation. There is considerable, sometimes great swelling of the abdomen. The inflammation may even become suppurative in character. More often, however, by the time the case comes to be examined post-mortem, the acute stage has passed off and evidence of chronic inflammation is found in the form of thickening of the peritoneum and generalised adhesions with matting of all the contents of the abdomen.

In these adhesions numerous caseous glands and areas are to be seen, and sometimes tuberculous abscesses in connection with ulcerations of the intestine.

The omentum is greatly thickened and shortened, forming a thick mass which can be felt from the surface of the abdomen. In it numerous tuberculous masses are to be seen.

Tuberculosis of Bones and Joints

Bone.—Tuberculosis is the most common disease of bone, attacking by preference the young. In a large proportion of cases the joints are affected concurrently. The disease may be primary in the bone or in the synovial membrane, probably more frequently in the former.

Tuberculosis appears to be especially prone to attack young growing bone, an injury frequently predisposing. This has been proved experimentally. Schüller found that after injecting living tubercle bacilli into the blood he could induce tuberculosis of bone by inflicting injuries which would otherwise have been recovered from.

The disease begins under the periosteum; in the medulla near the epiphysial cartilage; in the epiphysis, or spreads to the epiphysis from the synovial membrane. The bacilli reach the parts nearly always by way of blood-vessels, seldom through the nutrient artery, more commonly through the articular arteries, whence it happens that the epiphyses are more frequently affected than the shaft. Additional evidence of this is shown by the frequent concurrence of synovial and osseous tuberculosis of joints, the same blood-vessels supplying both these parts.

The disease attacks spongy bone in preference to compact bone; thus the vertebral bodies, or the bones of the tarsus or carpus, and the articular ends of long bones, are more often affected than the shafts of long bones. In the case of the vertebræ, tuberculosis frequently affects simultaneously the adjacent surfaces of two bodies. In the phalanges it may affect the whole shaft (Strumous Dactylitis).

In tuberculosis of bone the microscopic appearances resemble those met with in the disease elsewhere. Primarily the soft medulla is affected, cellular grey tubercles forming in connection with the arterioles. These extend in the usual manner, and the central part caseates. The bony trabeculæ become absorbed, so that the spaces of the bone become larger.

Alexis Thomson, from his own and König's observations, distinguishes three varieties of tuberculosis of bone.

1. *Localised Chronic Tuberculosis.*—Here the lesions are comparatively small. They possess a translucent, reddish-grey periphery, fairly well defined to the naked eye, with a greyish or more frequently yellowish caseous centre.

If extension is relatively rapid the caseous material contains gritty particles—the remains of thinned, incompletely absorbed, trabeculæ;

if slow, the bone is absorbed and an abscess forms.

Sometimes, however, the central part becomes densely infiltrated, and may become separated and lie as a sequestrum in a bath of pus, the cavity being lined with tuberculous granulation tissue. Whether or not this central sclerosis takes place, the granulation tissue may become organised into dense fibrous tissue, which later becomes the seat of osseous deposit.

The central part may cicatrise, and healing become complete, or it may remain soft and still contain active tubercle bacilli, which later may cause recrudescence of the disease. Even though bacilli be not demonstrable microscopically in such lesions they may produce tuberculosis on inoculation into animals (Hamilton, M'Weeney).

2. *Tuberculous Necrosis.*—In this condition a visible sequestrum is produced which, as König pointed out, remains in connection with living bone. The bone containing it may show no alteration in external conformation. If the sequestrum is exposed on the surface of a joint, it becomes eburnated and polished. Probably the change is primarily due to tuberculous ostitis, which produces increased density and finally death. Surrounding the sequestrum grey granulations are seen in the medullary spaces.

3. *Diffuse progressive tuberculosis* is more common than the other two forms. The lesions in this form differ from those just described by the absence of a limiting wall. It is met with most typically in short bones. The spongy tissue becomes diffusely infiltrated with yellow caseous material, and this spreads by direct continuity from one bone to another, causing destruction of the bony framework — caries — often associated with suppuration.

This is the common lesion in strumous dactylitis, where the caseous material dilates the shaft, and also induces secondary thickening of the soft tissues around.

In short bones, and in the ends of long bones, caseation may not be evident, but rarefaction may be extreme from replacement of the marrow by vascular granulation tissue, with great increase in the number of round cells and osteoclasts. On maceration a very delicate framework of bone remains. Such altered bone may be readily cut with a knife.

Healing may take place. From the granulation tissue new bone may develop, replacing the diseased, and cause anchylosis. This healing is sometimes only apparent, and recrudescence may take place.

In the neighbourhood there are accompanying proliferative changes, resulting in new formation of bone, especially under the periosteum — "osteophytic" outgrowths.

Results of Spreading Tuberculosis of Bone.—It may penetrate to the surface and produce exfoliation of bone with formation of a cold abscess, *e.g.* in the lumbar spine or in the popliteal space.

If near epiphyseal cartilage, a gross lesion may completely destroy it. If small, the lesion destroys part only, or may extend through it. The epiphyseal cartilage may sclerose, becoming converted on the diaphyseal aspect into calcareous or bony tissue, so that its bone-forming function ends and the limb becomes stunted.

If near joint-cartilage, the granulation tissue may fungate through it, or may spread underneath it, separating the whole. The cartilage may become undermined from the edge, or it may become ulcerated, in all cases tuberculosis of the joint resulting. The invading and destructive power is greatly increased if septicity is superadded. It may give rise to general tuberculosis. Zahn of Geneva found this to occur in 3·2 per cent of his cases.

TUBERCULOSIS OF JOINTS. — Tuberculosis of joints is usually secondary to that of bone; it may be primary in the synovial membrane, or the epiphyseal ends of the bones and the synovial membrane may be simultaneously affected.

The disease is mostly one of children.

When secondary to bone, the disease may erode the cartilage and attack the joint directly, or it may extend in the first place to the synovial membrane at the margin of the cartilage.

Tuberculous Synovitis.—This presents three varieties. (1) *Miliary Tuberculosis.* This condition is described only in connection with the knee-joint. The membrane is not greatly thickened, but numerous grey tubercles can be detected, especially round about the margins of the patella. The joint contains turbid synovial fluid, with or without flakes of lymph. Microscopically the tubercles are very cellular, and are arranged upon the blood-vessels. Alexis Thomson states that this form seems specially associated with previous genito-urinary tuberculosis.

(2) *Nodular Tuberculosis.*—In this form the synovial membrane is firm, and from it nodular, cauliflower-like formations project. The turbid synovia contains frequently loose bodies, fibrinous or composed in part of cartilage or bone. These are derived from detached portions of the nodules or semi-lunar cartilages, or develop from the granulation tissue which forms in the synovial membrane.

(3) The most common form, however, is that known as "*gelatinous degeneration*," "tumour albus" or white swelling, "strumous synovitis."

Here the synovial membrane becomes converted into vascular granulation tissue, pink, swollen, and pulpy, microscopically showing typical giant-cell systems, but rarely caseation. The exudation into the joint is serous, or serofibrinous. The altered synovial membrane after a time overlaps the edges of the cartilages, and aided by the changes already described in bone,

which usually accompany it, it begins to eat away the cartilage. In time this becomes wholly destroyed, and the whole interior of the joint becomes lined with tuberculous granulation tissue, and is practically an abscess cavity. The disease then begins to extend to the surrounding tissues; the gelatinous degeneration attacks the ligaments, tendons, and tendon-sheaths. These become softened and disorganised, permitting of a pathological dislocation of the bones entering into the formation of the joint.

Abscesses may also form outside the joint, and the pus burrowing to the surface produces at length a fistulous opening communicating with the joint. The connecting sinus is lined with tuberculous granulation tissue which fungates at the surface around the opening in the skin.

Then if the bone has been attacked secondarily, tuberculous caries extends in it until a great part of the epiphysis has been destroyed. Meanwhile, as a concurrent result, osteophytic outgrowths usually form on the shaft surface of the unaffected bone near.

Tuberculosis of Brain and Spinal Cord.—The disease generally occurs as a leptomeningitis. It occurs both in adults and children, but more commonly in the latter, the pia-arachnoid in children appearing to be specially prone to attack.

It may be a part of generalised tuberculosis, and, in this case, the primary focus is usually to be found in a softening lymph gland, either thoracic or abdominal, or it may be the only secondary manifestation present, following destructive tuberculosis of the lungs. In most cases, however, careful examination, microscopic if necessary, will disclose some evidence of the disease in other organs. It also follows distant disease of bones or of the generative organs.

Lastly, it may occur by direct extension from tuberculous disease of the bones forming the cranial cavity, more particularly disease of the temporal bone.

The writer found that out of 96 cases—all under twelve years—in which it could be approximately determined, the primary focus was in the bronchial glands in 60 cases (62·5 per cent), in the lungs in 15 cases (15·6 per cent), and in the remaining 21 cases (21·9 per cent) it was apparently abdominal.

In 8 of the 96 cases the child was under one year of age.

In 11 cases of tuberculous meningitis caries of the middle ear existed, and in 6 of these no other causative focus could be found.

In a typical case of cerebral meningitis in a child, on removing the calvarium and dura mater the convolutions are found to be flattened, and the sulci narrow. The surface of the arachnoid is dry, glistening, and sticky, and may be pale, or more often congested. A few grey or yellow tubercles may be detected along the Rolandic fissure, but rarely elsewhere on the vertex. The Pacchionian bodies in children may be mistaken for tubercles.

If one then proceed to remove the brain in the usual manner (*see* Post-Mortem Methods, vol. viii. p. 208) a large excess of serous fluid, turbid in appearance, escapes, this excess being a very common characteristic.

On examining the base, the structures from the chiasma backwards are veiled by recent deposit of tough, gelatinous lymph, usually greenish in colour, in the sub-arachnoid space. Similar deposit occurs on the tips of the temporal lobes, and over the anterior part of the superior vermiform lobe of the cerebellum.

When this deposit is at all thick the tubercles cannot be distinguished, but one can almost always detect them by separating the lips of the Sylvian fissure which are glued together, and examining the vessels there, using a magnifying glass if necessary. If they cannot be demonstrated even here, they can usually be made out distinctly on the upper surface of the cerebellum.

The grey tubercles occur as small, whitish spots on the line of the small vessels interrupting their continuity. When these are examined under the microscope, the condition appears as a cellular infiltration and thickening of the adventitia, giant cells being very seldom found, or the nodule may have extended more in the vessel wall, causing occlusion of it, and showing some necrosis in its centre.

In an ordinary typical case the tubercles are found closely aggregated in the Sylvian fissure, and spreading thence on to the surfaces in the neighbourhood. They also may extend up along the Rolandic area, diminishing in numbers as they go. They are also readily seen, but scattered, on the under surface of the frontal lobe and over the anterior part of the temporal convolutions. On the under surface of the cerebellum they are visible, but not so distinctly as on the upper surface extending outwards from the vermiform lobe. Adhesions, usually recent, exist, as a rule, in the longitudinal fissure. Grey tubercles may be seen in these, and also extending along the inner surface of the cerebral hemispheres close to the corpus callosum.

Entering the interior they may be detected in the choroid plexus, velum interpositum, and sometimes even on the ependyma. This usually is granular. The ventricles, chiefly the lateral, are dilated, and contain excess of turbid fluid, this excess being partly due to the inflammatory condition of ependyma and choroid and probably also in part to a degree of obstruction to the drainage of the ventricles produced by the thickened velum. The foramen of Majendie is never blocked as in posterior basic meningitis.

The appearance of the brain itself is also altered. Under the ependyma the white matter is softened, sometimes almost pulpy, and the corpus callosum tears very readily. It is difficult to explain this morbid change. It is

probably not a "white softening" so called, as no special signs of it exist during life. Some suppose that the inflamed ependyma allows the ventricular fluid to permeate the brain substance, and produce a condition resembling maceration. If it were due to the spread of tubercle nodules along the vessels, one should expect it to be specially evident in the cortex, where such spread takes place.

Yellow softening has been described as occurring in the area of cortex supplied by the middle cerebral artery (Barlow).

Localised Miliary Tuberculosis.—While this does occur in children, it is more common in adults than the generalised condition, which is commoner in children.

So far as the writer can determine from his own experience, such tubercles are usually yellower, larger, flat, and sometimes confluent. The patches occur on the vertex, often along the Rolandic area, and though the condition, as seen post-mortem, seems slight, it is nevertheless often rapidly fatal.

In tuberculous meningitis the disease may spread to the choroid—affording a physical sign of use in the later stages.

Occasionally one meets with fibrous areas on the convexity containing small caseous deposits, which would indicate that recovery may take place from this localised form. The generalised form may, to all intents and purposes, be looked on as invariably fatal.

The frequency of the occurrence of tuberculous meningitis in children may be inferred from statistics prepared by the writer from the post-mortem records in the Edinburgh Sick Children's Hospital. Out of 331 cases of tuberculosis, the central nervous system was involved in 158 cases; acute tuberculous meningitis occurred alone in 121 cases; acute tuberculous meningitis with caseous nodules in 26 cases; and caseous nodules alone in 11 cases. In the greater number of the second class the caseous nodules proved the centre of dissemination of the meningitis. Still found caseous nodules in 15 per cent of his cases.

Caseous tuberculous nodules varying in size up to that of a walnut occur in the substance of brain or cerebellum, or develop from the matres and grow downwards into the brain. The former appear to have no special proclivity for cerebrum or cerebellum. They also develop in the basal ganglia and, rarely, even in the choroid plexus. Surrounding them smaller tubercles are usually found, and they may give rise to an acute meningitis, usually localised to their neighbourhood, but sometimes general.

Spinal Cord. — The spinal meninges are usually found affected along with the cerebral, and secondary to them. Occasionally, however, they are free, as, for example, in 9 out of 35 cases examined at the Edinburgh Sick Children's Hospital. Occasionally the disease commences in the spinal cord, as, for example, by extension from a vertebral caries or from other localised tuberculosis near. This appeared to be the case in 4 out of the above-mentioned 35 cases.

Very frequently the naked-eye appearances are not so marked as in the brain, the surface being simply congested and granular, with delicate adhesions to the dura, but the tubercles may be well marked.

The condition varies in intensity in the different regions of the cord. It is frequently confined to the cervical cord. It may affect the cervical and lumbar cord, missing the dorsal, or it may affect the lumbar cord only, or it may be evident in the cervical region, and gradually become less so as one passes downwards.

Early Tuberculosis of the Dura Mater.—Occasionally a few flattened soft granulations are found on the dura. The writer recollects only three of such cases. They are never of large size.

Skin. — Lupus is the commonest form of tuberculosis of the skin. In this case lymphoid cells collect in masses round the vascular network in the corium, and in the meshes epithelioid and giant cells may be found. The giant cells differ somewhat from those found elsewhere, in not possessing distinct processes. Tubercle bacilli are present, but in such scanty proportion that they are hard to detect.

The sebaceous glands and hair follicles are destroyed, but it is said (Coats) that the sweat glands are spared. The condition undergoes degeneration and ulceration, or may cicatrise without ulceration. It may be unaccompanied by tuberculous manifestation elsewhere.

Lesions of Tuberculosis in Animals

On the whole, appearances similar to those found in man prevail. There are, however, certain minor variations in structure in certain animals, and bacilli are more readily demonstrated than in the tissues of man.

In *cattle*, tuberculosis is very common. The structures most frequently affected are glands and serous membranes, particularly the pleura and peritoneum, and the lungs.

In the serous membranes the tubercles form masses frequently of some size, which may form villous projections from the surface. These nodules have been compared to pearls, whence the terms "Pearl disease" or Perlsucht. When they are in the form of flattened fibrous tubercles they are compared to duckweed, and have been given the name Meerlinsigkeit. In internal organs, comparatively large rounded nodules and sometimes considerable infiltrations are met with.

These nodules are peculiarly prone to calcification.

Tuberculosis in *horses* somewhat resembles that in cattle. Occasionally large cellular masses

are formed which have been mistaken for sarcoma.

In *swine* tuberculosiss occurs frequently. It resembles in its general characters bovine tuberculosis. Invasion often takes place by way of the tonsils, as shown by Sidney Martin.

The disease is very common in *monkeys*. It usually begins in the lungs, but is apt to extend to other organs as a chronic general tuberculosis, so as to form comparatively large foci, which usually break down and produce cavities.

Tuberculosis of *fowls* is produced by a bacillus probably related to that of mammalian tuberculosis, but varying to some degree in morphological and biological characters. These differences most probably depend on environment. Nocard, Fischel, and Mafucci succeeded in bringing about approximation in external qualities.

Portals of Invasion and Channels of Spread in Man

The tubercle bacillus may invade the body by way of the respiratory passages, the alimentary tract, the skin, and more rarely by the generative organs during intercourse.

The first of these, the *respiratory invasion*, is by far the most common, both in adults and children. Even before Koch read his remarkable paper at the London Congress in 1901, this opinion was generally held. He considers that entrance is obtained so seldom by other passages and surfaces as to be a negligible quantity. It is a question, however, whether this dictum holds good so far as children are concerned, or at least, so far as this country is concerned, for statistics carefully compiled from the records of some of the more important British Hospitals for Sick Children would lead us to conclude that ingestion tuberculosis is still important, though much less important than formerly was thought.

In considering the chances of exposure to infection by this method of transmission it is necessary to remember that—(1) The tubercle bacillus is not an ubiquitous organism; it does not occur diffused throughout the whole atmosphere, but only in the neighbourhood of cases of the disease.

2. The bacillus dies when exposed to fresh air and sunshine, whether direct or diffused.

3. The chief danger is from sputum from cases of phthisis. If every consumptive could be taught to destroy his expectoration, the incidence of the disease would sink to insignificant proportions.

4. In ordinary quiet respiration the expired air is sterile. In forced expiration, *e.g.* coughing, a fine spray of moist particles containing many bacilli in the case of a consumptive is projected, and this being inhaled by another person, may produce the disease. Koch considers this point of special importance.

5. Sputum is dangerous if allowed to dry and become mixed with dust. When this is raised by currents of air or conveyed to the mouth by the hands, as in the case of creeping infants, it causes tuberculosis in a susceptible person. If such dust is protected from the sunlight it retains its virulence for years.

In respiratory tuberculosis the bacilli gain admission by any part of the respiratory tract——nasal passages and naso-pharynx, larynx, trachea, and bronchial tree. The larynx rarely shows primary infection; this being most common below that level. In some cases the organism seems able to pass through the bronchial mucous membrane without producing a local lesion, and to infect the lymphatic glands.

From the naso-pharynx infection of the middle ear may take place by way of the Eustachian tube.

Alimentary Tract.—The bacilli are able to gain entrance through any part of this tract except pharynx, œsophagus, stomach and duodenum. The parts most commonly invaded are the lower part of the ileum and the tonsils. Bacilli have been demonstrated entering the latter.

From Sidney Martin's experiments one concludes that the tonsils are chiefly attacked by bacilli in articles of food. In his inhalation experiments he could not get invasion of the tonsil. There may be no local lesion either in intestine or tonsil, but the glands in connection with these are affected, and from these the disease spreads. Even though some authorities, *e.g.* Benda, will not admit a primary tonsillar infection, the evidence obtained from post-mortems in children and experience of cases in which removal of scrofulous cervical glands—the only manifestation of tuberculosis in the body—cure the patient completely, compel us to believe that infection certainly can take place by way of the tonsils. The intestine is much more often affected, however, either primarily or secondarily, to a destructive pulmonary tuberculosis. The bacilli enter through the Peyer's patches or are carried in through these by leucocytes.

To determine the relative frequency of respiratory and ingestion tuberculosis in children, the writer in 1899 investigated the post-mortem records of the Royal Edinburgh Hospital for Sick Children, and found that out of 331 cases in which the primary focus could approximately be determined, 221 or 67 per cent were of respiratory origin, and 93 or 28·1 per cent were the result of ingestion. Two years later, in the light of Koch's paper, he examined the additional records, and found that during this period 43 cases of tuberculosis were sectioned, of which 11 or 25·6 per cent were alimentary in origin. We have thus a total of 374 cases of which 104 or 27·8 per cent were, so far as could be determined, alimentary in origin.

Dr. Geo. Still found, at Great Ormonde Street, London, that in 63·8 per cent of his cases infection had probably taken place through the respiratory tract; in 29·1 per cent the alimentary tract had been primarily affected.

Dr. Carr, out of 120 autopsies on tuberculous children at the Chelsea Children's Hospital, found that 16·7 per cent showed evidence of primary abdominal infection.

Dr. Guthrie, at Paddington Green Children's Hospital, found in a series of 77 cases that 19 of these, or 26 per cent, had been infected through the intestine. Kossel found in 14 cases of tuberculosis in children, varying in age from one and a half to ten years, that the primary focus in 4 cases was in the mesenteric glands. There was no symptom of this during life. Heller and Hueppe give similar evidence.

These figures, which receive additional value from the approximate results arrived at by widely separated and independent observers, afford a strong ground on which to rest in refusing to accept Koch's dictum that tuberculosis is practically never ingestive in origin.

Even allowing a wide margin for error, we must remember that, after all, these are percentages of *deaths* from tuberculosis and do not indicate the frequency of abdominal tuberculosis in childhood during life. Many cases of abdominal tuberculosis recover; some cannot be diagnosed with certainty during life, and these with kindred cases must necessarily bring up the percentage of cases of ingestion tuberculosis.

In this country, therefore, we cannot accept statistics from other countries as applicable to our own circumstances.

So long as our population prefers to drink its milk raw this danger will remain, and individuals who are susceptible will be exposed to the dangers of infection through the alimentary tract.

The bacilli may invade the *skin*, either producing a local tuberculosis—lupus or scrofuloderma—or passing on and attacking the glands.

Frequently, as for example in tuberculosis of bones and joints, or of epididymis, one cannot detect the point of invasion.

Again, even in well-marked thoracic or abdominal tuberculosis it is impossible to tell the point of invasion. In an apparently undoubted respiratory tuberculosis the bacilli may have entered through some part of the alimentary tract, and by circuitous routes arrived at the bronchial glands or lungs, setting up tuberculosis there. The reverse may also take place, so that the figures in statistics of primary respiratory or alimentary tuberculosis must be looked on as approximate only.

Channels of Spread in the Body.—1. *Along Epithelial Canals and Surfaces.*—This is one of the most important means of spread, particularly in the air-passages and alimentary system. It explains why pulmonary tuberculosis is such a progressive and fatal disease.

In the lungs it generally begins at one focus or group of foci. These break down, and bacilli and softened caseous material are discharged into bronchi. During breathing and coughing this gets into other bronchi, setting up secondary foci. Caseation is usually a characteristic of tuberculosis which spreads by way of the bronchi. Liberated bacilli may be carried to the trachea and lodge in the larynx, or may even be carried to and infect the tongue. They may be swallowed and affect the mucous membrane of small or large intestine, the sputum passing through the stomach undestroyed. A similar spread occurs in genito-urinary system as already described.

2. *Lymphatics.*—Nodules may spread along the lymphatic vessels and spaces in lungs, nerves, cerebral membranes, intestine. A tuberculous peritonitis may spread through the lymphatic spaces of the diaphragm to pleura and thence to lung.

In the lung a tuberculosis spreading along lymphatics tends to cause fibrous formation with giant cells.

Tubercle may attack the thoracic duct.

3. *Blood-vessels.*—Spread takes place chiefly by way of veins, as demonstrated by Weigert, though Ponfick has shown that the walls of arteries may be invaded in exactly the same way.

If a tuberculous focus ruptures into the lumen of a vein the organisms are carried to the lungs, setting up tuberculosis there. They may pass through the lungs and set up a general tuberculosis. Tubercle nodules are constantly forming in the walls of capillaries and veins. Generally they become sealed up by thrombosis or by proliferation of the endothelial cells, so that dissemination does not take place. General tuberculosis can only occur when a focus breaks into a still pervious vessel.

Mixed Infection in Tuberculosis

It has for some time been urged that the tubercle bacillus is not able of itself to produce all the lesions met with in the disease called tuberculosis.

The late Professor Kanthack wrote very strongly against the idea that each infectious disease depended on one specific germ, and that all other organisms found in the tissues were unimportant so far as the etiology of the disease was concerned.

He believed that most of the manifestations of these diseases, for example, pneumonia, cholera, etc., are due to a mixed infection; the special organism requiring the assistance of others accompanying it to pave the way or produce a suitable nidus for its development.

In tuberculosis, as in these diseases, the doctrine of mixed infection has strong support.

Prudden showed that excavation following experimental infection became much more active if pyogenic organisms gained entrance to the tuberculous tissues.

A. G. Foulerton and Prof. W. Ophüls read papers on this subject at the British Congress of Tuberculosis (1901), and from these the following abstract is made. Foulerton's paper dealt specially with mixed infections in pulmonary tuberculosis.

"While there can be no question as to the cardinal part which *bacillus tuberculosis* plays as the primary, or original, factor in the condition known as chronic pulmonary phthisis, the influence of the specific parasite in the production of the symptoms characteristic of this condition, and its importance as an ultimate factor in the excessive mortality caused by the disease, are not by any means so well established."

Frequently, indeed almost invariably, the terms chronic pulmonary tuberculosis and chronic pulmonary phthisis are used in clinical medicine as if synonymous; whereas, pathologically, the two conditions differ widely, although agreeing in the fact that *bacillus tuberculosis* is present in the lesions of each. In uncomplicated chronic pulmonary tuberculosis the single organism present, the specific bacillus, is one which, under the given conditions, does not manifest more than a quite low degree of virulence in the majority of cases, whether as regards its direct action on the tissues actually infected, or as regards its action on the system as a whole by the production of toxine. In chronic pulmonary phthisis, on the other hand, to the action of this parasite is added the more acute influence of pyogenic organisms, whose potentiality for evil—again under the given conditions—is incomparably greater, whether by direct action on the tissues infected or by the production of toxines.

Chronic pulmonary tuberculosis is rarely recognised by the physician in his practice, because of the absence of any definite symptoms produced by it in the majority of instances, such cases as are diagnosed being usually those in which a certain amount of cough has attracted attention to the chest, and the sputum has, in consequence, been examined. On the other hand, such a condition of uncomplicated pulmonary tuberculosis, as being the initial stage of chronic pulmonary phthisis, must, we know, be an extremely common one, and the frequency of its occurrence is emphasised when we consider, not only the number of cases in which secondary infections have determined the onset of definite pulmonary phthisis, but also the large number of cases of pulmonary tuberculosis which, there is reason to believe, never arrive at that stage, and are not recognised at any time during life. The frequency of these latter cases can only be estimated from the number of instances in which lesions, usually of limited extent, indicative of cured or arrested tuberculous infection, are found in the lungs of adults dying from other causes.

Our knowledge of the bacteria met with in tuberculous cavities has been arrived at by the examination of the sputum during life, and of the walls and contents of the cavities after death.

Practically all who have worked at the subject are agreed that various species of cocci are the predominating organisms found, especially *Streptococcus pyogenes*, *Staphylococcus pyogenes aureus*, *Staphylococcus pyogenes albus*, *Diplococcus pneumoniæ*, and *Micrococcus tetragenus*. The first of these is the most important. Their local effects are manifest in an active destruction of the tissues actually infected, by the occurrence of perinodular pneumonia, and by what may be described in general terms as a lowering of the powers of resistance—a lowering of the vitality of the tissues in the neighbourhood of those actually infected; the latter effect being partly due to the direct action of toxines produced by the bacteria, and partly to vascular disturbance in the proximity of the lesions.

Their action on the system at large may be expressed either by the results following on the absorption of the toxines (toxæmia), or those of an actual infection of the blood generally by the bacteria themselves. The latter may be excluded as an important factor save only as a terminal infection.

It is of some interest to note that the bacteria which are under normal conditions arrested in the fore part of the nasal passages are amongst those which are most frequently found infecting pulmonary cavities, *e.g.*, *diplococcus pneumoniæ*, *staphylococcus pyogenes aureus*, *streptococcus pyogenes*, and *bacillus pneumoniæ* (Friedländer).

We have good reason for believing that the tubercle bacillus by itself would not, at all events in a chronic infection, produce all the pathological features of fully developed chronic pulmonary phthisis during life, but we must allow that infection of a cavity by, for example, streptococcus pyogenes, will afford a satisfactory explanation of phenomena such as evidences of active destruction, and of hectic fever with its accompanying evening temperature, morbid sweating, and general wasting. The tubercle bacillus in pure infection never produces active destruction of tissues; it, of course, produces granulomata which tend slowly to break down as a result of interference with their blood supply, but their very presence is evidence of a low grade of virulence in the organism which produces them. A quite similar result may be produced by a purely mechanical irritation.

As to toxæmia, all one can say is that in all forms of chronic pure tuberculosis in parts other than the lungs, the symptoms referable to a toxæmia are either of the slightest or con-

spicuously absent; but given a breaking down of tubercles in the lung substance, with secondary infection of the cavities or an ulceration of tuberculous lesions in the intestine, and one finds the symptoms of toxæmia well marked. As a result of such observations it has been suggested that the treatment of the secondary infections should be the chief object aimed at.

The comparative failure of methods of treatment by tuberculin is probably almost entirely due to the ignoring of the effects of such secondary infections by pyogenic cocci.

It is well known that by keeping phthisical patients in an atmosphere practically free from germs, such as may be attained at a high altitude, and by enforcing as complete rest as possible, they are protected against reinfection by pyogenic cocci, and placed in a condition in which the intensity of the present pyogenic infection may be reduced.

Ophüls considers that some of the acute exacerbations, which usually assume the form of pneumonic processes, are produced by the tubercle bacillus alone, whereas others are produced by the mixed infections. He chiefly examined sections from the walls of cavities and parts of the lung affected.

The opinions here expressed, though received in their entirety with caution, are supported by the experiments of Délèpine and Ravenel, who found that injection of mixed human sputa could cause the disease in lower animals which resist infection by pure growths of human tubercle bacilli.

Pansini also found that the bacilli of human tuberculosis were less virulent in pure culture than in tuberculous material or sputum.

Is it not also possible that the ignoring of mixed infections may invalidate conclusions based upon experiments made by Koch and others with pure cultures of the bacillus? These inoculation experiments, to begin with, do not and cannot reproduce the conditions usually met in natural infection. The animals used were perfectly strong and healthy, in nowise in a condition analogous to that of the child or adult who is unable to resist infection.

Again, account must be taken of the varying virulence of the races of bacilli used, a bacillus of low virulence producing unimportant lesions. Taking all together, therefore, the negative results obtained were such as were to be expected, and the conclusions drawn were founded on data to which exception can be taken.

Considerations such as these lead us naturally to the consideration of the relationship of the human bacillus to those of the lower animals, and particularly to that of the bovine disease.

Tuberculosis in Man and in the Lower Animals.—Acute interest has been excited in the question of the intercommunicability of human and bovine tuberculosis by Koch's recent paper at the British Congress on Tuberculosis (1901).

Previous to that, one organism, the bacillus of mammalian tuberculosis, was looked upon as the proved cause of the disease in man and cattle. Another bacillus, somewhat distantly related to the former, was credited with producing fowl tuberculosis. Most other vertebrates are, or can be rendered, susceptible to tuberculosis, and the disease is described even in cold-blood animals such as fish.

In order to clear the ground we shall consider shortly avian and piscine tuberculosis in relation to the human disease.

Avian Tuberculosis.—The morphological and biological characters distinguishing the bacilli of this disease from the mammalian disease have already been detailed. Can any relationship to the mammalian disease and bacillus be proved?

Nocard succeeded in so modifying cultures of mammalian tubercle that they became like those of avian tubercle, and also resembled them in pathogenicity. He found that cultures of mammalian tubercle, if placed in collodion sacs and grown for a length of time in the peritoneal cavity of fowls, gradually became fatal to fowls and took on the cultural characters of avian tubercle. (Congress on Tubercul., Paris, 1898.)

Fischel and Mafucci (*Lubarsch u. Ostertag.*, 1895, p. 233) were able to modify the appearance of mammalian and avian tubercle bacilli by cultivating on different media and at different temperatures. For example, if mammalian tubercle were grown on serum made from hen's blood, the resultant culture was like that of avian tubercle. They also found that mammalian bacilli, when grown on eggs, and on boracic acid agar, or thymol-glycerine-agar, developed long filaments showing multiple branching. On dogs'-blood serum a development took place like that of avian tubercle. More important still, by growing on boracic acid agar, Fischel obtained cultures of mammalian tubercle which no longer showed marked virulence for rabbits and guinea-pigs, but displayed effects in these animals like those of avian tuberculosis.

The evidence with regard to experimental infection of fowls with human tuberculous material is very variable and at times conflicting. There are some instances reported of natural infection of fowls with human tuberculosis. This is to a great extent opposed to the experimental evidence, probably from the fact that in experimentation all the conditions of the natural infection are not realised.

They may be infected with tuberculous sputum from their attendants, and tuberculosis has been observed in fowls reared in abattoirs, where they have been fed on contaminated meat or tuberculous viscera (Nocard).

The case reported by M. Cagny is well known. In *La Semaine Médicale*, 1888, he describes a case of transmission of human tuberculosis to poultry.

A number of chickens belonged to a family, one member of which was suffering from pulmonary phthisis. The chickens all became tuberculous, and had to be destroyed. Tuberculosis of the liver was the principal manifestation found after death. M. Cagny learned that the sick man was in the habit of walking in the garden, and that it had been noticed that the chickens eagerly seized and swallowed the material he expectorated.

Sir Chas. Cameron (*Lancet*, July 1889) refers to this question, and quotes a case in which about fifty hens died within a period of three to four months. The deaths began to take place some weeks after the arrival of a consumptive patient in the house near which the fowls were kept.

Mallerée (quoted, *Lubarsch u. Ostertag.*, Abth. ii. 1895, p. 235) relates a case in which hens developed tuberculosis from ingestion of the expectorations of a consumptive, and in their turn, being eaten raw, passed on the infection to a woman. A mother and child developed consumption. A young woman in a neighbouring house, during a period of four months, ate eleven hens which had died in the consumptives' house, and with the idea of obtaining as much benefit as possible, she consumed them raw. Several of the hens had developed tuberculosis of the intestine and liver from eating the tubercular expectorations. The young woman developed tuberculosis.

These few cases are related simply to show that there is a possibility of infection of poultry from man, and *vice versa*. In any case the risk seems to be slight, and may, in practice, be disregarded.

Kruse thinks that among the larger birds the parrot alone may be considered susceptible to mammalian tuberculosis.

Piscine Tuberculosis.—Bataillon, Terre, and Dubard have described a condition in the carp resembling tuberculosis, containing bacilli and giant cells. They separated the bacillus, which grew best at 23° to 25° C. Growth was rapid. It branched dichotomously. Kral and Dubard claim that they have converted human and avian tubercle bacilli into a form similar to that obtained from the carp by passage through fish, frogs, lizards, etc. The fish were supposed to have been infected from human tuberculous sputum expectorated into the water.

Relation of Human and Bovine Tuberculosis.—This subject is the field upon which the battle-royal is at present being fought in connection with tuberculosis. Is bovine tuberculosis transmissible to man? Is human tuberculosis communicable to cattle? Do the bacilli found in these diseases belong to the same species, are they sub-varieties of the same species, the variation being due to change of environment, the bacillus by prolonged residence in one species of animal taking on certain peculiarities, or are they totally different organisms?

Previous to Koch's oft-quoted speech at the London Congress, the close relationship, if not identity, of these bacilli was accepted as a settled fact, though it was recognised that bacilli from a bovine source were more virulent than those from a human source, and Frothingham, Theobald Smith, and Dinwiddie had discovered that cattle in general were immune to pure cultures of the human tubercle bacillus. Koch was also forestalled to some extent by Adami, who in 1899 drew attention to the fact that the evidence in favour of the view that bovine tuberculosis is transmissible to man was not so strong as was generally thought to be; that while cases did exist of such transmission, they were few in number. Even before Adami, similar views were enunciated by M. Alexandre (*Le Progrès Médical*, Paris, 29th Sept. 1894), who held, as a result of the examination of 20,000 cows in the Department of the Seine, that the consumption of the flesh and milk of our domestic animals was not the cause of the terrible mortality from tuberculosis among mankind, and that it was important that this once-accepted belief be thoroughly rooted out of our minds.

Koch's paper was divided into two parts, the first dealing with the possibility of infection of cattle from the human disease, the second with the possibility of infection of man from cattle through ingestion of milk or flesh of diseased cattle. His conclusions were, firstly, that "human tuberculosis differs from bovine and cannot be transmitted to cattle"; and secondly, "that the extent of infection by the milk and flesh of tuberculous cattle and the butter made from their milk is hardly greater than that of hereditary transmission, and that it is inadvisable to take any measures against it."

A. In support of his first dictum, Koch brought forward very strong experimental evidence. He used cattle which had been proved by the use of tuberculin to be free from tuberculosis. He found that inoculation and ingestion of human tuberculous material and of pure cultures of the bacillus were invariably without result, except that, occasionally, small suppurative foci were found at the site of inoculation; whereas inoculation of bovine tuberculous material, as invariably, caused generalised tuberculosis.

He did not obtain such uniform results in feeding pigs with similar materials, as, in some of those fed with tuberculous sputum, "little nodules were found in the lymphatic glands of the neck, and in one case a few grey nodules in the lungs." With bovine material, on the contrary, they all, without exception, developed severe tuberculosis.

Similar results were obtained by injecting cultures into the vascular systems of asses, sheep, and goats, which, it must be noted, are

looked upon as, comparatively speaking, insusceptible.

Möller of Danzig strongly supports Koch, as he has completely failed to communicate human tuberculosis to cattle.

Since Koch thus confirmed the previous conclusions of American observers, other workers have taken up the subject. In general they have confirmed the opinion that cattle are usually immune if pure cultures of the human bacillus are used, but we must clearly distinguish between experimental tuberculosis and natural tuberculosis. As Adami remarks: "We are realising more and more that the mere presence of virulent bacilli is not sufficient to set up the disease, that an equally important factor is the condition of the system. Thus, in connection with this very matter of tuberculosis in man, we know that while all are exposed to infection, at most 7 per cent die of the disease; those in good health resist infection; that this infection is specially liable to occur when the system has been lowered by another infectious disease, so that an attack of tuberculosis is notably liable to be dated from an attack of *la grippe*, pneumonia, typhoid, or other acute infection."

At the Congress, Lister, Nocard, Bang, and Sims Woodhead on the whole agreed that Koch had proved that infection of cattle from man seldom took place, and most authors since then have also written to the same effect; but experiments performed by Ravenel, Nocard, de Jong, Arloing, and others would go to show that inoculation even of *pure human bacilli* can produce tuberculosis in bovines, and there is a considerable body of evidence now to prove that if a material such as *sputum* from man, which causes a mixed infection, is used, tuberculosis can be readily conveyed to these animals.

1. Experiments with pure cultures of human origin.

Thomassen inoculated the anterior chamber of the eye of a calf with a pure culture of the human bacillus isolated from a case of joint tuberculosis. It was killed in six weeks, and a pretty well generalised tuberculosis was found.

Nocard (*La Revue Vétérinaire*, 1st Jan. 1902) produced a rapidly fatal meningitis by inoculating a small amount of human tubercle bacilli into the pia-arachnoid.

Intravenous inoculation produced the disease in a cow, whose normal resistance had been greatly reduced by chronic diarrhœa, but which was free from tuberculosis, as shown by the tuberculin test.

De Jong states that seven cattle became tuberculous as a result of the injection of pure cultures of the human bacillus. In only one of these was the disease grave and widespread; in four the lesions were retrogressive, and in the remaining two they were progressive (*La Semaine Médical*, 15th Jan. 1902).

Arloing reports three recent experiments. With a culture of human origin separated in 1896 he inoculated a young heifer and a calf intravenously. The calf developed a typical eruption of minute tubercles all through the lung. The heifer, after 120 days, was found to be only slightly diseased.

With a culture obtained from a case of pleurisy a calf two-and-a-half months old was inoculated intravenously. Death ensued in seventeen days. The lungs alone were involved, showing many young tubercles.

The third culture, isolated from a case of pleuro-pericarditis with pulmonary tuberculosis, was inoculated into a young bull intravenously. Death resulted in thirty-two days, the thoracic organs being principally involved.

Ravenel's experiments are of special interest. He made use of three cultures isolated from man. The first experiment would go also to prove that bovine tuberculosis can affect man.

It is well to quote his exact words, as the same ideas he expresses have occurred to others: "For several years past we have endeavoured to obtain material from cases of tuberculosis in children in which there was evidence of infection through the alimentary tract, reasoning that if children contracted tuberculosis through the ingestion of milk from diseased cattle, we would be most apt to find bacilli of the bovine type in these intestinal or mesenteric lesions. We have isolated from the mesenteric gland of a child, in whom the evidence of primary intestinal infection was clear, a culture which has for cattle the most intense pathogenic power."

"Two calves inoculated into the jugular vein and peritoneal cavity died in nineteen and twenty-seven days respectively; and a grown cow which was inoculated similarly died in eighteen days. All of these animals exhibited marked symptoms from the day of inoculation. Examination of the lesions, both macroscopical and microscopical, leaves no doubt that the animals succumbed to a pure tuberculosis."

"What interpretation is to be given to these remarkable results? One of two propositions must be admitted: either we have found a human tubercle bacillus having a pathogenic power for cattle quite as great as any bovine germ, or else we have found in the mesenteric gland of a child the bovine tubercle bacillus. If we accept the law of diagnosis laid down by Koch, namely, the inoculation test, the latter is the true explanation. I myself am strongly of this opinion" (*Univ. of Pennsylv. Med. Bull.*, May 1902).

Ravenel separated another culture from the mesenteric glands of a child and found that this was unusually virulent when inoculated into guinea-pigs, rabbits, puppies, a grown dog, and a calf. The result in the calf led him to believe that the usual method of employing

only guinea-pigs and rabbits in testing the virulence of the tubercle bacillus does not always give entirely satisfactory results.

It has occurred to others, for example, Adami and Higgins, that the virulence of a given culture of human bacilli might be increased by successive passages through cattle.

Ravenel and Leonard Pearson proved this. At first, two calves were inoculated at intervals of a week. One lived for 106 days, and in the case of the other life was more prolonged. From caseous bronchial glands in one of them another calf was inoculated, which lived forty-eight days. The experiment was repeated with material from this calf, and the inoculated animal died in twenty-three days. Two other calves in succession were employed, the result demonstrating that a great increase in virulence had been attained.

2. As we have already remarked, however, these experiments do not reproduce the factors met with in natural infection. It is recognised that the chief danger is from a mixed infection. For example, in respiratory tuberculosis in man infection takes place from virulent sputum, either dried and conveyed about in dust, according to Cornet, or in a fine spray, projected during speaking, coughing, or sneezing, according to Flügge.

If, then, we can induce tuberculosis by a mixed infection, carrying out experiments either with sputum or with material from the lungs of tuberculous subjects, we would reproduce more closely, though still not quite satisfactorily (seeing that the animals used are healthy), natural conditions.

Chauveau (*Congt. Tuberc.*, Paris, 1891) with such material produced tuberculosis in a heifer six months old; in another eleven months old; and in a young bull about ten months old. In these animals the material was given in the food. He also was successful in infecting a calf by intravenous injection. On subcutaneous inoculation, in every case a typically tuberculous tumour developed at the seat of inoculation, no general infection, however, following. This does not accord with the statement of Koch, who referred to these experiments of Chauveau among others as proving that animals which were fed with human tuberculous material never developed tuberculosis.

Ravenel (*Lancet*, Aug. 1901) inoculated four calves of nearly the same age, intraperitoneally, with a uniform quantity of tuberculous sputum from different sources, but all containing numerous bacilli. One calf was unaffected. Of the remaining three, in which infection had taken place, two showed very extensive lesions of the nature of "grapes" or "Perlsucht."

In the case of pigs, he found that five out of six inoculated with human material died after a considerable time. From these pigs he inoculated others, producing a rapidly fatal diffuse tuberculous pneumonia, the virulence of the organism having apparently been raised by passage through the former animals.

To compare with these results he inoculated other animals under similar conditions with an equivalent dose of bovine tuberculous material, and found that this was much more virulent for all animals.

Böllinger (*Munch. med. Wochensch.*, Jan. 1894) records an experiment performed long before. He inoculated a healthy calf—three months old—intraperitoneally with tuberculous fluid from a human being. Seven months later characteristic "Perlsucht" of the peritoneum was found.

Délèpine, by inoculating a mixture of tuberculous sputa into calves, has succeeded in causing tuberculosis.

Klebs, Kitt, and Crookshank have all succeeded in producing tuberculosis in calves by intraperitoneal injection of human tuberculous material. Sidney Martin fed six calves with tuberculous sputum. Four of these showed lesions of the disease when killed, two remained healthy.

All these experiments, then, show that Koch's first statement, that human tuberculosis cannot be transmitted to cattle, is "erroneous and untenable" (Ravenel).

B. The second and more important question is whether bovine tuberculosis is transmissible to man.

Koch states absolutely that this transmission does not occur, or but rarely.

"Though the important question whether man is susceptible to bovine tuberculosis at all is not absolutely decided, and will not admit of absolute decision to-day or to-morrow, one is, nevertheless, already at liberty to say that, if such a susceptibility really exists, the infection of human beings is but a very rare occurrence. I should estimate the extent of infection by the milk and flesh of tubercular cattle, and the butter made of their milk, as hardly greater than that of hereditary transmission, and I therefore do not deem it advisable to take any measures against it."

He brings forward a very strong argument in his favour when he says further: "If the bacilli of bovine tuberculosis were able to affect human beings, many cases of tuberculosis caused by the consumption of alimenta containing tubercle bacilli could not but occur among the inhabitants of great cities, especially the children, and in reality this is not the case."

At the recent Congress on tuberculosis held at Berlin in October 1902, Koch still upholds his former contentions, and is still convinced that his view is correct. Due consideration must be given to the opinions, deliberately formed, of a man in his leading position, and the evidence against them must be very carefully weighed. Möller of Danzig strongly supports Koch.

Even at the outset, however, we may decline to accept statistics, wheresoever originating, as applicable to every country; and we are warranted, even granting that Koch were right so far as his own experience, or experience in Germany, is concerned, in refusing to allow his conclusions to dominate or alter our measures adopted after long discussion for the prevention of tuberculosis in Britain unless or until statistics from British sources applicable to our own circumstances and case show these measures to be mistaken. Even statistics gathered from different parts of Germany do not agree, and in different parts of Britain the incidence of tuberculosis varies. In London and Edinburgh the disease is common. In other large cities, *e.g.* Birmingham, it is not so rife.

Previous to the 1901 Congress the relationship, if not identity, of the bacilli was accepted as a settled fact, though it was recognised that the bacilli from a bovine source were more virulent than those from a human source.

We had also recognised the fact that respiratory infection was much more important than alimentary infection, even in children; but we were not prepared to accept, off-hand, Koch's dictum that the possibility of infection from infected alimenta is a negligible quantity.

As Montizambert remarks in his report to the Canadian Minister of Agriculture: "We shall all rejoice if Koch is proved to be right, and our duty is to hope that he is. But the question is, can his theory be safely accepted? That can only be decided by an examination of the facts upon which it is based. No man, however great, can lay down a law in science by the mere weight of his opinion."

[*Bovine and Human Tuberculosis. Report of Royal Commission.*—One of the results of Koch's pronouncement in 1901, that bovine tuberculosis was seldom a cause of the disease in man, was the appointment of a Royal Commission to inquire whether the disease in animals and man is one and the same, whether animals and man can be reciprocally infected by it, and, if transmission from animals to man takes place, what circumstances favour such transmission. The second interim report, issued in 1907, deals mainly with the results of an investigation of the effects of experimental infection with bacilli of bovine origin and bacilli of human origin. The Commissioners are able to answer the second reference to them in the affirmative, and to assert that animals and man can be reciprocally infected. The other questions remain as yet in abeyance. The main features of the report may be summarised as follows:—Two types of bacillus can be distinguished—the bovine and the human—but the difference between them is in the main one of virulence. The histological and anatomical features of the lesions caused by each are the same, except in degree. The bovine type of bacillus is much more virulent than the human type. The viruses obtained from cases of tuberculosis in man were found to be divisible into two groups, one corresponding to the human type of bacillus, the other to the bovine type. This fact is of peculiar significance when the source of the two groups of viruses is considered. Of 14 cases yielding a virus corresponding in all respects to the bovine type, 10 were primary abdominal tuberculosis, 3 cervical glands, 1 phthisis. Of 40 cases yielding virus of the human type, 8 were primary abdominal tuberculosis, 10 respiratory tuberculosis, 9 bone and joint disease, 2 genito-urinary tuberculosis, and the rest cervical glands. Most of the cases, therefore, yielding a virus resembling that of bovine tuberculosis, were instances of alimentary infection; most of the other group were instances of infection arising outside the alimentary tract. The possibility of the two types of bacillus being, or becoming under suitable conditions, interchangeable, is still under investigation by the Commission, and no definite statement can yet be made. The conclusions at which the Commissioners arrive are:—1. That a certain amount of tuberculosis, especially in children, is directly due to infection with the bovine bacillus. 2. That tuberculous milk is clearly a cause of tuberculosis, and fatal tuberculosis, in man. 3. That a very large proportion of tuberculosis contracted by ingestion is due to bovine bacilli. 4. That more stringent measures are required to prevent the sale or consumption of milk from tuberculous cows.]

That Koch's conclusion is incorrect, at all events so far as Great Britain is concerned, is proved by a considerable body of clinical and statistical evidence; and even in other countries evidence is obtainable which warrants us in doubting the absolute correctness for all peoples and climes of his sweeping statement.

In some countries, as in our own, the chances of infection are greater than in Germany. We must remember that, whereas the use of boiled milk is very common, if not the rule, in Germany, it is the exception in Britain. No doubt the use of "Pasteurised," "sterilised," or cooked milk is increasing in this country, but the flavour of boiled milk is not grateful to the national palate, and we still prefer raw milk with all its extraneous contents, derived from the cow, the milker, the byre, and later from the dust of streets and occupied apartments. As has been remarked, there is no article of food in common use which is more exposed to contamination in its progress from the producer to the consumer, an article which is produced sterile, and in nature is intended to be consumed without chance of contamination.

This is a point of very considerable importance which tends to affect the incidence of the disease.

Again, in healthy subjects consumption of

raw milk, with all its concomitants, causes no evil effects, as is proved by the fact that probably all of us have unwittingly partaken of tuberculous milk. Clifford Allbutt has tested this by allowing his own family and servants, as well as two families living near, to consume the highly bacilliferous milk of a favourite cow. Not one individual developed the slightest trace of tuberculosis. This is perfectly credible, but the conditions of the experiment, if so it may be called, do not fit the case. Pasteur, in investigating a disease of silk-worms, traced the evil to a micro-organism, but a far more valuable and suggestive outcome of his researches was the discovery that so long as the silk-worms were in health they laughed and grew fat on the micro-organisms; only when they deteriorated in condition and suffered from digestive derangement could they enter and cause the disease. Similarly, healthy individuals do not really acquire tuberculosis, but only such as are predisposed through a general weak condition of body, or through bad environment, or who are the subjects of intestinal or gastric catarrh. The majority of cases of respiratory tuberculosis may be left out of account, as these usually result by infection from a previous case in man, though all cases of respiratory tuberculosis must not be looked upon as necessarily respiratory in origin.

A great deal of information is gained from examination of suspected cases after death, but here again it must not be forgotten that only the very severe cases of abdominal tuberculosis come to the post-mortem room. Tuberculosis, and particularly abdominal tuberculosis, tends very frequently to heal. Records of necropsies at home and abroad indicate that traces of healed tuberculous lesions exist in from 20 to 50 per cent of all persons dying of other diseases.

Taking the case of abdominal tuberculosis specially, Sir William Gairdner, in *Lectures to Practitioners*, says: "A far too grave prognosis is arrived at in cases of tabes (mesenterica), consequently many cases of tubercular disease, not only of the mesenteric glands, but also of the peritoneum, recover, only the graver cases succumbing."

It is a matter of common knowledge and experience that cases of undoubted abdominal tuberculosis, with, frequently, no evident trace of tuberculous mischief elsewhere, will heal after, or are cured by, laparotomy.

Churton (*Lancet*, 1897, i. p. 594) gives instances of rapid absorption of tuberculous masses in the abdomen *without* operation, and others at the same meeting related similar cases coming under their own observation.

Woodhead (*Lancet*, 1888), after quoting statistics, says: "It is evident that tubercular conditions of the abdomen are much more common than can be inferred even from the figures quoted."

Hence post-mortem statistics must not be taken as indicating the prevalence of abdominal tuberculosis during life.

Koch states that he has only seen two cases of undoubted primary intestinal tuberculosis, and Müller, from 500 autopsies on children at Munich, concludes that the intestines are seldom the avenue for tuberculous infection in children. Though this, again, is not the experience of physicians and pathologists in this country, we must note that ingestion tuberculosis does not limit itself to producing intestinal ulceration, but, as all of us have seen, the organism may pass through the wall of the intestine without markedly affecting it, and cause a tuberculous peritonitis, or more often tuberculosis of the mesenteric glands. Following on this, other groups of glands may be infected, such as the retro-peritoneal, the cœliac, posterior mediastinal, bronchial, and root glands, and these, breaking down into a bronchus or vessel, may give rise to a secondary respiratory tuberculosis, or to a generalised tuberculosis, just as the condition may travel in the reverse direction from primarily infected bronchial glands to mesenteric glands.

Once more, in ingestion tuberculosis the bacilli may invade by way of the pharyngeal and faucial tonsils, and proceed downwards along the cervical chains of glands to the mediastinal and root glands. This course has been demonstrated experimentally in the pig by Dr. Sidney Martin, who failed to infect by inhalation of bacilli, but found that in feeding experiments the rule was that invasion took place by way of the tonsils, which are very prominent in the pig. By many, chronically enlarged tonsils in man are looked upon as predisposing to pulmonary tuberculosis. Sidney Martin lays great stress on his experiments as proving the etiology of the analogous cases of "strumous" glands in children (Report of Royal Commission on Tuberculosis, 1895-96). All practitioners have shared the experience of Wohlgemuth when he states that in such cases removal of the affected glands removes the focus of disease, and cures the patient.

Böllinger, F. Hueppe, Weleminsky, and Sims Woodhead also emphasise the importance of this method of infection in children.

The last named says (*Lancet*, 1894, ii. p. 958): "I am driven to the conclusion that this method of infection of the glands through the tonsils must be of comparatively frequent occurrence, especially in children living under insanitary conditions, and subjected to various devitalising influences." Further referring to the fact that in pigs the lungs may be affected secondarily to tuberculosis of the pharyngeal tonsillar structures through the connecting chains of glands, he says, and the writer's own experience supports his statement: "I have seen a similar line of invasion in children, sometimes

alone, but usually associated with tubercle extending from the mesenteric glands in the direction already indicated. In all these cases, it would appear that the glands at the root of the lungs become tuberculous and caseous before the pulmonary tissue itself shows any evidence of disease." Of course, in such cases, one must examine the lungs very carefully to exclude the presence of any old dried-up tuberculous focus or scar indicating disease of the lung antecedent to that in the glands.

Again, the same author says (*loc. cit.*, p. 960): "I have seen in case after case in children, and in animals fed on tuberculous material, the lungs markedly affected, but in a large proportion of these cases it has been possible to trace the course of invasion back from a caseous or old calcareous mesenteric gland, through the chain of retro-peritoneal glands up through the diaphragm to the posterior mediastinal and bronchial glands, and so on to the lungs."

Therefore, as has already been remarked, all cases of respiratory tuberculosis must not necessarily be looked upon as primarily so, and are not all due to inhalation.

Passing from this criticism of Koch's statements, which, when all is said and done, are based on negative experimental evidence only, we may proceed now to examine positive evidence in favour of the contention that tuberculosis may be transmitted to man from cattle.

Speaking generally, the great danger to be apprehended is from *dairy* cattle, which are kept to a large extent in ill-ventilated byres and cowsheds, rather than from cattle kept in the open; the former being herded together under conditions favouring the transmission of disease from one to another, and its retention in a fairly constant ratio.

"In places where cattle are allowed to graze at liberty in our Alps and in marshes, uncooked milk will often be drunk with impunity, but in the low country, and in towns where the dairy cattle are always crowded together in damp cow-houses and infection spreads from animal to animal, Koch's assertion that there is no cause for uneasiness would be an invitation to carelessness which would undo all that has been accomplished." (F. Hueppe, *Lancet*, 1901, ii. p. 613.)

As great a proportion as 50 to 70 per cent of cattle kept under such conditions has been found affected with tuberculosis by the tuberculin test, and the udders are found tuberculous in ½ to 2 per cent.

As to the general question, whether dairy cattle are responsible to any extent for the prevalence of tuberculosis in the human subject, we may first cite the investigations of E. F. Brush (*Archiv. Pœdiatr. Philad.*, 1890, p. 551), who considered the coincident geographical distribution of tuberculosis and dairy cattle.

He shows that in lands like Egypt the indigenous inhabitants retain immunity while associating for long periods with consumptive immigrants; while, on the other hand, in regions like Australia and the Sandwich Islands, the inhabitants have become infected after the introduction of dairy cattle. Referring to China, he speaks of the poor Chinese as a people who do not use milk, while the Tartars in that country are milk and meat consumers; and, therefore, the observations of medical men are very confused, and they confess that they cannot understand why the disease prevails among the dominant Tartar class and not among the poor Chinese, who, according to all preconceived notions, ought to be tuberculous.

In South America, where cattle are exceedingly numerous, but the use of milk almost unknown, or used only after being boiled, the natives enjoy still a large immunity.

Contrasting Spain and Morocco, where climate and other conditions must be nearly equal, he finds that Morocco, where there are no European dairy cows, is exempt from tuberculosis; while in Spain and Portugal, where dairying is carried on in the European style, tuberculosis prevails. He expresses a belief that dairy and beef cattle could be bred in such a way as to eliminate tuberculosis.

If this be the case, it is difficult to deny the connection between dairy cattle and the prevalence of tuberculosis.

Statistical Evidence.—The evidence of the mortality tables of the Registrar-General might be cited in support of the prevalence of ingestion tuberculosis, though, according to M'Fadyean, we should not lay too much stress upon it.

There has been in Great Britain during the last forty-five years a reduction of 27·9 per cent in the deaths from all forms of tuberculosis. The reduction in deaths from phthisis pulmonalis reached 66 per cent, while the corresponding reduction from tabes mesenterica only reached 3 per cent. In this connection Sir Richard Thorne-Thorne says: "In considering the latter cause of death, we are dealing with a totally different state of affairs. The matter, too, assumes a still more serious aspect if we limit ourselves to the first year of life, when milk is most largely used as food, for then we find that the reduction in the rate of deaths from the various forms of tuberculosis, which reduction has been going on at all ages for about half a century, not only disappears, but is actually transformed into a large increase, reaching no less than 27·7 per cent. This in itself is grave enough, but its significance is still further increased when we remember what are the circumstances under which this increase in the rate of deaths from tabes mesenterica has gone on synchronously with the decrease in that of other forms of tuberculosis."

Adami, criticising these statements and figures,

cannot otherwise explain them save on the supposition that impure and infected milk is an essential factor in the production of abdominal tuberculosis in young children. Not all these cases, it is true, should be regarded as due to milk. The habit of expectoration by adults, the creeping habits of children, their liability to put everything into the mouth, lead to a very great possibility of infection with human bacilli through the digestive tract. But admitting this, and admitting even that the hygiene of the house in Great Britain has not improved at the same rate as has the hygiene of the factory and of work-places in general—an improvement which is the main cause of the decrease in tuberculosis in adults in Great Britain—if mesenteric tuberculosis were in the main due to infection with human tubercle bacilli, then, with the lessening of tuberculosis in adults, there ought certainly to be a corresponding diminution in the number of cases of tabes mesenterica in children, and *this has not occurred.* (*Canadian Blue-Book*, A. 1902. *Sessional Paper*, No. 15, p. 126.)

The examination of post-mortem records in Hospitals for Children and statistics based on these give important information.

It has been long taught and believed that, in children, ingestion tuberculosis was by far the commoner form in which the disease occurred. That this is not so has been abundantly proved by recent statistics. These statistics, obtained from the post-mortem room, must of course not be taken as indicating the relative prevalence of inhalation and ingestion tuberculosis during life, the proportion of ingestion cases being almost certainly higher from the reasons already given.

In the Hospital for Sick Children in Great Ormonde Street, London, Dr. George Still was able to determine the channel of infection with some degree of certainty in 216 cases, and of these he refers 29·1 per cent to alimentary tuberculosis, the bacillus entering through the intestine. In the remainder, invasion had most probably taken place through some part of the respiratory passages.

In an investigation undertaken in 1899, in ignorance of the simultaneous work of Dr. Still, but with the same idea in view, the ascertaining of the proper proportion of inhalation to ingestion tuberculosis, and feeling that the ratio of ingestion tuberculosis had been placed too high, the writer examined the post-mortem records of the Royal Hospital for Sick Children in Edinburgh for a period of about sixteen and a half years. Out of 855 post-mortems, he found that 355 were for tuberculosis. Out of these 355 cases, thanks to the excellent manner in which the records had been kept, he was able with some certainty to make out the primary focus in 331 cases. Of these 331, 93, or 28·1 per cent, had to be referred to alimentary or ingestion tuberculosis. The result shows a remarkable agreement with that obtained independently by Dr. Still, and this circumstance increases its importance. In all these cases the bacilli had entered by some part of the alimentary tract, including the pharynx and tonsils.

Examining the condition of the intestine in relation to that of the mesenteric glands, the following results were brought out:—Forty-five cases showed ulceration of the intestines prior to affection of the glands; 12 showed caseation and matting of the glands apparently of older date than the condition in the intestine, the latter being quite healthy in 5 cases. (*Edin. Hospital Reports*, 1900.)

In this connection we must again quote Sims Woodhead: "In 14 out of 127 cases the mesenteric glands only were found affected. No tubercle was found elsewhere in the body. In these cases the glands had become calcified. There was no ulceration or cicatrisation in the intestine; no peritonitis; no secondary tuberculosis in any other part of the body; so that the tuberculosis in the mesenteric glands must be looked on as the primary lesion." (*Lancet*, 1888.)

Again, though Böllinger and others consider the intestine very resistant to infection, "the lymphoid structures in the alimentary tract are very vulnerable. They appear to be more readily denuded of their epithelium than other portions of the tract, and in addition, lymphocytes passing into them can take with them virulent tubercle bacilli" which give rise to ulceration in the lymphoid structures, or passing through them, to tuberculous adenitis of the mesenteric glands secondary to or preceding the intestinal ulceration. (Sims Woodhead, *loc. cit.*)

Bollinger also states that "tuberculosis of the peritoneal lymphatic glands occurs oftener in children than in adults, the cause for which is probably to be sought for in the feeding of young children with the milk of tuberculous cows."

After the meeting of the Tuberculosis Congress, the writer took the opportunity of examining his records at the Royal Hospital for Sick Children, Edinburgh, covering the period from June 1899 to August 1901, and found that out of 150 post-mortems 43 were for tuberculosis, and of these 11 or 25·6 per cent were alimentary in origin, thus affording still further proof of the accuracy of the former observations. He tried carefully to exclude any cases in which the etiology was in the least doubtful.

Adding these 43 cases to the 331 cases formerly reported, we have a total of 374 cases, and of these 104 or 27·8 per cent were alimentary in origin.

Other observers furnish somewhat similar

statistics. Dr. Carr out of 220 autopsies on tuberculous children at the Chelsea Children's Hospital found that 16·7 per cent showed evidence of primary abdominal infection. Dr. Guthrie (*Lancet*, 1899, i. p. 286), at Paddington Green Children's Hospital, found in a series of 77 cases that 26 per cent had been infected through the intestine. (Both quoted by Dr. Still, *Practitioner*, July 1901.) Kossel (*Zeitsch. für Hygiene*, Bd. xxi. 3, 59) found in 14 cases of tuberculosis in children, varying in age from one and a half to ten years, that the primary focus in 4 cases was in the mesenteric glands. There was no symptom of this during life.

Hueppe places the incidence of ingestion tuberculosis at 25 to 35 per cent.

We can therefore fall back upon a considerable mass of statistical evidence, and even allowing a margin for error, which, it must be remembered, is probably more than counterbalanced by the known fact that the prevalence of abdominal tuberculosis during life cannot be limited by post-mortem statistics, we have still very strong grounds to go upon in refusing to accept without further more positive proof Koch's statements with regard to this form of tuberculosis.

Clinical Evidence.—It is improbable that flesh, even from cattle with very extensive tuberculosis, causes the disease in more than a very small minority of cases.

"It is known that 'Knackers,' a class of people very liable to the consumption of tuberculous meat, suffer less from tuberculosis than the general population." (Kustner, *Munch. med. Wochenschr.*, 1889.)

It is also stated that Jews suffer less from tuberculosis than Christians, and this has been explained by their greater care in the selection of meat.

Tuberculosis of muscle is very rarely met with. From experiments Nocard holds "that muscles either destroy or digest injected bacilli of tuberculosis, or at least render them innocuous."

Whatever be the reason, the fact remains that muscle is markedly immune to tuberculosis. Possibly the explanation may not be unconnected with the continual unrest of this tissue, and to the fact that it is plentifully supplied with fresh oxygenated blood.

As Sidney Martin points out, meat is usually infected by the knife of the butcher, which cuts through broken-down caseous glands, and smears the tuberculous material over its cut surface. Experiments in which meat or meat-juice of tuberculous cattle has been inoculated into, or ingested by other animals, are generally productive of results so variable as to be untrustworthy; the bacilli, in the cases in which tuberculosis has resulted, being apparently derived from tuberculous foci in the intermuscular planes or in adjacent glands. Such experiments have been performed by Steinheil (*Munch. med. Woch.*, 1889), Sidney Martin (Royal Commission on Tuberculosis, 1895-6), and others.

It is quite another matter, however, where *Milk* is concerned, and much evidence in support of the virulence of tuberculous milk has been collected since Klebs and Gerlach first proposed, in 1868, that the milk of tuberculous cows may also be an agent of infection, and went so far as to hold it specially accountable for the tuberculosis of children.

Nocard summarises the question of infection by means of milk thus :—

1. "Milk of a tuberculous cow is virulent only when the udder is the seat of tuberculous lesions."

The udders are affected in from ½ to 2 per cent of tuberculous cows.

2. "Milk is dangerous only when it contains a large number of bacilli, and is ingested in large amount."—*The dose must be large.*

3. "In practice, this danger exists only in the case of individuals in whom milk is the exclusive or chief article of diet—for example, in infants and invalids.

4. "That, to avoid the danger, it suffices to boil the milk before use."

Bang has found that on churning most of the bacilli are carried off in the butter-milk (*petit lait*), but that a certain number remain always in the butter and cheese. Therefore Nocard believes that butter and cheese, even from an unknown source, may be eaten without fear, the number of bacilli being in any case small, and readily overcome in the alimentary tract. The common presence of acid-fast bacilli in milk and its derivatives must not be forgotten in connection with this question.

To sterilise the milk one need not expose it to a temperature of 100° C. Bang has found that exposure to a temperature of 85° C. for five minutes kills all the bacilli—they are often killed at 80° C.; by raising the milk to 70° C. for five minutes they are not killed, and can still give tuberculosis to animals if inoculated into the peritoneal cavity, but their vitality is so profoundly affected that they are quite incapable of causing tuberculosis in animals which ingest even large quantities of milk treated in this way. The same result is obtained by heating the milk to 65° C. for fifteen to twenty minutes.

While it is granted that bacilli are most numerous and virulent in the milk of cows whose udders are extensively diseased, it must be remembered that bacilli, apparently true tubercle bacilli, have been found in the milk of tuberculous cows in which the udders were healthy, and Benda has demonstrated them in the cells lining the lacteal ducts of such animals. In such cases the numbers present, particularly when the milk is mixed with the rest from the herd, are usually too small to cause any infection.

The bacilli in milk may be derived either

directly from the diseased cow or indirectly from the inspissated sputum of a phthisical patient, but in the latter case they can seldom collect in large quantities in the milk; and remembering that a large dose is necessary to convey the disease, the probability is that in most cases in which milk is to blame the crowds of bacilli have been derived directly from affected cows.

Many cases of infection in man from the use of tuberculous milk are reported in medical literature. Some of these are well known, for example those mentioned by Nocard and Ollivier, but a few less familiar cases may be noted.

Baum (*Lubarsch u. Ostertag*, 4 Jahrg. 1897, p. 868) describes several eases.

A fourteen-year-old girl of healthy parentage, quite healthy in childhood, died of tuberculosis of the larynx, ileum, and cæcum, slight of lungs. It was found that she had been drinking warm milk from a tuberculous cow.

Two children of healthy parents throve until weaned, and soon thereafter died of tuberculous meningitis. The milk given was from tuberculous cows.

Another child, six months old, of healthy parents, died of tuberculous meningitis. It had been brought up solely on milk from a tuberculous cow.

A boy, four years of age, died of tuberculosis of the abdominal glands, of the serous membranes, and of the lungs. He had been fed on warm milk from a tuberculous cow.

Johne (*loc. cit. idem*) gives a case in which one child, two and a half years old, who had been fed on tuberculous milk, died of acute miliary tuberculosis. Another child of the same family, who had not been exposed to the same infection, remained sound.

Ebers reports six cases, collected by several observers, of tuberculosis in children, attributed to the milk of tuberculous cows. (*Lubarsch u. Ostertag*, 1899.)

Bang, through inquiries in Denmark, has collected reports of nine persons in whom infection could be traced with reasonable certainty to milk from tuberculous cows. (Quoted by Strauss.)

Von Ruck reports the cases of a father and child which are very convincing, and says that he has observed several others in which there was very good reason to believe that milk was the agent of transmission of the tubercle bacillus. (*Jl. of Tuberculosis*, Ap. 1899.)

Klebs and Rievel have recently reported two cases which came under the observation of the former. A healthy young man employed by Klebs to assist in making some investigations on milk infection had the habit of drinking the milk of the tuberculous cows used in the experiment. In a few months he died of miliary tuberculosis. The second case was of a child who died at the age of two years of tuberculosis of the cord and meninges. This child was the only one out of six fed on cow's milk and the only one that developed tuberculosis. (*Deut. thierartz. Woch*, Jan. 18, 1892. Quoted by Ravenel.)

Similar cases might be multiplied.

Epitomising the case against milk, we may conclude that tuberculous milk can cause disease, but, as a rule, only in weakly individuals whose resistance is greatly lowered, and secondly, that such milk is dangerous only when the bacilli are present in great numbers or when it is ingested for a length of time.

Natural Infection by the Cutaneous Surface

It is well known that pathologists and assistants in post-mortem rooms often develop tuberculous warts on the fingers. It is also known that butchers develop similar warts, the infection having been received in cutting up the carcases of tuberculous cattle.

Veterinary surgeons are also occasionally inoculated in a similar way. Take, for example, the well-known cases reported by Pfeiffer and Tscherning.

Ravenel, in the paper in the University of Pennsylvania *Med. Bull.*, July 1902, formerly referred to, gives the four following additional instances:—

Dr. Kurt Mueller (*Zeitsch. f. Fleisch- u. Milchhyg.*, Feb. 1902), a surgeon of Erfort, describes the cases of two healthy young men who were under his care. Both were butchers, and in both cuts were sustained while working on tuberculous cattle, the wound opening the synovial sheath of a tendon in the hand. In each case an operation was necessary, and it was found that the wall of the sheath, as well as the tendon itself, were thickened, while upon them were large numbers of yellow nodules, proven to be tubercles by microscopical examination. These tubercles were placed very thickly near the scar, gradually becoming fewer as the scar was left. In one of the men the trouble extended from the finger to the forearm, and there was some evidence of tuberculosis even at the muscular attachment of the tendon. In the second case the tendon was attached to the sheath, and the disease limited to an area 10 cm. in length. Examination of the removed tissue proved the presence of tubercle bacilli. Both of these men had good family histories, and were free from tuberculosis elsewhere, so that Dr. Mueller has no doubt that they were inoculated with bovine tubercle bacilli.

De Jong (*La Semaine Méd.*, Jan. 15, 1902) observed the case of a man who injured his finger while examining the mesentery of a tuberculous cow. The wound did not heal, the edges became indurated, and considerable swelling ensued, with pain which increased steadily and failed to yield to ordinary treatment. Curettage

and cautery were finally used with success. In the scrapings tubercle bacilli were demonstrated.

In Berlin two men who are employed in the slaughter-house to carry the condemned tuberculous meat to the place where it is destroyed, have developed tuberculosis of the skin of the hands. At a recent meeting of the Medical Society of Berlin, Professor Lassar (*La Semaine Méd.*, Jan. 1, 1902) presented cases of verrucous tuberculosis of the skin. He had observed this type of skin tuberculosis in butchers who had handled tuberculous meat. Of 34 cases seen by him 4 were in butchers; while on the contrary, among those affected with other forms of tuberculosis localised to the skin of the hands, none belonged to this calling. Liebreich agreed with the view taken, and said he had been able to assure himself that verrucous tuberculosis of the skin was much more common among those whose duties require handling tuberculous meats than among others. Blaschko spoke of the case of a cook seen by him in whom a tuberculous lesion followed the prick of a piece of bone.

Drs. Joseph and Trautmann (*Deut. med. Woch.*, March 30, 1902) report three cases of verrucous tuberculosis of the skin occurring among employees of the municipal abattoir in Berlin, who worked exclusively with tubercular animals.

M. P. Krause (*Munch. med. Woch.*, June 24, 1902, quoted in *Gazette des Hôpitaux*, July 3) reports a case of infection through a skin wound.

A man, 30 years old, of robust constitution, with no family history of tuberculosis, a worker in a slaughter-house, had always enjoyed perfect health. Three years ago he injured the right thumb with a splinter of wood, and immediately afterwards had to skin a tuberculous cow. After several days the right arm commenced to swell, the swelling progressing slowly till it reached the axilla. He was treated by several physicians, all of whom diagnosed lymphangitis and tuberculous glands. A series of cold abscesses had to be opened. Examination of a gland and of a piece of ulcerated skin confirmed the diagnosis.

Lastly, the patient was examined at Professor Kast's medical clinique at Breslau. The lungs and all the other viscera were found healthy. In the right axillary fossa palpation revealed a small induration; below this were several indolent glands of the size of haricot beans. At the inner border of the biceps there was a gland as large as an almond, which was removed and submitted to microscopic examination. It contained tubercle bacilli and giant cells. One cannot doubt that in this case there was an infection from a bovine source.

It would seem, therefore, that infection through wounds is not so uncommon as was formerly supposed, and that bovine bacilli can apparently be implanted even in healthy tissues.

Conclusions. — Consideration of the whole subject leads us to conclude that the bacilli found in tuberculosis in man, cattle, other mammals, and most probably in birds, are closely related, most probably descendants of one original germ modified by successive passage through individuals of a certain species.

Bacilli by this successive passage through the same species have acquired a special virulence for animals of that species, and have lost their virulence to some extent for animals of any other species. Thus the bovine bacillus is more virulent for bovines and the lower mammals than for man, who is naturally insusceptible.

The three bacilli have gradually attained morphological and biological characteristics which are transmissible to their direct descendants, and distinguish them from each other.

Their characters, nevertheless, by suitable treatment can be approximated.

Bovine and human tuberculosis are, under favourable conditions, intercommunicable. "Therefore, bovine tuberculosis is a menace to the health of man; to what extent we cannot at present positively define, but that it does really exist cannot be denied. There has been exaggeration in the past, but however great this may have been, it does not now justify any attempt at belittling the risk, and it is folly to blind ourselves to it" (Ravenel).

Infants, those of early ages, and weaklings are specially liable to be infected by bovine tubercle bacilli, and chiefly through the agency of milk. The statistics bearing upon the increased frequency of tuberculosis in infants, and upon the relative frequency of ingestion tuberculosis in children, must be accepted as fairly conclusive evidence on this point. Even if it could be shown that tuberculosis was not conveyed by milk, the precautions taken in the past have had such an important result in lessening the mortality from a still more fatal disease—diarrhœa —that we are justified in continuing them.

Until the question is finally decided, the duty of guarding our milk supply is laid as heavily upon us, and no relaxation can be allowed in our attempts to obtain a healthy supply, particularly for children.

Tuberculotoxine.—A toxine developed by tubercle bacilli.

Tuberculum. *See* Tubercle; Tongue (*Development, Tuberculum Impar*).

Tuberiform Abscess. *See* Axilla (*Diseases, Superficial Inflammations*).

Tuberose.— Resembling a tuber, or rounded swelling, *e.g.* a tuberose mole, or one covered with wart-like prominences. *See* Pregnancy, Ovum and Decidua (*Fleshy Mole*).

Tuberosity.—A projection, usually more

or less rounded, from a bone such as the humerus or ischium.

Tubo-.—In compound words *tubo-* means relating to a tube and specially to the Fallopian tube, e.g. *tubo-ovarian* (related to Fallopian tube and ovary), *tubo-peritoneal*, *tubo-uterine*, etc.

Tubule.—A small tube or cylindrical canal, *e.g.* the tubules of the kidney, of the mammary glands, of the testicles, etc.

Tuffier's Disease or Syndrome.—A state of tissue debility, generally congenital, which results in various displacements of organs and parts. *See* ENTEROPTOSIS; UTERUS, DISPLACEMENTS (*Prolapse*).

Tufnell's Treatment.—The treatment of aneurysm by rest and a restricted diet, with the purpose of diminishing the total amount of blood and of making it more fibrinous.

Tugging, Tracheal. *See Cross References* under TRACHEAL TUGGING.

Tulpius, Valve of.—The ileo-cæcal valve. *See* INTESTINES, DISEASES OF (*Anatomical Considerations*).

Tumours.

INTRODUCTORY REMARKS 152
CLASSIFICATION 153
CONNECTIVE-TISSUE TUMOURS—
Lipomata 153
Chondromata 153
Osteomata 154
Odontomes 154
Myelomata 155
Sarcomata 155
Angeiomata 157
Lymphangeiomata 158
Endothelioma 158
Myomata 158
Neuromata 159
THE EPITHELIAL GROUP—
Papillomata 160
Adenomata 160
Carcinomata 160
DERMOIDS—
Sequestration Dermoids . . . 162
Tubulo Dermoids 162
Moles 163
Ovarian Dermoids 163
Teratomata 163
PHANTOM TUMOUR 163
INOPERABLE TUMOURS 164
TUMOURS OF THE SKIN 168

See also ABDOMINAL TUMOURS, DIAGNOSIS OF; ACTINOMYCOSIS (*Microscopical Anatomy*); ADRENAL GLANDS (*Tumours in*); APPENDIX VERMIFORMIS (*Symptoms, Tumour*); AXILLA (*Tumours*); BLADDER, INJURIES AND DISEASES OF (*Tumours*); BONE, DISEASES OF (*Tumours*); BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF (*Pressure*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Hæmorrhage, Diagnosis*); BRAIN, TUMOURS OF; BRAIN, CYSTS OF; BRAIN, SURGERY OF (*Cerebral Tumours*); BRAIN, CEREBELLUM, AFFECTIONS OF (*Tumours*); BROAD LIGAMENT, DISEASES OF (*Tumours*); BURSÆ, INJURIES AND DISEASES OF (*Neoplasms*); CAPILLARIES, DISEASES OF (*Angioma, etc.*); CHEST-WALL, AFFECTIONS OF (*Tumours*); CHOROID, DISEASES OF (*Gumma, Sarcoma, etc.*); CICATRICES (*Keloid*); COLON, DISEASES OF (*Malignant*); CONJUNCTIVA, DISEASES OF (*Tumours*); CORNEA (*Tumours*); DIAPHRAGM, MEDICAL AFFECTIONS (*Tumours*); EAR, AFFECTIONS OF TYMPANIC MEMBRANE (*Tumours*); EAR, EXTERNAL, DISEASES OF (*Exostosis, Epithelioma*); EYELIDS, AFFECTIONS OF (*Growths*); FALLOPIAN TUBES (*Tumours*); GALL-BLADDER AND BILE DUCTS, DISEASES OF (*Tumours*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Morbid Processes, Tumours*); HIP-JOINT, DISEASES OF (*Tumours near Hip*); HYSTERIA, SURGICAL ASPECTS OF (*Tumours*); INTESTINES, DISEASES OF (*Malignant Growths*); JOINT, DISEASES OF (*Tuberculous, Tumor Albus*); JOINTS, DISEASES OF (*Tumours and Cysts*); KIDNEY, SURGICAL AFFECTIONS OF (*Tumours*); LABOUR, PRECIPITATE AND PROLONGED (*Tumours of Ovaries, Uterus, etc.*); LACRIMAL APPARATUS, DISEASES OF (*Tumours of Gland*); LARYNX, BENIGN GROWTHS; LARYNX, MALIGNANT DISEASE; LARYNX, LARYNGEAL PHTHISIS (*Tumours*); LIVER, DISEASES OF (*Innocent Tumours, Malignant Disease*); MAMMARY GLAND, DISEASES OF (*Neoplasms*); MEDIASTINUM (*Growths*); MENINGES OF THE CEREBRUM (*Tumours*); MOUTH, DISEASES AND INJURIES OF MOUTH AND JAWS (*Tumours*); NECK, REGION OF (*Cysts, Cystic, and Solid Tumours*); NERVES, PERIPHERAL (*Tumours*); NOSE, DISEASES OF NASAL ORIFICES AND SEPTUM (*Benign and Malignant Tumours of Nasal Fossæ*); NOSE, POST-NASAL ADENOID GROWTHS; ŒSOPHAGUS (*Growths*); ORBIT, DISEASES OF (*Tumours*); OVARIES, DISEASES OF THE (*Cystic and Solid Tumours*); OVARIES, DISEASES OF (*Broad Ligament Cysts*); PANCREAS, DISEASES OF (*Malignant*); PAROTID GLAND, DISORDERS OF (*Tumours*); PENIS, SURGICAL AFFECTIONS OF (*New Growths*); PERICARDIUM, DISEASES OF (*New Growths*); PERITONEUM (*New Growths*); PLEURA, DISEASES OF (*Malignant*); PLEURA, SURGICAL AFFECTIONS OF (*Tumours*); PREGNANCY, DISEASES OF PLACENTA AND CORD (*Tumours*); PREGNANCY, HÆMORRHAGE IN (*From Tumours*); PROSTATE GLAND (*Tumours, Simple and Malignant*); RECTUM, DISEASES OF (*Cancer*); RETINA AND OPTIC NERVE (*Glioma of; Retina and Tumours of Optic Nerve*); SCROTUM AND TESTICLE, DISEASES OF (*Tumours of Scrotum and Testicles*); SHOULDERS, DISEASES AND INJURIES (*Tumours*); SPINAL CORD, MEDICAL (*New Growths*); SPINE, SURGICAL AFFECTIONS OF (*Malignant Disease*); SPLEEN, MEDICAL AFFECTIONS OF (*Tumours*); STOMACH AND DUODENUM, DISEASES OF (*Cancer*); THYROID GLAND, SURGERY

OF (*Goitre, Tumours*); TONGUE (*Tumours*); UNCONSCIOUSNESS (*Causes, Tumours of Brain*); URACHUS (*Tumours*); UTERUS, NON-MALIGNANT TUMOURS OF; UTERUS, MALIGNANT TUMOURS OF; UVULA, DISEASES OF (*New Growths*); VAGINA, DISORDERS OF (*New Formations*); VESICULÆ SEMINALES (*Cysts and Malignant Disease*); VULVA, DISEASES OF (*Cysts and Tumours*); WARTS AND VERRUCÆ.

ONCOLOGY is a branch of pathology which is particularly concerned in the investigation of the structure (histogenesis) and causes (pathogenesis) of those abnormalities of animals and vegetables known as tumours. The term tumour is very ancient and only signified a "swelling," but the careful microscopic investigations conducted by histologists with the aid of differential staining of the tissues, and the study of the relations of micro-organisms to those swellings called tumours has led to a revolution of our knowledge, and gradually the term has been stripped of its former wide significance; although in clinical work the term is not likely to completely disappear, it has lost its importance to the pathologist. Even with our modern knowledge it is impossible to frame a definition of a tumour, although we set very clear limitations to the use of the word.

In this article the conditions which will be described under the heading "Tumours" are local irregular swellings of the connective-tissue system (fat, cartilage, bone, etc.), which are not due to recognised infections such as tuberculosis, syphilis, glanders, actinomycosis, and the like; abnormal conditions of secreting glands, skin, and mucous membrane known as adenomata and cancer; and the remarkable group known as dermoids. Each of these groups will receive separate consideration. One of the earliest classifications of the heterogeneous formations termed tumours, arranged them into a malignant and an innocent division. The malignant tumours inevitably destroyed life, whereas innocent tumours did not display such destructive propensities. Although this division was founded solely on clinical observation it is sound, because tumours belonging to the connective-tissue group, as well as those of the epithelial group which display malignancy exhibit, in many instances, structural characteristics which enable competent histologists to recognise them; thus the two terms innocency and malignancy, as applied to tumours, are of the greatest significance in relation to clinical medicine. The most satisfactory method of classifying tumours would be to take their cause as a basis, but as we are absolutely ignorant of the pathogenesis of tumours, we have to rely on their structure for taxonomic purposes. It is noteworthy that whenever the cause of a disease characterised by tumour-formations is detected, that particular disease is removed from the list of tumours; one of the most striking instances of this is the parasitic disease known as actinomycosis, which used to be classed with the sarcomata. The study of the minute structure (histology) of tumours led to a great increase of knowledge concerning them, the earliest investigators in this field being J. Müller and Virchow.

The use of animal bodies for food induced men to apply names to the various parts, so that in the vulgar tongue bone, skin, fat, gristle, and the like are thoroughly familiar structures. The anatomist speaks of nerves, muscle, tendon, artery, and so forth; but the discoveries of the microscope revealed that the body is composed of fundamental tissues, which enter into the composition of organs of the most diverse form and function. The basis of animal bodies consists of what is known as connective tissues which comprise bone, fat, cartilage, fibrous, muscular, and nervous tissue, and a peculiar material which permeates the body and enters into the composition of every organ, and is known as connective tissue. This ubiquitous web is stout and strong where it forms fascia and periosteum, but extremely delicate in the nervous system, and reaches such extreme fineness in the retina that it needs careful preparation to render it perceptible even to the microscope. The connective tissues practically form the framework of the body, and constitute a sort of sustentaculum in the compound organs such as the intestines, liver, kidney, and the like for the support of epithelium, and as a mesh in which blood-vessels and lymphatics can ramify to supply the liquid tissue (blood), from which the cells can obtain material to form the secretion characteristic of the particular gland to which they belong. The careful and critical study of the minute (microscopic) structure of tumours revealed that they were composed of material exactly like that of the normal tissues of the body, and pathologists began to realise that the structure of a primary tumour invariably corresponded with the tissue in which it arose; for example, an osseous or bony tumour arises from the bone, a fatty tumour from fat, a cartilage tumour from cartilage, and so on. This is known as the "law of prototypes," and a careful study of it enables pathologists to predict the various genera of tumours to which an organ may be liable, as well as to explain why primary epithelial tumours do not grow in bones and muscles, or myelomata and osteomata in the liver, kidney, or brain. It is important to indicate that although the tissues of an organ determine the species of tumours to which it is liable, their relative frequency can only be gathered from observation. It is also curious how organs vary in their liability to tumours; for instance, the heart is rarely occupied by a tumour, but the uterus, also a muscular organ, is with extreme frequency infested with fibroids; whilst

voluntary (skeletal) muscles are rarely attacked by tumours of any kind, and they never arise from the true contractile tissue.

CLASSIFICATION. — Tumours form three groups:—

1. Connective-tissue tumours.
2. Epithelial tumours which comprise adenomata and cancers.
3. Dermoids.

Each group contains a certain number of genera, and each genus has one or more species; in describing the various genera care will be taken to define their structure, distribution, and clinical peculiarities, as well as the modes by which they may imperil life, because it must be borne in mind that a so-called innocent tumour may be as rapidly fatal as the most malignant tumour should it arise in, or involve, a vital organ. This is a matter depending on environment, and will receive careful consideration with each genera.

GROUP I.—CONNECTIVE-TISSUE TUMOURS

Genera.—1. Lipomata (fatty tumours). 2. Chondromata (cartilage tumours). 3. Osteomata (bony tumours). 4. Odontomes (tooth tumours). 5. Myelomata (marrow tumours). 6. Sarcomata. 7. Angeiomata (blood-vessel tumours). 8. Lymphangeiomata (tumours from lymphatics). 9. Myomata (muscle-tumours). 10. Neuromata (tumours connected with nerves and brain).

Lipomata.—These are tumours composed of fat. The genus contains a single species with a distribution as wide as the fatty or adipose tissue.

The common situation in which to find lipomata is the subcutaneous fat of the trunk and trunk-end of the limbs; they occur as encapsuled irregularly lobulated lumps of fat, more or less adherent to the skin. In the majority of instances one tumour is present, but as many as twenty or more may grow concurrently. A lipoma weighing sixteen ounces is a tumour of fair size, but it may attain twenty times this dimension. In many instances the very large tumours which have been reported as fatty were examples of molluscum fibrosum, and some examples in which they were particularly numerous, the disease was an example of general neuro-fibromatosis. Subcutaneous lipomata sometimes grow on the fingers, the palms, soles, and occasionally on the scalp. They have been known to exist on one individual for more than thirty years, and in a few exceptional instances the fibrous septa among the fatty lobules have calcified; in a few big tumours the fat has undergone saponification. The subcutaneous fat sometimes undergoes an irregular overgrowth, and forms what is known as *diffuse lipomata*. Precisely similar changes arise in the fatty stratum which underlies serous membranes such as the peritoneum and pleura. The fat beneath the serous covering of the intestines is occasionally the source of lipomata, which sometimes invade the muscular coat of the bowel and project into its lumen as a polypus. Such lipomata have, in a few instances, led to fatal intestinal obstruction.

The synovial membranes of joints possess a fatty layer, and lipomata occasionally arise therein and project as stalked bodies into the cavity of the joint.

Submucous tissue contains fat which serves as a source of lipomata. They have been found beneath the gastric mucous membrane, as well as in the lips, beneath the conjunctiva, and in the glosso-epiglottic folds.

Lipomata also arise in the adipose tissue between muscles, as well as in the belly of a muscle such as the biceps or the deltoid. Occasionally they spring from the sheath of nerves and the periosteum of bone. The connective tissue between the dura mater and the bony wall of the spinal canal contains fat, which has, in a few carefully recorded instances, furnished a lipoma; but probably one of the rarest situations in which a fatty tumour has been found, was between the layers of the dura mater lining the sella turcica. The patient, a woman forty-four years of age, suffered from periodical pain in the head and ptosis; the tumour was as big as a hen's egg.

Relation to Age.—Lipomata may arise at any age,—infancy, adolescence, and extreme old age. Occasionally they are congenital.

Clinical Characters. — The subcutaneous variety of fatty tumour rarely gives rise to difficulty in diagnosis, its intimate relation to the skin, doughy consistence, and the indefiniteness of its boundaries are very characteristic features. Lipomata in unusual situations often constitute clinical puzzles, especially when seated in or between muscles, or in the tongue; whilst those connected with periosteum often simulate sarcomata.

Lipomata never recur if completely removed. They are only dangerous to life when they arise in the cranium or spinal canal; or when they grow in the walls of the small intestine and protrude into its lumen and cause intussusception.

Chondromata.—This genus consists of tumours composed of hyaline cartilage. The most typical examples occur in long bones, and especially in relation with the epiphysial cartilages, hence the chondromata are more frequent in children and adolescents. They occur as distinctly encapsuled tumours, and often form deep cup-shaped hollows in the bone from which they grow. They are particularly liable to arise in the metacarpal, metatarsal, and phalangeal bones, and the frequency with which chondromata occur in the bones of those who were rickety in early life, had led to the suggestion that they probably arise in the untransformed islets of cartilage found in the immediate neigh-

bourhood of rickety epiphysial cartilages. When multiple, chondromata rarely grow big, but a single chondroma in the head of the tibia or the mesethmoid will sometimes attain formidable proportions. Cartilage tumours may calcify and ossify; frequently they undergo mucoid softening.

Ecchondroses.—This is a species of chondroma which occur as small local overgrowths from the edges of articular cartilage, the cartilages of the larynx and the triangular cartilage of the nose.

Loose Cartilages.—This species of chondroma appears in the synovial cavity of joints, where they occur as pedunculated bodies hanging from the membrane, or loose in the joint. In many instances they are due to the chondrification of the synovial fringes; in others they are detached ecchondroses. A joint may contain one loose cartilage and the opposite joint is also occupied by one of similar size and shape. In rare instances, two hundred loose bodies have been found in one joint. Perhaps the most extraordinary situations in which chondromata arise are the parotid, submaxillary, and lacrimal glands. Here they violate all histological rules. This will be referred to when dealing with sarcomata.

Clinical Characters.—The usual situations for these tumours are the bones of the limbs in children and adolescents; they are distinctly circumscribed, and often so hard as to resemble osteomata. When they occur in the skull bones, then the diagnosis is difficult. They are only dangerous to life when they grow in the skull and compress the brain, or in a vertebra and squeeze the spinal cord.

Loose bodies in joints impair the utility of the limb and cause pain, but they are easily and safely removed.

Osteomata.—These are tumours composed of bone, and may be defined as ossifying chondromata, for every growing cancellous osteoma has a cap of hyaline cartilage which has the same relation to it that an epiphysis bears to a long bone.

This genus contains two species:—1. Compact osteoma. 2. Cancellous osteoma.

Compact osteomata are in structure identical with the hard tissue which forms the shaft of a long bone. They grow most commonly from the bones of the skull, especially in the neighbourhood of the external auditory meatus, the orbits, and from the angle of the mandible.

Cancellous osteomata grow from any bone and resemble the cancellous tissue of bone. They may be pedunculated or sessile. A single osteoma is the rule; often they occur in pairs and are bilaterally symmetrical. Multiple cartilage-tipped osteomata are occasionally met with in children; they are congenital, hereditary, and fairly symmetrical so far as position is concerned.

Clinical Characters.—Osteomata are easily recognised on account of their extreme hardness and in being localised to bones; they rarely cause pain except when growing in the vicinity of, and pressing upon, the trunks of nerves. Osteomata near the external auditory meatus will occasionally induce deafness, and this becomes serious should the tumours be bilateral. An osteoma growing from the odontoid process has been known to fatally compress the spinal cord, and large osseous tumours of the facial bones will produce hideous deformity and occasionally destroy the eyeball.

It is a remarkable fact that occasionally in cases of congenital multiple osteomata, one of the cartilage-covered tumours grows, disseminates, and destroys life like a sarcoma.

In clinical as well as in pathological writings care should be taken to keep a wide distinction between the—(*a*) ossification of tendons; (*b*) calcification of inflammatory exudations; and (*c*) the subungual exostosis. These are not true osteomata, but, like cervical ribs, supracondyloid ridges, and third trochanters, are often loosely classed as osteomata.

Odontomata.—These are tumours composed of dental tissues in varying proportions and different degrees of development, arising from teeth-germs or teeth in the process of growth.

This constitutes the most complete genus of tumours, and the various species are determined according to the part of the tooth-germ concerned in their formation. Those that arise from the enamel-organ in its early stages are soft and known as epithelial odontomes, whilst those which are known as follicular odontomes are due to aberrations of the follicle when the tooth is completed and ready to erupt.

Occasionally the follicle itself becomes converted into an encapsuled mass of fibrous tissue, in which a rudimentary or a badly formed tooth is embedded: this is a fibrous odontome and is, in some individuals, associated with rickety changes in the skeleton. When fibrous odontome ossify the hard tissue resembles cementum, and the tumour is called a cementoma. Sporadic dental changes in a fibrous odontome produce very curious results whereby the tumour contains numerous fragments of hard dental tissues—denticles; such are known as compound follicular odontomes. When the changes affect the roots of teeth after the crown is completed, the tumour is called a radicular odontome, and when it implicates the crown, neck, and roots, and produces an ill-formed lump of cementum, dentine, and enamel intermixed in an irregular way, it is called a composite odontome.

Clinical Characters.—The germ of any of the permanent teeth may become converted into an odontome, and occasionally two or more teeth may be involved in the same tumour. They occur with equal frequency in the upper and lower jaws, and the cystic species (follicular odontomes) may occur in multiples and attain

great dimensions. Of the hard species the composite odontome is the most frequent, and in the maxilla such a tumour may invade the antrum and grow to the size of a hen's egg. Many of these large odontomes have been erroneously described as exostoses. In the mandible they rarely exceed the size of a dove's egg. A singular and important clinical fact in connection with the hard varieties is this: they rarely cause trouble until like teeth they begin to erupt, and this phenomenon generally happens between the twentieth and twenty-fifth years, and this draws attention to them. They are generally diagnosed as dead bone. In the past, operations unnecessarily severe have been undertaken for their relief.

Myelomata.—This genus consists of tumours composed of tissue identical in structure with the red marrow of young bone. These tumours were formerly classed as myeloid sarcomata, but they differ in many points from sarcomata, and above all things in the fact that they are not malignant.

A myeloma arises in the cancellous tissue of bone. When fresh its cut surface is deep red, and looks like a piece of fresh liver and is very vascular. Microscopically this tissue abounds in multinuclear cells (giant cells) embedded in round or spindle cells. The giant cells are so abundant as to constitute the greater proportion of the tumour. The distribution of the myelomata is that of red marrow, but they exhibit a striking preference for certain bones. In the lower limb the tibia, especially its head, is the most frequent situation, and the lower end of the radius is the common situation in the upper limb. In the head the body of the mandible has the preference. Myelomata have also been observed in the clavicle, humerus, and ulna, the femur and patella, and the jaws.

Clinical Characters.—Myelomata are rare tumours after the age of twenty-five. They grow slowly, and very gradually expand the bone immediately investing them, and occasionally it becomes so thin as to crackle on pressure. The soft tissue of the tumour sometimes perforates its capsule, and pulsates synchronously with the beat of the heart. These tumours do not infect lymph glands or disseminate; and if removed before the tumours perforate the capsules they do not recur. This fact is important in regard to treatment, for instead of amputating limbs to cure them, surgeons now excise the affected section of the bone whenever this is possible and leave the patients' useful limbs. Successful treatment of this kind has been carried out in the ulna, radius, humerus, clavicle, and even with the head of the tibia. Paget proved that some of the myelomata may be enucleated or scraped out of their capsules, and his example has in several instances been successfully followed.

Myelomata are rare tumours, and a careful perusal of the periodical literature of hospital reports shows that at each of the eleven large hospitals in London one myeloma a year is above the average.

Sarcomata.—The term sarcoma is applied to any connective-tissue tumour which exhibits malignant characters. It will therefore be useful to briefly describe the structure of these tumours, and then relate the manner in which they betray their malignity.

Sarcomata are named according to the prevailing type of the cells composing them; thus the round-celled kinds consist almost entirely of round cells, each possessing a large round nucleus which is always conspicuous in stained sections. There is very little intracellular substance; blood-vessels are abundant, but lymphatics are absent.

The round-celled sarcoma is the most generalised tumour that affects human beings; it occurs in bone, muscle, nerves, glandular and muscular organs, ovary, testis, thymus, adrenal, and even in the delicate tissue of the retina. It may grow at any period of life, and even attacks the unborn child.

One variety of the round-celled sarcoma reproduces the delicate structure of a lymph gland. This is known as lymphosarcoma, and arises in definite situations where this peculiar tissue abounds, *e.g.* the tonsil, root of tongue, superior mediastinum, and the retro-peritoneal tissue.

The spindle-celled sarcomata are composed of cells resembling spindles, and though the individual cells of a tumour display extraordinary uniformity, there is great diversity in the size of the cells in different tumours. In some the cells are small and oat-shaped, others are fusiform, or they may be extremely attenuated, and occasionally the cells are large and plump. In some situations spindle-celled sarcomata contain tracts of immature hyaline cartilage, these are termed chondrifying sarcomata. But the most extraordinary feature found in these tumours is the occasional occurrence of spindles with a tranverse striation like that which is met with in the young muscle cells. These are known as myosarcomata, and are found in the most unexpected situations, such as the testis, sinus of the kidney, and the neck of the uterus, where muscle cells of the striped variety do not occur under normal conditions.

The presence of hyaline cartilage in sarcomata of secreting glands like the parotid and the mamma, and the occurrence of tissue indistinguishable from striated muscle tissue in the testis and kidney, and also in lipomata of the periosteum and in the spinal canal, has never received an adequate explanation, although it has thoroughly aroused the curiosity of pathologists and led to much unsound speculation.

One of the most striking species of the sarcomata is that to which the adjective melanotic is applied on account of its blackness. This is

due to the presence of a pigment called melanin. The melano-sarcomata may consist of spindle or round cells, and always arise from pigmented tracts of skin, and especially the black moles and congenital pigmented warts; but the common situation is the uveal tract of the eyeball. The colour of melano-sarcomata varies greatly; some only contain faint traces of pigment, whereas others are inky black.

There is probably nothing more remarkable in the whole of Oncology than these pigmented tumours; an individual may have an area of the skin less than an inch square occupied by a black mole which was present at birth. This has never caused any inconvenience, and then when he has arrived at the age of fifty or perhaps seventy it begins to enlarge, it may even pulsate; then the adjacent lymph glands are noticed to be enlarging, and in a few months he has melanotic nodules in skin, lungs, liver, and other organs, and daily discharges large quantities of the pigment melanin in the urine. Of the cause of this mysterious condition nothing—absolutely nothing is known. There is an excessively rare variety of melanotic tumour which occurs in the eyeball, arising from the ciliary glands detected by Teacher Collins. It has the characters of a typical carcinoma.

Melano-sarcomata illustrate, too, a fact in connection with the tissue prototypes of tumours. The connective tissue underlying a mole has a characteristic alveolar arrangement, and this is also manifested in many of the tumours, and by some writers has been made into a separate species as alveolar sarcoma.

Sarcomata are distinguishad from the other chief genera of connective-tissue tumours in that they rarely possess capsules, and when they do it is generally a spurious encapsulation depending on environment, such as when they arise in the eyeball, medullary cavity of a bone, or in the sinus of the kidney. Having no restraining capsule they are able to infiltrate the adjacent tissues.

The vascularity of sarcomata varies; in all the circulation is mainly capillary, and the very soft, quickly growing and badly organised round-celled species often have such an abundant supply of arterial blood that they distinctly pulsate.

Sarcomata of the spindle-celled kind grow slowly, and are not so rich in blood-vessels; but all the species lack lymphatics. The intimate relation of sarcomata to the blood-vessels has an important relation to the distribution of sarcoma cells to other parts of the body; for example, the cells from the tumour find their way into the venules which return the blood into the venous trunks and are directly conveyed as minute emboli to the lungs; these cells are arrested in the pulmonary capillaries and they act as grafts, retain their vitality, and form what is known as secondary nodules or deposits. There is no limit to the number of these secondary deposits, and a hundred or more may be counted in the lungs. When the primary sarcoma is situated in a part from which the venous blood enters the portal circulation the emigrant cells from the tumour are received by the capillaries of the liver, and thus the secondary nodules are found in the territory of the portal circulation and especially in the liver. This remarkable phenomenon is known as dissemination, and it is one of the most dangerous features displayed by malignant tumours.

The fact that sarcomata are usually unencapsuled allows the cells of the tumour to permeate or infiltrate the adjacent tissues, and this peculiarity becomes extremely dangerous when the sarcoma arises in the midst of important structures. Thus in the mediastinum an infiltrating tumour may involve the œsophagus, trachea, lung, pneumogastric nerves, pleura, pericardium, vena cava or the aorta. A sarcoma of the kidney is very liable to creep into the renal vein, and a prolongation of the tumour will extend into the inferior vena cava; a sarcoma of the thyroid gland may open into the trachea or the internal jugular vein.

This infiltrating property of a sarcoma, apart from its effect upon neighbouring organs, has an important bearing on treatment. For example, when the surgeon attempts to remove a sarcoma effectually it is of the utmost importance to cut well wide of it, for if small portions of the tumour are left the sarcoma grows again from these remnants. When the tumour returns in this way it is spoken of as recurrence, and this is a common mode by which sarcomata manifests malignancy. There are few things which demonstrate the deadly nature of some of these tumours better than a sarcoma springing from the periosteum of the femur. A youth will present himself with one of these tumours in the lower end of his bone, and it may be about the size of a goose's egg. The limb is amputated at the hip joint, and in a few months the sarcoma reappears in the stump and grows in a few months into a large bleeding mass and quickly destroys him. It is not an uncommon event for a sarcoma to appear in connection with the femur or the humerus and destroy life either by dissemination, or by recurrence after operation within a year of its detection.

Although sarcomata as a genus are ubiquitous, nevertheless they occur in some situations more frequently than in others, and present some striking age-relations that are worth careful study.

Take for example the eyeball: this sense organ is liable to two very distinct kinds of sarcoma. Before the age of twelve years, sarcomata arise in the retina and possess the minute structure of the sustentacular framework of the retina as it exists in the embryo.

These tumours often attack both retinæ, and though they rarely disseminate they are extremely prone to recur after removal of the eyeball. The adult is liable to sarcomata which arise in the uveal tract, and is almost invariably of the pigmented (melanotic) species. It is always unilateral, and though it is not so liable to recur locally as retina sarcoma, it is dreadfully liable to disseminate and infect almost every organ in the body, and occasionally to dye the whole skin black. The proneness with which sarcoma attacks bilateral organs during infancy and childhood is somewhat remarkable, and is especially manifest in the retinæ, kidneys, ovaries, and adrenals. In quite a large number of instances sarcoma attacking these organs during infancy may be limited to one gland, but in a fair proportion of cases both are affected. This bilateral disposition is not seen when sarcoma arises in these parts in adults; not only is the disease unilateral, but it exhibits, as has already been stated in regard to the eyeball, diversity in its minute structure.

The variations in the liability of different organs to sarcomata is not greater than the variations observed among organs of similar structure and functions. Take for instance the salivary glands. Sarcoma of the parotid gland is quite common: it is rarely seen in the submaxillary and lachrymal glands, and is equally rare in the pancreas.

Sarcomata occur far more frequently in the structures of the lower limb than in those of the upper. This is certainly the case in regard to bones. Sarcomata are rare in voluntary muscles and in nerve-trunks, and the majority of the recorded cases, excluding the optic nerve, have been observed in the muscles and nerves of the lower limb.

The ovaries and the uterus are organs closely associated in functions, but sarcomata are frequent in the ovaries but unusual in the uterus.

Lastly, there is the important question, can an innocent connective-tissue tumour change its character and become malignant? In the majority of instances the facts indicate the insufficiency of our means of distinction rather than an actual metamorphosis of tissue.

A long study of the histogenesis of tumours has convinced the writer that the clearly innocent and the decidedly malignant tumours present distinct histological features, but there are intermediate varieties which cannot be sharply defined in relation to these points, and this comes out in a very striking and suggestive way when an individual possesses supposed innocent tumours in multiples. For example, take uterine fibroids when they are multiple; tumours may be selected from one uterus of which some are so hard as to need a saw to divide them and others so soft as to be almost diffluent; such myxomatous tumours will recur after enucleation: and careful records are accessible in which fibroids of simple structure have disseminated and destroyed life. This is true of multiple and hereditary cartilage-clad osteomata; and also true of the nodular form of molluscum fibrosum.

It is so difficult to decide between the slow-growing spindle-celled sarcomata, the fibrifying sarcoma, and the gelatinous myxoma, that it is safer to include myxomata and the hard tumours called fibromata with the sarcomata until distinctive methods have been detected by the histological chemist, biologist, or the bacteriologist.

These difficulties are further exemplified by the peculiar but excessively rare disease known as *chloroma* or green cancer, in which sarcoma-like masses form on the bones of the skull and face, especially in the neighbourhood of the orbits, and secondarily infect other organs. After death the colour of the tumour-like masses is, on section, grass green. The nature of the disease is excessively obscure: some writers regard it as a form of leukæmia. It has lately been carefully studied by Melville Dunlop (*British Medical Journal*, 1902, vol. i. p. 1072).

Angeiomata.—These are tumours composed of an abnormal formation of blood-vessels. The genus contains three species: 1. Simple nævus. 2. Cavernous nævus. 3. Plexiform angeioma. The simple nævus is of very common occurrence and appears in the skin or mucous membrane as a patch of discoloration, which may be pink or deep blue. When the nævus is due to aberrations of venules it will be blue, but when arterioles preponderate it will be pink. The vessels of the nævus are embedded in fat, and a few lymphatics are also present. Nævi vary in size: some are scarcely perceptible, whilst others may involve the skin of half the trunk or a whole limb. They may occur in multiples and in any part of the skin, lips, labia, conjunctivæ, or tongue. Small nævi tend to disappear, some remain stationary and a few become converted into cavernous nævi. These are sometimes called erectile tumours, because they structurally resemble the cavernous tissue of the penis. These tumours are common in the skin, and they have been observed in the rectum, subperitoneal tissue, liver, bladder, the larynx, wall of the right auricle of the heart, and in voluntary muscles such as the deltoid, the semimembranosus, and the gracilis.

The plexiform angeioma or "aneurysm by anastomosis" consists of a number of abnormal blood-vessels of moderate size arranged parallel to each other, as in the retia mirabilia in the fore-limbs of sloths. Some of the plexiform angeiomata consist of arteries only, others of veins and arteries. Often the vessels are very tortuous. Fortunately this species of angeioma is rare: it has been observed on the scalp, forearm, and in the perineum surrounding the urethra.

All angeiomata are congenital, or arise in the first few weeks after birth; it is instructive to watch a small red fleck, at first no larger than a split pea, slowly spread and then begin to stand above the level of the skin: a few months later it may form a raised oval pulsating lump, which may be red or blue, and at last become a tumour demanding some courage as well as skill on the part of the surgeon to deal with it. Occasionally a simple nævus will be converted into a cavernous nævus, and the central parts of the latter fuse and form a central space so that the tumour resembles a cyst with very vascular walls. Such have been called nævoid cysts.

Lymphangeiomata.—These are tumours composed of an abnormal formation of lymphatics. They bear the same relation to lymph vessels that angeiomata bear to the hæmic capillaries. This genus contains three species: 1. Lymphatic nævus. 2. Cavernous lymphangeioma. 3. Lymphatic cyst.

The lymphatic nævus is, as a rule, colourless, but as it contains hæmic capillaries it may be of a pale pink. In the tongue the lymphatic nævus is sometimes of a faint yellow or serum colour. Sometimes a lymphatic nævus will affect a tract of mucous membrane like that of the tongue and produce the condition known as macroglossia, or it may affect the lips and cause macrochelia. Cavernous lymphangeioma resembles in structure and distribution the cavernous nævi, and differs from them only in containing lymph instead of blood.

Lymphatic cysts have been described under a variety of names, and the most familiar examples are those known as "hydrocele of the neck." They appear as congenital translucent cystic swellings, and though most common in the neck, are also found in the axilla and on the anterior and posterior wall of the thorax. These cysts are filled with lymph and the walls are composed of very vascular nævoid material. The most remarkable fact in connection with them is the great tendency they exhibit to shrivel and disappear. They are very prone to inflame, and this event generally precedes their disappearance. In some of the instances it has been recorded that "the cyst was burst by the child falling upon it," and the accident led to its cure. The proneness of lymphatic cysts to spontaneous cure explains their rarity after puberty; this is fortunate, for their treatment by surgical means is uncertain and often extremely dangerous.

Endothelioma. — This is a rare species of tumour, which is supposed to arise from the inner walls of blood and lymph vessels. The tumours rarely attain a large size, but are excessively prone to degenerate, and they also exhibit malignant characters, which are displayed more especially in the form of local recurrence.

Endotheliomata have been described in relation with the mucous membrane of the jaws (gum), palate, in the skin, especially in connection with moles and warts: also growing from the pleura.

It is probable that many tumours described as epithelioma of the gums and palate, and some remarkable malignant tumours described as cystic disease of the jaws, were endotheliomata.

The peculiar tumours (often called sarcomata) of the parotid and occasionally of the submaxillary glands are probably of this nature, and it is known that lymphatics abound in these organs. The tumours themselves are remarkable for the variety of tissues they contain, as well as for their curious clinical features.

It is also necessary to mention that there exist good grounds for the belief that tumours allied to the sarcomata also arise from the walls of blood-vessels, and in many respects behave like the endotheliomata.

Myomata.—These are tumours composed of unstriped muscle tissue. They are met with exclusively in organs containing this tissue, *e.g.* the œsophagus, stomach, and bladder, but in these situations they are extremely rare. The common situation is the uterus, where they are known as fibroids. It was formerly supposed that these tumours were composed of fibrous tissue, until Virchow demonstrated that they consist mainly of unstriped muscle tissue.

The fibroid or fibro-myoma may arise in any part of the uterus, but are more common in the body and fundus than in the neck of this organ. The tumours are always encapsuled; they vary very much in density, some are soft and gelatinous, others are as firm as cartilage, and some undergo calcification and resemble porous stone. That these are varieties of the same tumour is shown by the fact that the different kinds of fibroids may be found in the same uterus, and not infrequently the gelatinous, the firm, and the calcified tissues may be found in the same tumour.

A uterus may contain one large fibroid or it may be occupied by ten or more. The writer has met with one remarkable uterus which contained one hundred and twenty fibroids; they varied in size from a mustard seed to a dove's egg.

Clinical Characters.—Fibroids are only met with after puberty, and are extremely rare before the age of twenty-five; from the thirtieth to the forty-fifth year they become very common, and it is probable that they never arise after the termination of menstruation.

Fibroids are inimical to health and dangerous to life in a variety of ways: they cause under certain conditions profuse and even fatal bleeding; they are apt to become infected and destroy life from septicæmia. Very rarely they become sarcomatous and disseminate. Very large tumours fill up the pelvis and interfere

with the bladder, and thus set up changes in the kidneys. In many instances they coexist with pregnancy, and may obstruct the transit of the child or even prevent the pregnancy going to term. In some individuals the stalk of a pedunculated fibroid will entangle a coil of bowel and produce fatal intestinal obstruction, or an impacted fibroid will press on the rectum and lead to the same result. In very many instances a woman may have many fibroids in her uterus and never suffer the least inconvenience.

Neuromata and Neuro-fibromatosis. — The tumours of nerves formerly grouped together as neuromata by surgeons have never satisfied pathologists, but in recent years a large amount of careful investigation has been conducted into the histology of certain lesions of nerves and skin, which enable some remarkable and apparently diverse conditions to be included under the heading neuromata.

The nerve centres, brain and spinal cord; the nerve trunks and their terminal twigs; the sympathetic nerve cords and their ganglia are all pervaded with connective tissue of varying degree of texture; for instance, it is coarse in the great sciatic nerve but of extreme delicacy in the retina and brain, where it is known as neuroglia.

The common and familiar swelling to which the term neuroma is usually applied is an ovoid encapsuled tumour composed of tissue identical with that of the nerve sheath. These tumours may occur singly or in multiples upon any nerve or nerve roots. Some of them are no bigger than cherry stones; when one grows on the terminal twig of a cutaneous nerve it gives rise to such pain when touched that it is called in consequence a "painful subcutaneous tubercle." The common solitary neuroma has received a variety of names, depending on its minute structure, such as myxoma, myxo-fibroma, fibroma, and so on, the varieties of texture being due to degenerative changes in the tissue of the tumour. It has, however, been definitely proved that multiple tumours sometimes form in the skin and resemble lipomata, but on careful examination they have been found to contain nerve elements, and particularly nerve cells. These are known as ganglionic neuromata, and it is supposed that they arise on the terminal twigs of the sympathetic nerves distributed to the cutaneous branches of arteries.

It has been known for many years that individuals occasionally come under observation with tracts of skin excessively developed in the form of overlapping folds, and these may form on the trunk, head, or limbs. This condition is known under a variety of names, but that accepted by the most recent writers is molluscum fibrosum. This disease may also manifest itself in the form of a multitude of discrete nodules in the skin, varying in size from a mustard seed to a billiard ball. Recklinghausen, who studied the histology of these molluscum nodules with great care, has expressed the opinion that they arise from the terminal twigs of cutaneous nerves. These molluscum nodules occasionally occur in individuals who have also a multitude of irregular swellings on their nerves, and in one remarkable instance two thousand tumours were observed on the skin and nerve trunks of one man. It is also an important fact that though neuromata, molluscum nodules, and molluscous folds usually occur separately in individuals, nevertheless the three conditions are sometimes met with in the same patient.

It is now usual to speak of the generalised form of the disease as neuro-fibromatosis, and an important feature connected with the extreme forms of the disease is the liability of the patients to sarcoma; this may develop primarily, or arise as a malignant change in a molluscum nodule which has existed many years. In some cases of generalised neuro-fibromatosis the patients die from gradual exhaustion due to ulceration or septic changes in the large pendulous folds of skin.

Pathologists have long been familiar with a peculiar overgrowth of the delicate connective tissue of the brain and spinal cord under the name of glioma; this appears as a translucent swelling imperfectly demarcated from the surrounding tissue, and, as a rule, this gliomatous tissue has little more consistency than vitreous humour; microscopically it has all the characters of neuroglia. Virchow points out that when a glioma is situated near the surface of the brain it appears like a colossal convolution, and when it attacks an optic thalamus this structure bulges into the third ventricle as though overgrown; and occasionally the disease attacks the cerebral crura in children under twelve years of age, and produces peculiar enlargement of this part of the brain.

It is an extraordinary fact that the connective tissue, especially the endoneurium of nerves, is liable to a similar change; this may be, and usually is, limited to a particular nerve and its branches, *e.g.* the anterior crural, the musculo-spiral, or the great occipital. The effect is very striking, for the nerves become thickened, elongated, and tortuous, and when cut across the nerve sheath is found filled with gelatinous tissue and produces an appearance not unlike the cut surface of an umbilical cord. Microscopic examination of the enlarged nerve reveals the axis cylinders embedded in overgrown endoneurium. To this condition the term plexiform neuroma is applied, and it is necessary to mention not only that the disease is congenital, but that the nerves most affected are often covered by pigmented skin; frequently the overlying skin is in the condition known as a hairy mole.

The plexiform neuroma is not only allied to gliomata by the peculiar changes of its endoneurium, but its pathological relationship to molluscum fibrosum and localised neuromata is demonstrated by the occurrence of a plexiform neuroma beneath a molluscum fold; and a large oval neuroma may form on a nerve, the sheath of which is thickened throughout. There are also good grounds for the inclusion of retinal glioma in this group, for the optic nerve and retina are processes of the brain, and the parallel exhibited in the age distribution of the gliomatous enlargement of the pons and crura met with in children under twelve years of age and the limitation of glioma of the retina to infancy are significant features. The malignancy of retinal glioma has its parallel also in the occasional occurrence of sarcoma in a molluscum nodule.

Group 2.—The Epithelial Group

In the tumours of this group the epithelium is not only present but is the essential and distinguishing feature. The difference in the disposition of the epithelium enables the tumours to be arranged in three genera: 1. Papillomata; 2. Adenoma; and 3. Carcinomata (cancer).

1. *Papillomata.*—These are commonly known as warts, and may arise on any surface covered with epithelium. Structurally a wart consists of an axis of fibrous tissue containing blood-vessels, surmounted by epithelium. The wart may be simple and present a uniform surface, or be so covered with secondary process as to look like a mulberry. When the warts grow from a surface covered with stratified epithelium the cells on the warts have the same disposition; this is seen when they arise on the skin or the mucous membrane of the larynx. When they grow from the mucous membrane of the bladder or the pelvis of the kidney warts have an appearance not unlike chorionic villi, and are termed in consequence villous papillomata; this species is very delicate and extremely vascular. A rare but important form of papillomata grows from the choroid plexuses of the cerebral ventricles, and are apt to become calcified and form a tumour composed of peculiar concentric bodies. These tumours are known as psammomata. Villous papillomata are apt to grow in cysts, which arise in dilatations of the milk ducts of the mammary gland; these are known as intracystic papillomata, but the most remarkable examples of this species arise in certain ovarian cysts, where they form dendritic masses which burst the cyst wall, and then sprouting into the abdomen resemble a large cauliflower.

Warts are under certain conditions inimical to life. Thus the large solitary congenital pigmented wart is apt to be the starting-point of a melanoma. Warts in the larynx sometimes induce suffocation. Villous papillomata of the fourth ventricle give rise to fatal pressure on the important nerve centres on the floor of that cavity, and the vascular villous tumours of the bladder and kidney occasionally cause death from oft-recurring bleeding.

An important change to which skin warts are liable is the slow conversion of the surface cells into horn. Such horns may attain a length of nine inches.

Adenomata.—These are tumours constructed upon the type of and growing in connection with secreting glands. They occur as encapsuled tumours in such organs as the mamma and liver; and in glands like the parotid and thyroid. When they arise in the mucous membrane of the rectum, intestine, or uterus they are pedunculated (or polypoid). A single adenoma may arise in a gland, but it is not uncommon for them to occur in multiples. In size they vary greatly, some are no bigger than peas, whereas an adenoma in the mammary or the thyroid gland may form a tumour as big as a man's head. An adenoma contains glandular spaces, and though a tumour of this kind does not furnish the secretion peculiar to the gland in which it arises, it nevertheless produces in many instances a perverted secretion which leads to great distension of the spaces in the tumour, and converts it into a cystic body. Tumours of this kind occurring in the breast are sometimes termed "adenoceles," and in the thyroid gland "bronchoceles." Some of the largest occur in the ovary (ovarian adenomata), and sometimes attain a weight of a hundred or more pounds. The effects of adenomata depend in the main on the situations in which they grow. For example, a very large mammary adenoma may be, and usually is, painless, but it annoys by dragging on the parts and disfigures by its size, whereas a small adenoma of the prostate gland may so thoroughly plug the vesical orifice of the urethra as to produce complete obstruction to the flow of urine. So too in the thyroid gland, a bronchocele may be so big that the patient has to support it with her hands whilst she walks about, and yet it does not endanger life, whilst a solid adenoma no bigger than a walnut may compress the trachea and induce fatal dyspnœa.

The following statements are true of all adenomata: when completely removed there is no fear of recurrence; they do not infect lymph glands; nor give rise to secondary deposits.

A tumour in many points closely allied to adenomata is that known as the sebaceous cyst, so common in the skin and particularly of the scalp. These tumours arise in the sebaceous glands. They may occur singly or in multiples. Sebaceous cysts may inflame, suppurate, or form cutaneous horns.

Carcinoma (cancer).—The term carcinoma is applied to a malignant tumour arising in epithelium. When the source of the cancer is the epithelium of a secreting gland, it is described as a glandular carcinoma, but when it

arises from a surface covered with squamous epithelial cells, it is called squamous-celled cancer, or, more particularly by British writers, epithelioma.

The microscopic structure of a cancer is very simple, for it consists of columns of cells, so that when these columns are cut at right angles the section has the appearance of a number of alveolar spaces filled with epithelium. The walls of these alveoli consist of fibrous tissue of varying degrees of density in different species of cancer, and in this tissue the blood and lymph vessels ramify. These cell-columns are not always simple but branch and ramify in different directions, producing in some sections very complicated appearances. The softness or hardness of a cancer depends in a large measure on the amount of cells as well as on the density of the fibrous tissue composing the septa. The cells in the columns take their characters from the cells in which the cancer arose, and so constantly do they preserve the type of the cells and so frequently mimic the architecture of the gland in which the cancer is situated, that an experienced histologist can often determine the source of the carcinoma from a study of its microscopic features.

Although cancer may arise in any epithelial-covered surface or secreting gland it is exceedingly common in some and very rare in others. Thus the three commonest situations are the mammary gland, the stomach, and the neck of the uterus; it is rare as a primary tumour in the liver, the thyroid gland, and the kidney, and it is a significant fact that it is more prone to arise in glandular organs which have been injured, or are the seat of chronic disease, than in those that are healthy; this is borne out especially in the case of cancer arising in the neck of the uterus, for this disease is almost exclusively confined to women who have been pregnant, and in the thyroid gland, for cancer of this gland is much more frequent in countries where goitre is endemic.

A striking feature of cancer is the fact that it is not circumscribed, and it is not possible in naked-eye examination to define the limits between the tumour and the surrounding tissue: this indefiniteness is more obvious when in the course of an operation the surgeon cuts into it, and when the parts are subjected to microscopic scrutiny it is difficult to determine with accuracy the limits of the cancerous territory.

It is this illimitation which constitutes one of the greatest difficulties in dealing with cancer surgically, and has led surgeons to advocate the complete extirpation of cancerous organs whenever possible with the hope of eradicating the disease. When cancer extends beyond the organ in which it arises, the cancerous elements will quickly involve, or implicate, adjacent organs, whether they consist of skin, fat, muscle, or bone. This tendency to implicate neighbouring organs is a grave feature of this disease, and is a common cause of death, as well as being a bar to efficient operative efforts.

A very dangerous character of cancer is the frequency with which it infects lymph glands. All epithelial structures are rich in lymphatics, and these convey the cancer elements to the lymph glands associated with the cancerous part; and such infected lymph glands may form a tumour far exceeding in size the primary cancer. It is a difficult matter to determine in a given case the extent to which the lymph glands have been infected, and this adds to the uncertainty in estimating the value of surgical interference.

Cancers are exceptionally prone to disseminate, and secondary growths may make their appearance in any organ or tissue and, not infrequently, in bone. The cancerous particles that give rise to these secondary nodules are transported by lymph and blood-vessels, and when these minute cancer-emboli are lodged in suitable situations they multiply and give rise to nodules which in their histologic features exactly resemble the parent tumour. The amount of dissemination varies greatly: in some cases secondary growths may be only found in the liver or lung, yet in another and apparently identical case, so far as the structure and situation of the tumour are concerned, secondary knots appear in almost every organ, including the skeleton.

The vitality and power of independent growth possessed by minute detached portions of cancer is extremely remarkable. These minute epithelial emigrants not only live and grow but they reproduce the peculiarity of the primary tumour. It is astonishing to find a secondary mass of cancer in the humerus with all the characters of the glands of the rectum; nodules on the lungs exactly reproducing that peculiar form of hepatic cancer that arises in the biliary ducts; or the familiar closed follicles of the thyroid gland reproduced in the spinous process of a vertebra; or a nodule with the structure of a mammary cancer growing in the choroid coat of the eye.

This great power of independent growth possessed by cancerous epithelium is a very dangerous feature, and it occasionally happens that in the course of operations cancerous elements are spread about the wound and give rise to a form of rapid local recurrence which accelerates the fatal course of the disease.

The Cause of Cancer.—There are many facts which indicate that cancer is induced by microparasites, for those glands which are direct communications with the air or intestinal gases are most prone to become cancerous, *e.g.* the mamma, rectum, and stomach; whereas cancer of the thyroid and prostate is unusual.

The great frequency of cancer in the cervical endometrium, in comparison with its infrequency

in the corporeal endometrium, and its extreme rarity in the mucous membrane of the Fallopian tubes, are significant facts in relation to invasion by micro-parasites. The greatest difficulty in connection with the micro-parasitic or infective theory as to the origin of cancer is the extraordinary fact that the secondary nodules are structurally identical with the primary tumour independently of the tissue in which they are deposited. This is quite contrary to the behaviour of disseminated foci in such typical micro-parasitic diseases as glanders and tuberculosis. It is possible that in cancer the cell-emboli with the micro-parasite when lodged in foreign tissue multiply in virtue of the presence of the parasite. However, the latter is as yet purely hypothetical.

It has already been mentioned that the frequency with which cancer attacks secreting glands varies greatly. In some it is fearfully common, in others extremely rare, and in a few almost unknown. Although lymph-gland infection and dissemination occur in all forms, nevertheless there is great variation: for example lymph-gland infection is a most common and striking feature of squamous-celled cancer (epithelioma), especially when it occurs near the mouth, anus, or on the scrotum, and dissemination is the exception. Yet in the peculiar form of cancer which attacks the face (rodent ulcer) lymph-gland infection is a very rare phenomenon and dissemination unknown. In mammary cancer the lymph glands are early infected and wide dissemination is the rule. In cancer of the neck of the uterus, enlarged lymph glands and dissemination only occur in the late stages of the disease.

The situation of secondary nodules can with a fair measure of success be foretold. Thus in mammary cancer it is the lungs which are most commonly infected: when the intestine is the seat of the primary disease the liver becomes the repository of the secondary nodules, and when the cancer erodes its way into a serous cavity it may directly infect it. This is seen when the primary disease is in the ovary, intestine, or gall-bladder.

It is an unexplained fact that some organs though frequently occupied by secondary deposits are rarely attacked primarily: this is true especially of the lung and liver.

Cancer, though structurally the same in all the situations in which it occurs, and exhibits all the dangerous features of this disease in regard to infection and dissemination, nevertheless displays great variation in its effects according to the secreting gland in which it arises. All these peculiarities, as well as the variations in age-liability, modes of death, and the results which follow surgical interference, will be most appropriately dealt with in the articles devoted to the particular consideration of each organ.

Group 3.—Dermoids

These are tumours furnished with skin or mucous membrane occurring in situations where these structures are not found under normal conditions. They only possess tissues which naturally belong to skin or mucous membrane. Dermoids may be arranged in four genera:—1. Sequestration Dermoids; 2. Tubulo-dermoids; 3. Dermoid Patches (moles); and 4. Ovarian Dermoids.

1. *Sequestration Dermoids.*—These arise in detached or sequestrated portions of skin chiefly in situations where, during embryonic life, coalescence takes place between cutaneous surfaces. Thus they are met with in the middle line of the back; in the scrotum; in the middle line of the sternum, and more especially in the situation of the facial fissures. They also occur frequently on the scalp. As a rule they do not exceed the dimensions of a tangerine orange, but occasionally they attain the size of a cocoa-nut. They are congenital, and consist of a central cavity lined with skin furnished with hair and the cutaneous appendages met with in the normal skin adjacent to the dermoid. As a rule a sequestration dermoid is not inimical to life. Occasionally, however, a dermoid in connection with the cranial dura mater will lead to fatal intracranial pressure, and a dermoid of the spinal dura mater has produced death from paraplegia. A dermoid arising from the internal aspect of the chest-wall will interfere with the lung, and if air gain access to it will simulate an empyema complicated with what is known as "hair-spitting."

2. *Tubulo-Dermoids.*—This species arises in connection with what are known as obsolete canals, *e.g.* the branchial clefts, the post-anal gut, and the thyro-glossal duct. Dermoids arising in the neck in relation with the anterior border of the sterno-mastoid muscle have long been associated with the branchial clefts. On the other hand, tumours of this character occupying the floor of the mouth or situated between the genio-hyo-glossi muscles have been many times observed, but were described as sebaceous cysts. A more thorough examination of their structure, aided by a better knowledge of the details of the development of the tongue and mouth, has proved that they are dermoids, and that many of them arise in the glossal segment of the temporary passage between the tongue and the embryonic thyroid gland, known as the thyro-glossal duct.

Post-Rectal Dermoids are by many associated with the extension of the primary gut which, in the embryo, extends behind the anus, and which for a time is in communication with the central canal of the spinal cord by means of the neurenteric canal. The best examples of dermoids arising in the post-anal gut are always congenital, and generally of large size, and

contain gland spaces and sometimes well-formed Lieberkühnian follicles. Another variety is situated between the rectum and the sacrum; some of these are undoubtedly teratomata. A third and rarer variety takes the form of a pedunculated skin-covered pilose tumour hanging from the mucous membrane of the rectum.

3. *Moles* (*Dermoid Patches*).—Moles are pigmented and usually hairy patches growing on the skin. They are congenital, or appear during the first few weeks after birth. These patches vary greatly in size; many are no bigger than split peas, whilst others cover an extensive area on the face, trunk, the limbs, and even on the scalp.

The common variety consists of a slightly raised brown patch; it is sometimes quite black, and is as a rule pilose, the hair being short. The hairs are furnished with sebaceous glands, and the skin often contains sweat-glands even when the mole is situated on the conjunctiva. The most striking feature in the histology of a mole is the fact that the tissue immediately underlying it is arranged in alveoli, like that described as characteristic of an alveolar sarcoma. Moles bleed freely when their surfaces are abraided, they are liable to ulcerate, and later in life are occasionally the sources of melano-sarcomata. They occur singly or in multiple, as many as fifty may occur on one individual; sometimes one mole may be so big as to cover half the trunk. Moles on the conjunctiva are often associated with defects in the eyelids, such as coloboma and total absence of the lids.

Ovarian Dermoids.—Cystic tumours containing skin and often furnished with the appendages of skin, such as hair, sebaceous and sweat-glands, teeth, horn, and bone, occur with exceptional frequency in the ovary. In rare instances a mammary gland furnished with a nipple and areola has been met with, and also an organ which in structure resembled a thyroid gland. Many of the loculi in ovarian dermoids are lined with mucous membrane. These tumours may be unilocular or multilocular, and when the skin elements predominate the cavities are filled with a mixture of fat, sebum, shed hair, teeth, etc. When the loculus contains mucous membrane, then the cavity is filled with mucus of the consistence of thin jelly. In size ovarian dermoids vary greatly: some are no bigger than an orange, whilst others attain a weight of twenty or forty kilogrammes. The average dimensions of these tumours is that of a cocoanut. They occur at all periods of life, and have been met with in a child a year old and in a woman of eighty-three. There is no authentic observation of a dermoid in a new-born child.

Some extraordinary theories have been advanced to account for these tumours in the ovaries, such as pathogenesis aud imperfect conceptions, etc., but very careful histological investigations of small specimens have shown that these tumours arise in that portion of the ovary in which the ova are lodged (the oöphoron), and that the epithelial lining of the ovarian follicles is the source of the structures found in ovarian dermoids.

These tumours are dangerous to life in a variety of ways, for they may lead to intestinal obstruction, they may offer a great and sometimes an impassable obstruction to the birth of a child, or they inflame and suppurate. Occasionally from its mere bulk an ovarian dermoid will interfere with the diaphragm, and compress the lung, death being due to interference with respiration.

They are innocent tumours, and when removed have never been known to recur. Ovarian dermoids are often bilateral. Occasionally they burst into the general peritoneal cavity, and the epithelium may engraft itself on the peritoneum and form tiny dermoids. This phenomenon may be conveniently termed "epithelial infection."

Teratomata.—Strictly the consideration of teratomata belongs to the department of pathology known as Teratology, but some teratomata are very apt to be confounded with dermoids. In the majority of the specimens there is no difficulty, because a teratoma is an irregular conglomerate mass containing the tissues and segments of viscera of a suppressed fœtus attached to an otherwise normal individual. Tumours of this character are common in the neighbourhood of the sacrum, and are known as one variety of the congenital sacro-coccygeal tumour; they also occur around the cephalic termination of the notochord, and appear in the pituitary fossa or in the pharynx, either as sessile or pedunculated tumours. The rarest form of teratoma occurs as a retroperitoneal tumour. The variety of teratoma which it is most difficult to distinguish from a dermoid is that which appears as a skin-covered tumour in the nasopharynx or between the rectum and coccyx.

All sequestration dermoids and all teratomata are congenital, but no one has observed a congenital ovarian dermoid, and this is a distinguishing feature of great significance in considering the pathology and pathogenesis of dermoids.

Phantom Tumour.—This term is applied to a tumour which appears and disappears without any obvious reason. In the limbs a phantom tumour assumes the form of a smooth, uniform, resistent swelling; it is generally painless, and usually subsides during sleep, to reappear when the patient wakes, but it always vanishes under deep anæsthesia. Phantom tumours occur in men, but they are commoner in women, in whom they assume the curious form of mimicry known as "spurious pregnancy" or pseudocyesis, but it simulates ovarian and other abdominal swellings as well as pregnancy.

The symptoms of this form of phantom tumour are briefly these: a woman will fancy she is pregnant or suffering from a tumour, and states that her abdomen has been gradually increasing in size.

These cases rarely give rise to difficulty. When the abdomen is submitted to physical examination, it will be found everywhere resonant, and loud intestinal gurgling is usually present; by cautiously engaging the patient in conversation during the manipulation, the belly may be pressed quite flat. In such a case the age gives important indications, especially when the woman has passed the menopause. Again, in younger women, the other signs of pregnancy, such as enlargement of the breasts, morning sickness, increase in the size of the uterus, and amenorrhœa, are usually wanting.

If after a physical examination the surgeon still feels in doubt, an anæsthetic will decide the question; as the patient becomes unconscious the abdomen diminishes in size until it becomes quite flat, as she returns to consciousness the abdominal distension reappears.

Phantom tumour occurs more especially in sterile women who have married late in life, and in women who have a morbid desire for pregnancy. It is occasionally met with in women who have borne children, and now and then in young wives. Sometimes it is seen in women who have subjected themselves to illicit intercourse and fear the results.

Smollett furnishes an admirable description of spurious pregnancy occurring in an elderly woman, in his account of the supposed pregnancy of Mrs Trunnion (*Peregrine Pickle*, chap. x.).

A remarkable case is recorded by Havelock. The patient was seduced, and, becoming alarmed at the possibility of pregnancy, fell into a state of religious ecstasy. The rest of the family, consisting of four adolescent sons, and four daughters and the parents, fell into an ecstatic condition and remained so many days. Eventually they came to their senses, but the girl was retained in the asylum. She had many signs of pregnancy: amenorrhœa for several months, milky fluid could be expressed from the breast, and the areolæ were altered. Gradually the mental condition improved and the signs of pregnancy disappeared (*Obstet. Trans. Ed.*, vol. xix. p. 183).

As to the cause of the singular mimicry no good explanation is forthcoming; conditions resembling it have been reported in the ass, and are not uncommon in petted bitches.

It is difficult to understand how this condition could be mistaken for an abdominal tumour, yet more than one case has been recorded in which the abdomen was opened to remove the supposed tumour.

In 1824, Lizars performed abdominal section in order to remove a supposed ovarian tumour, and was surprised to find only fat and distended intestines. This was in 1824, and with the accumulated experience of three-quarters of a century to guide them, this error is even now occasionally committed by surgeons.

Phantom tumour is occasionally imitated by intermitting hydronephrosis, movable kidneys, and ovarian and parovarian cysts when they burst.

Tumours, Inoperable, Treatment of.

General Management 165
Special Therapeutics 167
Diet 167
Drugs 167
Bacterio-Therapy 167
Serum-Therapy 167
Organo-Therapy 167
Photo-Therapy 168
Electricity 168

Treatment of Inoperable Tumours.—It is not often that a simple or benign tumour is beyond removal, and when it is so, it seldom endangers life, being only a source of disfigurement and inconvenience by reason of its bulk. Consequently, this article has to deal entirely with those neoplasms known as malignant, the chief varieties of which are the Carcinomata and the Sarcomata, the former composed of a type of cell essentially epithelial, and the latter of embryonic connective tissue. Unsatisfactory as is our present knowledge of the etiology of new growths, there is very general unanimity of opinion in the profession that early and wide excision by the surgeon's knife is the proper treatment of those of a malignant nature, special attention being directed in the case of the carcinomata to the removal of affected lymphatic nodes and vessels. This tendency to involve the adjacent lymphatics is characteristic of the relations of carcinomatous growths to the surrounding tissues. The latter seem unable to offer any resistance to them, so that all the structures and organs of the body, the bones, the blood-vessels, the nerves, the air-passages, the different parts of the alimentary canal, and the various viscera are affected by them. Absorption, pressure, ulceration, and metastasis are the agencies constantly at work, so that it is not surprising that tumours are frequently met with that are inoperable owing either to the existence of internal visceral or glandular deposits, or of external growths in inaccessible positions, or of great weakness and exhaustion on the part of the patient. Such cases are the most distressing that can be met with. Of recent years it is an encouraging fact that they are attracting the attention they are very properly entitled to, not only on the ground of humanity, but also because they afford an excellent field of scientific research in connection with the etiology of malignant disease.

The treatment of all inoperable tumours is best considered under two heads:—

1. The general management of all such cases.
2. The adoption of any special therapeutic measures.

General Management.—The principles underlying this are common to all these cases. They call for generous diet, fresh air and good hygienic surroundings, with digestive and other tonics. In addition, however, special measures are needed, based on the *causes* which actually bring about death.

It has been already pointed out that the leading feature of inoperable tumours (malignant) is their progressive character, so that they kill by invading adjacent tissues, the factors that are constantly at work being ulceration, pressure, and metastasis. Consequently, there are induced by these not only certain local, but also constitutional effects, and death may be the result of any of the following conditions:—

1. Hæmorrhage.
2. Exhaustion from excessive discharges.
3. Septicæmia and sapræmia.
4. Continuous pain and mental depression.
5. Stricture of vital parts.
6. Visceral deposits.

It will be as well to say a few words on each of the above points separately.

Hæmorrhage.—Bleeding in inoperable tumours is often associated with a sloughing ulceration of the tissues and with great vascularity of the new growth, so that the ordinary methods of arresting hæmorrhage cannot always be employed. It would be useless to attempt to tie a bleeding vessel in the substance of a breaking down malignant growth, or in a soft pulsating sarcoma, composed, as it is, mainly of a mass of blood-vessels. The best hæmostatic agents to rely on are pressure, cold, heat, and styptics.

Pressure, when used, must be employed thoroughly and judiciously. Small pads of dry medicated absorbent wool or strips of dry antiseptic lint, such as boric, should be carefully packed into the deeper parts of the wound or ulcer, and over these a firm compress applied. In some cases the "graduated compress" is very useful, while for the vagina and rectum "plugging" may require to be resorted to. Such plugs and pads used for the arrest of bleeding should not be disturbed too soon, yet not left too long untouched in case of septic absorption. *Cold* is a valuable hæmostatic, and may be applied in the form of cold water or ice, or by the ether spray. This latter is a very effective agent in cases of hæmorrhage. Strange as it may seem, the same may be said of *heat*. It must, however, be used at a certain temperature. Water should not have a less heat than 120° F., when it acts as a stimulant, causing contraction of muscular tissue through the vaso-motor nerves. Its value in uterine hæmorrhages has long been recognised. The actual cautery, too, is often of great service in cases of bleeding. The old cautery irons have now been replaced for most purposes by the more modern galvano-cautery, and by Pacquelin's thermo-cautery, this latter being a specially serviceable apparatus. In employing any of these to stop bleeding they should be used at a dull, almost invisible red heat, and the tissues should be made as dry as possible by previous pressure. This latter precaution applies also to the application of *styptics*—substances which often materially help in the arrest of hæmorrhage. They may act mechanically, as collodion, cotton wool, or tow, or, by reason of their astringency, as hazeline. This drug is a very useful hæmostatic, and may be applied in its fluid state or by means of pads soaked in it. Other well-recognised substances are powdered alum and tannin, perchloride of iron, sulphate of copper, sulphate of iron, and nitrate of silver. Not only are these astringent, condensing the tissues and coagulating the albumin, but they are also caustic in their action, and this has to be borne in mind. Thus, the strong liq. ferri perchloridi, though invaluable as a hæmostatic, may cause considerable sloughing of the tissues if too freely applied. Probably one of the best of these iron applications is Ehrlich's "styptic cotton." It may be packed directly into the recesses of a bleeding cavity, or may be enclosed in sterilised gauze and used as a pad or plug. Possibly experience may show that the more recent adrenalin solution may prove to be our most serviceable and useful hæmostatic. In cases of bleeding from inoperable tumours there is not much scope for the internal use of any drugs, but, after taking the measures mentioned for preventing further loss of blood, recourse must be had to combat the effects of what has been already lost. Nothing is more beneficial than saline infusion. A couple of pints of sterilised salt solution introduced into the rectum, or, by means of the ordinary simple infusing apparatus, into the cellular tissue of the body, is a powerful restorative. Further, in these cases everything should be done to equalise the circulation and insure absolute rest and quietness. The food should consist of iced milk and cold soups, while all alcohol should be well diluted.

Exhaustion from Excessive Discharges.—This condition arises in connection only with ulcerated growths, and must be met by the ordinary surgical principles on which all wounds are managed. Simple cleanliness, the prevention of decomposition, suitable dressings (either dry, watery, or oily), are all points requiring attention, while the avoidance of movement and of all irritation, together with attention to drainage, must on no account be lost sight of. The prevention of decomposition is of importance, not only on account of its local effects, but as bearing on that other factor in these

cases, viz. blood-poisoning or septicæmia. How to obviate this will best be considered under that subject.

Septicæmia.—This is seldom seen in inoperable tumours; the form usually present arises from the absorption of the products of decomposition present in the ulcerated surface (sapræmia). Sometimes it is associated with special organs, as in the septic pyelitis of the kidney, so frequently present in advanced cancerous disease of the uterus. It is really represented by the so-called cachexia of malignant disease. It must be prevented by the use of weak solutions of the ordinary antiseptics, such as creolin, boracic acid, carbolic acid, sanitas, and peroxide of hydrogen. Where there is much superficial sloughing, charcoal poultices, or dressings of wet boracic lint covered with jaconet, keep down the odour and set up a healthier action in the growth. Of the dry dressings the most suitable are ones impregnated with an antiseptic and of an absorbent character. Hartmann's wood-wool wadding, boric and salicylic wool, Martindale's moss, carbolised tow, and gamgee tissue are all excellent applications. They must be frequently changed, and are often best held in position by the triangular bandage. In the removal of dressings great gentleness should be used, and they should be well wetted if adherent, so as not to set up bleeding. In inoperable cancer of the uterus vaginal plugs soaked in a 5 per cent glycerine solution of ichthyol have a cleansing and deodorising power, and they may be introduced every second day, a warm weak creolin douche being given after their removal. Sometimes curetting may be added to the use of the plugs. In some of these distressing uterine cancer cases, where the disease has eroded the bladder and a vesico-vaginal fistula exists, great comfort is derived and bed-sores prevented by the use of a specially made rubber bed-pan on which the patient constantly lies. At the Cancer Hospital it has been found of the greatest use. By keeping the bed dry it lessens the tendency to bed-sores, and it saves frequent moving of the patient. If necessary it may contain some antiseptic, such as sanitas, and thus it keeps down any odour in the discharges. Cases of inoperable cancer of the mouth and tongue are specially difficult to manage, and are often very offensive. The liquid food necessary should be given by means of a drinking-cup with a piece of tubing attached and placed far back in the fauces. Any loose teeth should be extracted, and the mouth should be syringed out and washed out constantly with Listerine or lotions containing eucalyptus. This latter seems the most efficient deodoriser in these cases. If the case is very offensive a formaline lamp may be burnt in the room, care being taken not to render the air too irritating. Where the flow of saliva is very great, belladonna as atropia may be administered. Space does not allow of individual cases being further dealt with, but to all of them the principles of cleanliness, prevention of decomposition, and avoidance of irritation must be applied, and when emaciation sets in the prevention of bed-sores by the use of water-beds and all the recognised local measures must be kept in view.

Pain.—This, together with a certain amount of mental depression, is more or less present in all cases of inoperable growths, but the pain is not always of that agonising character that popular fancy associates with cancer, and its injudicious management is, I am sure, responsible for much additional distress in many of these cases. The too early and indiscriminate use of narcotics is not to be followed. Pain and suffering must be relieved, but this may be done by analgesics without recourse to morphia, a drug which not only upsets the digestive organs, leading to emaciation and anæmia, but has the very great drawback of destroying the patient's self-control, so that when once begun it has to be constantly renewed to meet the restless, clamouring craving for it that is set up. These remarks do not apply to the systematic administration of opium in inoperable cancer, as advocated therapeutically by Dr. Snow, not with the object of killing pain, but of checking the growth of the cancer cells. They refer more to the hypodermic injections of morphia resorted to in a routine manner. Of the analgesics that may be employed my preference is for phenacetine, administered in 10-20 gr. doses, to relieve peripheral neuritis and restlessness, either occasionally or regularly during the day and night. It has a remarkable effect in soothing the general uneasiness and mental depression present in these cases. During the daytime it usually suffices, but sometimes it has to be replaced at night by trional, bromidia, nepenthe, or morphia hypodermically, so as to ensure a quiet night. This, however, is the exception, and it occurs more towards the close of a case. From considerable experience at the Glasgow Cancer Hospital I am satisfied that observance of the above suggestions is of importance to the patient, and renders easier the general management of the case. In some cases neurotomy may be of service, as of the lingual nerve in cancer of tongue.

Stricture of Vital Parts.—It is in connection with growths of the œsophagus, pylorus, and rectum that this factor comes into play. Every case must be judged on its own merits, but there are few cases of œsophageal disease where gastrostomy is called for. In the majority of them the narrowing may be met by the judicious use of bougies or Symond's tubes, combined with a liquid diet. In pyloric obstruction gastro-enterostomy may advantageously be resorted to, while in cases of rectal disease a left iliac colostomy prolongs life, relieves distressing symp-

toms, and materially adds to the patient's comfort. In these latter cases a special apparatus may conveniently be worn.

Visceral Deposits.—All that can be done for these is to mitigate the distressing symptoms they give rise to. Digestive and pulmonary irritation must be met by the ordinary remedies, while the distress caused by pleural effusion and by ascites must be relieved by frequent tapping or the use of Southey's trocars. In the persistent vomiting which often accompanies cancer of the alimentary canal, not arising from obstruction so much as more commonly from a subacute form of peritonitis accompanying the disease, pills of oxalate of cerium and cocaine, and of cocaine and morphia have been specially praised. Abdominal counter-irritation by turpentine stupes and by linseed and mustard also does good.

The Adoption of any special Therapeutic Measures.—In addition to the general management of these cases we have always to keep an open mind on the possibility of any special therapeutic measures doing good in any particular case, for experience has emphasised two points in connection with inoperable tumours, viz.: (1) the *capriciousness* of malignant disease, and (2) the reality of the *retrogression* of cancer. All medical men are familiar with the fact that the characteristics and the rate of progress of malignant disease vary with each individual, while there seems, apart from errors in diagnosis, reliable evidence that in a certain number of cases of cancer spontaneous retrogression of the disease has taken place. Both these factors have to be taken into account in testing the value of any new methods of treatment, and at the same time it is clear that, while viewing with distrust all the so-called "cures" for cancer, our management of these cases should be carried on in a spirit of hope rather than of despair, and that attention should be directed towards any suggested therapeutic measures. As several of these have attained a certain measure of success, in that there has been under them some amelioration of symptoms and prolongation of life, they are entitled to some consideration.

As pointed out in the remarks on the general management of these cases, the use of the knife for neurotomy, gastrostomy, gastro-enterostomy, and colostomy may improve the patient's condition and in many ways benefit him, so its employment for the ligature of the vessels supplying a tumour has been advocated and practised with some benefit in a certain number of cases. Entirely palliative as these measures are, others have been suggested having a more curative aim. They may be classified as follows:—

1. Diet.
2. Drugs—(*a*) Internally, (*b*) Injection.
3. Bacterio-Therapy.
4. Serum-Therapy.
5. Organo-Therapy.
6. Photo-Therapy.
7. Electricity.

Diet.—Complete abstinence from red meats and from stimulants has been advocated, but results have not endorsed the idea, and though milk and white meats seem to suit some, the majority of these cases do best with a generous diet.

Drugs. — Internally: celandine (Spivach), chian turpentine (Clay), arsenic (Campbell de Morgan), opium (Snow), cacodylate of soda (Paque), have all been tried internally, but have not established their reliability. The same may be said of the parenchymatous injection of various irritating substances such as acetic acid, alcohol, methyl-violet, venom of cobra di capello, formalin and soap, or of agents calculated to produce suppuration artificially, such as oil of turpentine, arsenious acid, and calcium carbide.

Bacterio-Therapy.—Under this head come inoculations of the streptococcus of erysipelas, and of this micro-organism modified by admixture with the bacillus prodigiosus and known as "Coley's fluid." The use of the former has been largely replaced by that of the latter, which is practically useless in cases of cancer, but has undoubtedly been of value in a certain number of *spindle-celled sarcoma*. It must be used cautiously. A beginning is made by injecting subcutaneously in the vicinity of the tumour ½ minim of the fluid, the reaction caused being watched, and the amount increased to 2 minims. If there is no response to treatment in three to four weeks there is no good continuing.

Serum-Therapy.—Anticancerous serums, such as those of Vlaieff and Roquette, and the thymus serum of Foulerton, have been tried, but without any definite results. Foulerton admits that so far nothing more than mere transient effects have followed the injection of his serum.

Organo-Therapy.—This includes the administration of thymus extract, lymph gland extract, thyroid extract, and oophorectomy. Thymus extract, as administered by Foulerton, has not had an extended trial, but so far it would seem, like the serum, not to produce any distinct effects. The same may be said of Snow's lymph gland extract. Thyroid extract, on the other hand, has been a good deal used, and opinions differ as to its utility. In the medical journals some practitioners speak well of it, while others have no faith in it. Theoretically it should be of service, because of its known effect on the lymphatic system, the one that is paramount in importance in carcinoma. My own experience is in favour of it, but its effects should be watched, and only those cases chosen that it agrees with. The body weight is a good guide on this point. It should be begun in small doses of 3 grains daily, and increased up to 15 grains.

Oöphorectomy was originally put forward

seven years ago as a beneficial measure in inoperable mammary cancer, and the experience gained since then has been confirmatory of that view. It has not been universally successful by any means, and time has shown that many of its effects are transient, but judged by the principles underlying all operative work in cancer, viz. the prolongation of the patient's life and the alleviation of the local trouble, it has not been found wanting, for cases of inoperable mammary cancer are forthcoming in which the beneficial effects of relief from pain and discomfort, of wasting and disappearance of masses of cancer and enlarged glands, and of marked improvement in the general health, as shown by gain in weight and improvement in strength and spirits, have continued for some years. Applicable only to inoperative mammary cancer with its local and glandular recurrences, it would seem that oöphorectomy cannot be relied on in every case, and that it should not be resorted to in cases where there are evident visceral deposits. When performed, experience is in favour of combining it with the administration of thyroid extract.

Photo-Therapy.[1]—As curative agents the Finsen's light and the X-rays have been of late resorted to in cases of sarcoma and carcinoma. There is sufficient evidence already forthcoming to show that in both of them we have a successful method of treatment for rodent ulcer and for surface carcinomatous disease. Under them healing takes place, pain lessens, and discharge diminishes. Their action seems selective of cancerous tissue, and to be irritating to and destructive of it. We are not yet in a position to say whether these apparent curative effects are permanent. From what I have seen I am satisfied that the X-rays, especially in these cases, may be dangerous, and that they require careful watching. Space will not allow of the details of application being given here, but each "sitting" lasts usually about ten to fifteen minutes, and is given on alternate days. No cases of sarcoma being benefited by photo-therapy have yet been published.

Electricity.—This has been used in different forms from time to time. We have had the "electric puncture" of Reading of Philadelphia, and the "kataphoresis" of Massey, where the positive pole is coated with mercury, forming an oxychloride of mercury, an infinitesimal part of which acts lethally on the cancer cells and leaves the normal cells undisturbed. More recently we have had high-frequency currents introduced, and in the hands of some of the operators very striking results are said to have been obtained. I have not had any personal experience in their use, and I know of no published cases that prove satisfactorily the value of electricity thus employed in inoperable tumours.

[1] This subject is further discussed under X-RAYS.

In conclusion, I would say that as matters stand at present we have no remedial measures in inoperable tumours that can be spoken of as *curative*. Many of them are decidedly *palliative*, and as such are entitled to proper recognition. Coley's fluid, oöphorectomy, thyroid extract, Finsen light, X-rays, have all their appropriate sphere of action, in suitable cases prolonging life and ameliorating the patient's condition. A great deal has, however, yet to be learnt about them, and it behoves everyone working at this subject to do so in a scientific spirit, making absolutely sure of the exact nature of the cases dealt with, so that the results obtained may have a real value. Above all, let not valuable time be spent in operable cases in having resort to any of the above measures in preference to that of early, free, and wide removal by the surgeon's knife that all experience points to as being the only safe and reliable method of treatment in cases of malignant disease.

Tumours of the Skin.

CARCINOMA 168
Varieties 169
Treatment 169
ENDOTHELIOMA CUTIS 169
SARCOMATA 169
IN LEUKÆMIA 170
MYCOSIS FUNGOIDES 170
CHLOROMA 171
RHINOSCLEROMA 171
CYSTICERCUS CELLULOSÆ 171
MOLLUSCUM CONTAGIOSUM . . . 171
ADENOMA SEBACEUM 172
EPITHELIOMA ADENOIDES CYSTICUM . . 172
KELOID 172
NÆVI 173
LYMPHANGIECTODES 173
MYOMA MULTIPLEX CUTIS 173
XANTHOMA PALPEBRARUM . . . 174
,, MULTIPLEX 174
BOTRYOMYCOSIS 174

See also TUMOURS; WARTS, etc.

GROWTHS in this situation may be dealt with broadly under the headings of malignant and benign. Taking the former class first, they may be divided up as follows:—

CARCINOMA.—Apart from the lips and anus, cancer of which parts has been dealt with elsewhere ("MOUTH," vol. vi.; "RECTUM," vol. viii.), it is very rare for true epithelioma to develop without some precedent morbid condition, usually of long standing, or as a complication of congenital benign formations, such as nævi, moles, and so forth.

Crateriform Ulcer.—Under this name Hutchinson has described a clinical variety of epithelioma cutis which he has seen arise in a primary manner; but it has also been observed as developing on a previously existing rodent ulcer. Like the latter it affects much the same regions

of the upper part of the face, but, unlike rodent ulcer, it runs a rapid course. It may commence as a bossy rounded lump, which quickly increases in size and breaks down at the summit, giving rise to a crateriform appearance. Histologically it is an epithelioma. Radcliffe-Crocker records a case in which it developed about the inner canthus, and was at first thought to be a rodent. The microscope showed it to be epitheliomatous. A somewhat similar kind of growth was figured by Rayer among his drawings of cancer.

In old people the dirty, wart-like growths (senile warts, or senile, flat, seborrhœic warts, *fleurs de cimetière*) which occur about the face and body may in some instances become epitheliomatous.

Epithelioma sometimes develops in *lupus vulgaris*. This complication is infinitely more rare in *lupus erythematosus*.

Chronic infiltrated patches of dermatitis are in very rare instances liable to become cancerous, especially if scratched and worried, and may lead to death, by involving the corresponding lymphatic glands, and becoming generalised in the viscera secondarily.

The same malignant development may take place in old scars and ulcers.

Other instances of epithelioma arising as a result of precedent skin affection are cancer following *eczema ani*, and in another case developing from a *cornu cutaneum*.

Paget's disease has been described elsewhere (vol. vi. p. 173), but it may be incidentally mentioned here that the condition has been observed on the penis, the scrotum, and vulva.

Chimney-sweep's cancer need merely be alluded to in passing. Workers in tar and paraffin may be similarly affected.

Cancer en cuirasse must also be referred to.

Seaman's skin cancer is the name given by Unna to cancerous complications occurring after erythemata and hyperkeratosis of long standing, and as a result of exposure to weather.

Cancer has also been described as developing in old *syphilitic ulcers* and *scars*, on *psoriasis* patches, in the *scars* of old *burns*, and so forth.

Melanotic cancer has of late years been shown to be the true state of affairs in cases formerly included under the melanotic sarcomata. Cruveilhier, however, many years ago *clinically* designated melanotic growths under the former heading. These cancers arise in pigmented moles.

Cancer about the *nails* is of very rare occurrence. I have referred to a case of this kind, recorded by J. Hutchinson, jr., in my article on "Affections of the Nails" (vol. vi. p. 522).

The prolonged use of *arsenic*, by leading to epithelial changes, may give rise to cancer of the skin. An unusual case of *arsenical keratosis* becoming cancerous after arsenic had long ceased to be taken has been noted by Radcliffe-Crocker and Pernet.

Korowin had a case of primary cancer of the hairy scalp, which generalised, leading to more than 200 metastatic embolic nodules in the skin of the face, trunk, and limbs. The lungs were also involved. An anomalous case of multiple carcinomata has been recorded by Allworthy and Pernet, in which growths developed about the right buttock and adjacent parts. The patient died three and a half years from the onset, but no necropsy was allowed.

The large growths which sometimes develop in *Xeroderma pigmentosum* may also be epitheliomatous. A recent instance of this has been described by Pernet.

Secondarily, subcutaneous multiple carcinomata occur in the last stages of cancer of the stomach, liver, and so forth. Their surface generalisation is a sign that the end is not far off.

The treatment for cancer of the skin is removal, widely and deeply, whenever excision is possible. This is specially important in the case of the melanotic growths. The sooner a growing pigmented mole is removed the better, for generalisation and a fatal issue are rapid in neglected cases. When there are numerous subcutaneous growths, secondary to internal cancer, removal is, of course, out of the question.

Where excision cannot be carried out for various reasons, the X-rays might be tried, but not much is to be expected from their application. When caustics, such as Vienna and other pastes, are used they should be applied very thoroughly. The growth sloughs away, and it has been recommended to graft when the wound is granulating healthily. Arsenic internally is no use. Morphia and opium should not be withheld where there is pain. In hopeless cases, euthanasia must be aimed at.

ENDOTHELIOMA CUTIS may be mentioned in this place. Under this name a rare condition has been described, in which the scalp becomes affected with multiple growths, giving rise to a characteristic appearance. They grow very slowly, and do not lead to metastases, the general health remaining good. These scalp tumours have been found in two and more generations. Histologically they have been described as developing from the endothelia of capillaries. The face and upper part of chest may be affected, as in Ancell's case, which apparently was an example of the disease.

SARCOMA.—Under this heading German writers have formed a complex group. Space will not allow in this place of much refinement in the direction of division of the subject. Here the following will be dealt with:—

MELANOTIC SARCOMA.—This well-known condition may arise in pigmented moles. It is intensely malignant, and may lead to the formation of numerous growths in the skin, varying in size from peas to filberts, and also attack the viscera, with a rapidly fatal result.

It is of the greatest importance, therefore, to remove at once any mole that is growing, becoming more deeply pigmented or ulcerating, for if not dealt with early a fatal result will certainly ensue. Tinkering treatment is especially to be reprobated in such cases.

Under the name of *melanotic whitlow*, Hutchinson has described a form which begins as a chronic onychitis, with very little pigmentation, followed by the formation of a fungating tumour. Generalisation rapidly takes place.

Idiopathic multiple pigmented Sarcoma.—This condition, extremely rare as far as this country is concerned, but more frequent in Austria, Russia, and Italy, was first described by Kaposi. Since the fact has been made out that the coloration is due to blood, the designation of Kobner, sarcoma idiopathicum multiplex hæmorrhagicum, is more to the point, and has been accepted by Kaposi.

The disease apparently affects Jews in a special manner. One of the cases observed in England was a Jew from Poland. But it affects non-Jewish individuals also.

It first attacks the soles and palms, hands and feet, and gradually and successively the forearms and legs, arms and thighs. It may spread to the face and trunk.

The tumours are brownish-red to bluish-red, vary in size from a shot to a hazel nut. Their surface is smooth and their consistence firm and elastic. They remain either discrete or coalesce. They may undergo some involution, or break down from gangrene. Towards the last other parts become involved, mucous membranes of mouth, and so forth, as may also the liver, etc., the disease going on altogether for two, three, or more years before death closes the scene. Recovery may in rare instances take place. Males are more frequently attacked than females, and the younger the patient the more rapidly fatal is the disease.

Histologically they are very vascular, spindle-celled sarcomata, with hæmorrhages and deposition of pigment, occurring in them.

As to treatment, the only thing that has proved of any avail is arsenic in subcutaneous injections. This was first introduced by Köbner. He used a solution of liq. arsenicalis in the strength of one part to two of aq. dest., and injected two and a half to four drops once a day, raising the dose to seven and a half and nine drops. This treatment requires to be carried out steadily for months. Cacodylic compounds might be tried in the same way, but with care.

In the case of *non-pigmented sarcoma cutis*, the growths may be very numerous, but they are mostly subcutaneous in situation, and the result of generalisation from internal sarcoma. A sarcomatous development has occurred as a complication of *Recklinghausen's disease* (*i.e.* the association of multiple fibromata, neuro-fibromata, and pigmentation of the skin).

Congenital multiple sarcomata cutis is extremely rare. An interesting case of the kind was observed in Dr. Radcliffe-Crocker's clinic, University College Hospital. It occurred in the practice of Dr. Jordan Harvey. Some of the growths were examined by Pernet. There were seventy growths about the face, back, and flexor aspect of limbs. Histologically they were of the small, round-cell variety, very vascular (angeio-sarcomata), and situated in the corium. Unfortunately no necropsy was permitted, so it could not be decided whether the growths were primary or secondary. One or two other cases have been recorded, but Ramdohr's was most like the one just referred to, and in that case the kidneys and lungs contained growths also.

C. Boeck of Christiania has given the name of *multiple benign sarcoid of the skin* to a condition which may be considered in this place, benign though it be. It consists of a widespread, symmetrical eruption about the head and extensor surfaces of trunk and extremities. It is made up of firm nodules, varying in size from a hemp-seed to a bean. They involve the whole skin, and are movable with it. In addition, the lymphatic glands are much swollen, and there is a slight increase in the number of white corpuscles. Histologically it has affinities with sarcoma, and also with rare cases of so-called pseudo-leukæmia. This benign sarcoid clears up under arsenic or spontaneously.

In *leukæmia*, the skin may become affected by growths varying from a pin-head to a hazel nut in size, discrete or confluent, pale to brownish-red in colour, and distributed about the face, trunk, and extremities. The growths are made up of lymphoid cells. Growths have also been described in *pseudo-leukæmia*, but Nékàm and Pinkus differ in their conclusions.

Tumours of the skin occur in *mycosis fungoides* in the various forms of that complaint, either in the later stages, or they may be the first manifestation (*forme à tumeurs d'emblée* of French writers); or again in the course of the so-called *lymphodermia perniciosa* (of Kaposi), now included under mycosis fungoides. The growths may attain the size of a mandarin orange, and do not recur after removal. German writers include this disease *à la suite* of sarcomatosis cutis. But as it has been shown that it is microscopically of granulomatous structure (and named for that reason *granuloma fungoides* by J. F.

Payne), it should be kept separate from the sarcoma group.

Chloroma is the name given to light green tumours of very rare occurrence, which chiefly affect the periosteum of the skull-bones, and consist of round cells with reticulated framework. Ziegler places them under the lympho-sarcomata. These tumours may involve the skin.

Rhinoscleroma.—This is a very rare disease in this country, but it is not uncommon in Austria, where it was first described by Hebra and Kaposi. Other cases have been recorded in Russia, Italy, Brazil, and other countries.

It is a growth of stony hardness to the touch, which affects the nares and adjacent parts, both true skin in one direction, such as that of the upper lip, and mucous membranes in the other, in the latter case slowly invading the naso-pharynx, and even involving the trachea and larynx. Nodules and flat infiltrations are observed. It is microscopically made up of granulation-tissue cells, with vacuolisation and hyaline degeneration in parts. The latter altered cells contain the specific bacillus.

Although the disease is not malignant in the ordinary sense of that word, as it does not affect the general health and is localised, yet its slow but sure extension, and its rebelliousness to treatment, exclude it from the benign group of tumours. There is no tendency to spontaneous involution, and the disease goes on for years. It is, of course, a bad look-out when it involves the larynx and pharynx, for dangerous symptoms of obstruction would have to be dealt with.

Excision is no use, as the growth recurs. Mechanical means must be employed to prevent the blocking up of the nares. Lang used metallic tubes covered with salicylic plaster to introduce into the nostrils; at the same time he injected a 1 per cent solution of salicylic acid into the growth, and exhibited the drug internally. When blocking has taken place, sharp spooning or boring through with caustic potash would be the line to take. A masterly inactivity is indicated when there is no discomfort.

The most likely things it could be mistaken for would be epithelioma and syphilis. As to the former, the fact that rhinoscleroma does not break down and ulcerate, and the very slow growth, together with the situation and so forth, would help to differentiate it from cancer. With regard to syphilis, specific remedies have no effect on rhinoscleroma. The microscopical examination of a portion of the growth would reveal its true nature. The bacillus stains well with methyl-violet.

Before going on to *benign tumours of the skin*, a brief reference to *cysticercus cellulosæ cutis* may be made here, chiefly on account of the possibility of these subcutaneous growths being taken for malignant manifestations. But they have also been taken for fibromata, gummata, and so forth. They occur as firm, elastic tumours, from about a pea to a hazel nut in size, disseminated about the trunk and limbs. Wolff says as many as 2000 have been counted. The skin over them is unaltered either in colour or otherwise. The complaint is very rare in this country, but is less so where raw or improperly cooked pork is indulged in, as in Germany. Patients may also have cysts embedded in the mucous membranes of lips and tongue. When the tumours are well filled with fluid, they may give the impression of solid growths. Excision or puncture would reveal the characteristic hooklets of the parasite. When the skin alone is affected, the disease does not give reason for anxiety. But the prognosis becomes grave when fits occur, as this shows involvement of the brain by the parasite. Cystic growths may also occur about the heart coverings. The tumours may calcify and shrink in time.

Benign Tumours

Molluscum Contagiosum.—In this disease, growths varying in size from a large pin-head or so, in the earliest stage, to a pea or larger, occur about the skin, chiefly the face, breasts, and nape, but they may also develop on the abdomen and external genitalia. They are firm, sessile, prominent, solid tumours, hemispherical in shape, with slightly flattened top, pearly white or pinkish in colour, and usually umbilicated. These are the points that make the diagnosis.

The morbid process takes place in the Malpighian layer of the epidermis. Microscopical sections show wedge-shaped lobules, the result of special changes in the cells, which enlarge and undergo degeneration, the so-called molluscum bodies, accompanied by an obliteration of the papillary processes. The accumulation of these cells leads to breaking down of the horny stratum at the centre of the tumour. By some observers these molluscum bodies are looked upon as coccidial parasites. The contents can then be squeezed out, leaving a hole behind. Or the growth may undergo a process of ulceration, leading in some instances to an angry-looking sore, which, when situated about the mouth or face, may give rise to a suspicion of primary chancre. But the glands are not enlarged, and the characteristic molluscum lesions would be found in the neighbourhood, if carefully looked for.

Very large growths have been recorded. Colcott Fox recently showed an extremely extensive case—a woman in whom there were a number of growths on the scalp and adjacent parts of the face, the condition having been overlooked and allowed to progress unchecked before the patient came under his observation. Clinically, it might to unpractised observers have suggested malignant growths.

As the name indicates the disease is contagious. It may be seen to occur in mother and suckling, about the breasts of the former and the face of the infant.

The condition is also found about the nape of those who indulge in what the late Sir Richard Burton referred to as the "absurdly called" Turkish bath, and who neglect to spread their towel over the blocks used as head-rests.

Salzer has recorded an interesting case of the eyelids in a lady in the habit of feeding pigeons. The development of the growths in her case coincided with an epidemic disease among the birds, the chief symptoms of which were growths about the beak, and emaciation. The growths on the beaks were like those in Böllinger's plate of *epithelioma contagiosum* in fowls. (This name, by the way, is the German equivalent for our *molluscum contagiosum.*) Shattock has exhibited growths of the same kind in buntings; and I have myself had a canary which developed a small tumour of the beak, became emaciated, and died, the growth no doubt belonging to this category.

The treatment is to lay each little tumour open with a knife, the back of which is directed towards the skin, and thoroughly squeeze out the contents between the thumb nails or the handle of the scalpel and thumb nail, as Radcliffe-Crocker recommends. Even the smallest should be dealt with, for they will grow and become a source of fresh ones. They may be touched with a wooden match-end dipped in acid nitrate of mercury, but a small incision as aforementioned is the better way.

ADENOMA SEBACEUM —This condition had been described under various names before it received the one of adenoma sebaceum. It consists of small growths, from a millet seed to a split pea in size, distributed about the sides of the nose and naso-labial folds, in which situations the lesions are usually very closely aggregated, whereas on the chin, cheeks, and forehead they are more discretely scattered. They are more or less convex, varying from an almost colourless hue to a bright red, the latter being due to small dilated vessels.

The disease is congenital, and the lesions gradually increase in size and number. In the majority of cases the patients are mentally dull or epileptics. They are to be found in asylums for idiots. But in some instances there is no sign of intellectual inferiority.

In many, small fibromata are scattered about the skin. Radcliffe-Crocker has pointed out that associated with the disease there is frequently a flat fibrous-looking infiltrated patch about one or other iliac crest.

A careful examination would differentiate it from acne rosacea, which it resembles in distribution, but not in the history, mode of development, and character of the individual lesions.

It is essentially a sebaceous growth, but the microscopical features have varied somewhat in individual cases.

Owing to the toughness of the growths, sharp spooning has to be done vigorously, and even then recurrence may take place. Where the lesions are few in number Radcliffe-Crocker found electrolysis effectual; in another case, where the lesions were semi-confluent about the naso-labial folds, he excised portions, bringing the edges together to heal by first intention.

EPITHELIOMA ADENOIDES CYSTICUM is the name given by Brooke of Manchester to a condition of the skin of the face chiefly characterised by small pearly or pinkish growths, usually rounded and smooth, from a pin-head to a small pea in size, distributed about the eyelids, the root of the nose, forehead, naso-labial folds, and chin, but they may also occur on the upper part of the trunk. It begins usually about adolescence, affecting both sexes, but giving no trouble except for the disfigurement.

Microscopically it is an epithelial growth, but it is benign (hence name of *multiple benign cystic epithelioma*, which has also been given to it). But the possibility of true epitheliomatous development must be borne in mind; this occurred in a case of White's of Boston. Authors have described variations in the microscopical features, hence a whole string of names which need not be enumerated here.

Curetting or excision are the best ways of dealing with these small growths. But electrolysis and cauterization have also been recommended.

FIBROMA, NEURO-FIBROMA, and NEUROMA (*see* general article on "TUMOURS," p. 151).

KELOID has been dealt with in vol. ii. p. 149. But a few remarks may be allowed here. In the first place, true keloid must be distinguished from hypertrophic scarring. In the latter case, when the hypertrophy remains limited to the original scar, without a tendency to spread into the surrounding tissues in the characteristic claw-like manner, it cannot strictly be included in the category of true keloid, but the dividing line is sometimes a fine one.

Keloidal or hypertrophic scarring has been described as occurring after vaccination. In a case of Ehlers, of Copenhagen, this complication attacked five vaccinated areas in a child of six and a half. The interest of this case lies in the fact that the lesions were subjected to the Finsen light treatment, but without any good effect. Radcliffe-Crocker has employed the X-rays in a similar case. It would be interesting to know if negroes, who are racially very prone to true keloid, frequently react in this way to vaccination.

Another mode of treatment for hypertrophic scars is the local injection of an 8 per cent

solution of thiosinamin.[1] Twenty minims are injected at a time with a hypodermic syringe at various points along the border of the lesions, once or twice a week. In this way good results have been obtained in Dr. Radcliffe-Crocker's clinic. Multiple scarification—mincing up the scar really—has been recommended, but it is painful, as are also local creasote injections. Electrolysis is also useful—at any rate it acts beneficially where the irritation and pricking is a marked feature.

Keloid complications (? really hypertrophic scarring in some cases) have been observed as a result of syphilides. I have myself seen it follow ulcerative secondary lesions. An extensive case of the kind has been recorded by Arthur Shillitoe. These growths may or may not disappear under specific treatment.

Hypertrophic scarring is well known in bad cases of *acne vulgaris*. And in this place the *acne keloid* of the occiput, in which tufts of hairs project through the thickened scar, may be alluded to. It is the result of longstanding suppurative folliculitis of this part, to which Kaposi has given the unwieldy name of *dermatitis papillaris capillitii*. The process may be associated with severe acne vulgaris and sycosis barbæ.

Hypertrophic scarring may also be a sequela of other eruptions.

Nævi have been dealt with under "Electrolysis" (vol. iii. p. 66); and Moles have been considered under "Tumours" (p. 163). A point to bear in mind is that nævi may give rise to cancers, and moles to melanotic malignant growths.

As to the treatment of nævi, it is sometimes possible, where the situation and size are favourable, to obtain a cure by vaccinating upon them with due care.

In dermatological literature numerous cases of extensive nævi and moles have been recorded, but they need not detain us here.

Lymphangiectodes, also known in this country as Lymphangioma circumscriptum (Malcolm Morris), is an affection which was first described by English writers. It is a localised disease consisting of closely aggregated deep-seated vesicles looking like frog-spawn, occurring in patches about various parts of the body—face, lip, neck, trunk, and limbs, but limited to one region. Apart from the grouped vesicles, these may also occur scattered about in twos and threes. They vary in size from a large pin-head to a hemp seed or so; and may be either colourless, pinkish, or red, or even of a blackberry hue (hæmorrhagic). They may occur as closely aggregated lesions, situated on an infiltrated patch, of a fibro-fatty consistency to the feel. Vesicles of a similar nature have also been observed on the conjunctivæ and tongue, and when pricked lymph-like fluid flows.

The disease begins in infancy or early life, and is chronic. Microscopically, the striking features in the sections are the dilated, cystic-looking, lymphatic spaces in the corium.

The designation of lupus lymphaticus employed for this condition by Hutchinson is misleading, for the disease has nothing whatever to do with lupus.

The treatment is to excise the patch or destroy it with caustics. In either case a recurrence may take place in the neighbourhood of the resulting scar. Radcliffe-Crocker has had some success with electrolysis. But it may be wise to leave it alone altogether, unless thorough excision can be carried out. In the latter case this might be followed up by grafting with the whole depth of skin from elsewhere, according to the method of Wolff; or even Thiersch-grafting might be tried. It would depend on situation and other circumstances.

Lymphangioma tuberosum multiplex was the name first used by Kaposi for another unusual condition, and although subsequent research has shown the condition to arise from the blood-vessels of the skin, and not to be of lymphatic origin, it is as well, perhaps, to stick to the original accepted designation rather than adopt such a name as hæmangio-endothelioma tuberosum multiplex.

The disease is a very rare one, and consists of nodules about the size of lentils or larger; round, or, in the case of the larger ones, oval; of a yellowish to brownish red hue and smooth surface. They are moderately elevated above the level of the skin. In distribution they may affect the front of the body, upper part of chest, the back of the neck. The disease usually begins in childhood or youth, but it may begin later in life. The nodules increase slowly in numbers and more slowly still in size.

Microscopically, the striking feature is the number of cyst-like formations containing a colloid mass. Descriptions vary according to authors, but the growth appears to be really an endotheliomatous one springing from the vessels. It must be distinguished from epithelioma, adenoides cysticum, or, as Unna has named it, *Acanthoma adenoides cysticum*.

Myoma Multiplex Cutis.—This is another very uncommon disease. It has been especially studied by Besnier. These small tumours of the skin vary roughly from a pin-head to a bean in size, and firm to the touch. They are some shade of red or yellowish-red in colour, but they may be pale like the skin. The growths are usually grouped. They have been observed on

[1] I am indebted to Mr. Harold Wilson, Teacher of Pharmacy, University College Hospital, London, for pointing out that 8 is a better solution than 10 per cent, as in the latter the thiosinamin is thrown down in part and necessitates heating of the solution before using, whereas an 8 per cent solution remains clear. The formula is: Thiosinamin, 8 parts; glycerine, 20 parts; sterilised (distilled) water, 72 parts.

the trunk, limbs, back, over the lower jaw, etc.; and are embedded more or less in the skin, and are movable with the latter. Paroxysmal spontaneous pains may occur in them; they may also be painful on pressure. They appear at various ages in adults. Malcolm Morris has shown instances of these growths occurring in father and daughter.

Histologically the growths are made up of smooth muscle fibres, and no doubt originate from the arrectores pilorum. The van Gieson method of staining is a good one for sections. Excision is the only effectual treatment.

URTICARIA PIGMENTOSA is sometimes nodular, as in the case depicted by Radcliffe-Crocker. (*Urticaria pigmentosa* will be dealt with under Urticaria.)

XANTHOMA PALPEBRARUM is the name given to yellow plates of new growth which affect the eyelids. It usually begins about the internal canthus of the left upper eyelid, but it may start elsewhere—for instance, near the external canthus of the right lower eyelid. It then spreads, and later involves the other eyelids, in some cases forming more or less of a semicircle round the eyes.

There can be no difficulty about the diagnosis. The plates are embedded in the skin, and are of the colour of chamois leather. The disease shows a family prevalence and may be hereditary.

XANTHOMA MULTIPLEX is more rarely seen. It affects, in addition to the eyelids, the flexures and mucous membranes, and in severe cases may be widely distributed, involving also the elbows and knees, palms and soles, and so forth. It is often associated in adults with chronic jaundice, malignant disease of the biliary apparatus, and so forth, but in youths there may be no derangement whatever of the general health, and nothing more than the yellow growths about the knees and elbows, which give no trouble. In the latter case it may occur in more than one member of a family. When the lesions, instead of being flat plates, are rounded, prominent, nodular, the disease goes by the name of xanthoma tuberosum, to distinguish it, clinically merely, from xanthoma planum.

There are differences of opinion as to the pathology. Microscopically the growth consists essentially of masses of "xanthoma" cells in the corium. The yellow and brown granules are partly in the ordinary connective-tissue cells, partly in the epithelioid cells, partly free.

Xanthoma elasticum (Balzer), or *pseudo-xanthoma elasticum* (Darier). This is a rare condition, in which Balzer found that elastin formed the greater part of the tumour; but more recently Darier concluded it was not xanthoma, as he did not find "xanthoma" cells.

The treatment of xanthoma consists in excising the growths. Great care must be exercised in dealing with the eyelids so as not to produce a condition of ectropion or epiphora. But most patients will prefer to be left alone.

COLLOID-MILIUM of the skin was first described by Wagner. The growths, which vary from a pin-head to a split pea in size, are glistening, of a pale yellow tint, and embedded in the skin, especially about the cheeks, orbits, and forehead. A jelly-like mass can be got out of them when opened. It is extremely rare.

BOTRYOMYCOSIS. — Under this name softish tumours which occur singly about the hands have been described. The growths are pedicled, about the size of a Barcelona or hazel nut, and are some hue of red with exuding granulations on the surface. They are painless.

A case of this kind came under my observation when I was assistant to Sir Victor Horsley at University College Hospital in 1892. The patient was a woman of about fifty, with a pedicled growth the size of a Barcelona nut on the dorsum of the terminal phalanx of the left little finger, just behind the nail. It had begun as a pimple which had been cauterised several times; its surface was blackish when I saw it. At that time I did not know what it was, but I removed it in the out-patient's department under an anæsthetic, taking a circle of healthy skin round the base of the pedicle with it. It was dressed with sal alembroth gauze, and it did well.

Since Poncet and Dor first described it in 1897, there has been some discussion as to its cause, but everything points to its being of staphylococcal origin, and in this the veterinary surgeons agree.

Tumultus.—Irregular action or commotion, *e.g.* of the heart (*T. cordis*) or of the speech (*T. sermonis*).

Tunbridge Wells. *See* BALNEOLOGY (*Great Britain, Chalybeate*); MINERAL WATERS (*Chalybeate*).

Tunic or Tunica.—A coat or covering or enveloping membrane, such as the tunics of the eye, uterus, bladder, or blood-vessel, etc.; *e.g.* the *tunica adventitia* (of blood-vessels), *tunica costalis* (the pleura), *tunica fibrosa* (of a Graafian follicle), and the *tunica vaginalis testis*.

Tunicata. *See* HEART, PHYSIOLOGY OF (*Comparative Anatomy, Tunicata*).

Tuning Fork. *See* EAR, EXAMINATION OF (*Investigation of Hearing, Tuning-Fork Test*).

Tunnel-Anæmia. — Ankylostomiasis or Miner's anæmia, called tunnel-anæmia because of its frequency among the workers on the St. Gothard tunnel. *See* PARASITES (*Nematodes, Uncinaria duodenalis*).

Tunnel Disease. *See* SPINE, SURGICAL AFFECTIONS OF (*Caisson Disease*).

Tunny. *See* SNAKE-BITES AND POISONOUS FISHES (*Thynnus or Tunny*).

Tupelo. — A material (*nyssa aquatilis*) used for making tents. *See* GYNÆCOLOGY, DIAGNOSIS IN (*Cervical Dilators, Tents*).

Turacin.—A crimson colouring matter. *See* PIGMENTS OF THE BODY AND EXCRETA (*Colouring Matters of the Lower Animals*).

Turacoporphyrin. *See* TURACIN.

Turbinal.—A bone of spongy texture and consistence (Lat. *turbo*, a top), *e.g.* the sphenoidal turbinals or bones of Bertin.

Turbinated Bones. — The scroll-shaped bones of the skull known as *conchæ inferiores* (inferior turbinated), also the turbinated processes of the ethmoid bone (*concha superior, concha media*); they are covered by a peculiar soft erectile and vascular mucous membrane; excision of a turbinated bone (*turbinectomy*) or incision of it (*turbinotomy*) is occasionally performed. *See* NOSE, EXAMINATION OF (*Anterior Rhinoscopic View*); NOSE, DISEASES OF NASAL ORIFICES AND SEPTUM (*Hypertrophy of Turbinals*).

Türck's Bundle.—A bundle or tract of nerve fibres which passes from the cortex of the brain (temporo-sphenoidal lobe) to the internal geniculate body, passing through the pons.

Türck's Column.—The anterior or direct pyramidal tract.

Türck's Trachoma. — Laryngitis sicca. *See* LARYNX, ACUTE AND CHRONIC INFLAMMATIONS (*Laryngitis Sicca*).

Turkey. *See* BALNEOLOGY (*Turkey*).

Turkish Bath. *See* BALNEOLOGY (*Historical*); HYDROPATHY.

Turmeric. — The rhizome of *Curcuma longa*; it is a condiment, and it used to be official in the B.P.; unsized paper which has been steeped in tincture of turmeric may be used as a test for alkalies (being turned brown by them).

"Turn of Life." *See* MENOPAUSE.

Turpentine. *See also* ASEPTIC TREATMENT OF WOUNDS (*Disinfection*); DERMATITIS TRAUMATICA ET VENENATA; DRUG ERUPTIONS (*Erythematous*); LARDACEOUS DEGENERATION (*Etiology*); NEPHRITIS (*Etiology*); URINE, PATHOLOGICAL CHANGES IN (*Odour*).—Oil of turpentine (Oleum Terebinthinæ). Obtained by distillation from the oleo-resin (common turpentine) of *Pinus sylvestris* and other species of *Pinus*. It is a colourless fluid with a strong peculiar smell. It contains various hydrocarbons (terpenes) all having the formula $C_{10}H_{16}$. It is very inflammable, and takes fire if mixed with any strong mineral acid. It dissolves resins, sulphur, and phosphorus. It is insoluble in water, but soluble in alcohol, ether, and chloroform. *Dose*—2-10 m.; 3-4 ʒ as an anthelmintic. *Preparations*—1. Linimentum Terebinthinæ. 2. Linimentum Terebinthinæ Aceticum.

Externally turpentine is used as a counter-irritant in bronchitis, pleurisy, arthritis, etc. It is usually applied to the chest in the form of a stupe or hot cloth which should be removed as soon as it causes discomfort. The liniments are useful for rubbing over joints, and in lumbago, sciatica, myalgia, etc. Turpentine has been employed as a parasticide in ringworm. Sanitas contains turpentine which has been allowed to oxidise in the air, its active principle being peroxide of hydrogen. Oil of turpentine is used internally in certain abdominal conditions for the purpose of overcoming tympanites. It may be given as an enema (1 oz. to 15 oz. of mucilage of starch), and by the mouth (30-60 m. in mucilage). As an anthelmintic it is given in starch in large doses followed by castor oil. It may arrest intestinal hæmorrhage, as in typhoid fever, and bleeding from the stomach, as in gastric ulcer. In persistent diarrhœa during convalescence from typhoid fever it is of great benefit. It is not much used as an inhalation in respiratory diseases. In giving turpentine internally its liability to cause inflammation of the kidneys must always be borne in mind, and it should never be administered to patients suffering from nephritis. Plugs of gauze or lint soaked in turpentine may be employed for styptic purposes in bleeding from deep wounds or cavities. It is less irritating than perchloride of iron and has the advantage of being readily obtainable in emergencies.

Turpeth or Turpethum. — The dried root and stem of *Ipomœa turpethum*, used as a powder, in doses of 5 to 20 grains, for the same purposes as jalap; there is a *tinctura jalapæ composita* which contains one part of turpeth to eight of jalap and two of scammony; it is official in the Indian and Colonial Addendum to the B.P. of 1898. The glucoside which is its active constituent is called turpethin ($C_{34}H_{56}O_{13}$).

Tussiculation.—A frequent, dry, and hacking cough (Lat. *tussis*, cough).

Tussis Convulsiva. *See* WHOOPING COUGH.

'Tween-Brain. — The diencephalon or inter-brain, consisting chiefly of the optic thalami. *See* EMBRYOLOGY (*Fourth Week, Brain*).

Twelfth Nerve. *See* HYPOGLOSSAL NERVE.

Twins. *See* PREGNANCY, MULTIPLE. *See also* LABOUR, OPERATIONS (*Embryotomy, Complex Labours*); TERATOLOGY (*United Twins*).

Twitching. *See* BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Œdema, Symptoms*); URÆMIA (*Symptoms*).

Tyloma.—A thickened patch of skin due to hypertrophy of the stratum corneum of the epidermis; a callus (Gr. τύλη, a swelling).

Tylophora Leaves.—The dried leaves of *Tylophora asthmatica*, having the same sort of action as ipecacuanha; the dose is ¼ to 2 grains as an expectorant, and 15 to 30 grains as an emetic; it is official in the Indian and Colonial Addendum to the B.P. of 1898.

Tylosis.—A state of callosity of the skin, especially of the palms and soles (*tylosis palmæ vel plantæ*), which would seem sometimes to be hereditary; also, a form of blepharitis. *See* ICHTHYOSIS (*Tylosis*); PREGNANCY, INTRA-UTERINE DISEASES OF THE FŒTUS (*Tylosis Palmæ et Plantæ*).

Tympana. *See* TYMPANUM.

Tympanic.—Relating to the tympanum of the ear; *e.g.* the tympanic membrane, artery, nerve, notch, and ring. *See* EAR, EXAMINATION OF (*Tympanic Membrane*); EAR, AFFECTIONS OF TYMPANIC MEMBRANE; EAR, INFLAMMATION OF MIDDLE; EAR, MIDDLE, CHRONIC SUPPURATION; HEARING, ARTIFICIAL AIDS TO (*Artificial Tympana*).

Tympanites.—Distension of the abdomen, due to flatus in the bowel or air in the peritoneal cavity. *See* COLON, DISEASES OF (*Dilatation*); INTESTINES, SURGICAL AFFECTIONS OF (*Obstruction, Diagnosis*); PERITONEUM, TUBERCULOUS PERITONITIS (*Symptoms*); TYPHOID FEVER (*Symptoms*).

Tympanitis.—Inflammation of the tympanic membrane of the ear.

Tympanum.—The drum of the ear, or, simply, the middle ear (*plural, tympana*). *See* TYMPANIC and *Cross References*.

Type, Menstrual. *See* MENSTRUATION (*Phenomena, Type*).

Typesetters. *See* TRADES, DANGEROUS (*Lead-Poisoning*).

Typewriter's Cramp. *See* NEUROSES, OCCUPATION (*Writing*); SPASM (*Varieties*).

Typhlitis.—Inflammation of the cæcum. *See* APPENDIX VERMIFORMIS (*Appendicitis*); TYPHOID FEVER (*Diagnosis*).

Typhlo-.—In compound words *typhlo-* (Gr. τυφλός, blind) means relating or belonging to the cæcum; e.g. *typhlolithiasis* (the formation of calculi in the cæcum), *typhlostenosis* (stenosis of the cæcum), *typhlotomy* (section of the cæcum), etc.

Typhoid.—Resembling typhus (Gr. τῦφος, mist or stupor), *e.g.* the typhoid state seen in fevers where the nutrition is seriously interfered with; also, especially *typhoid fever* (*q.v.*).

Typhoid Fever.

DEFINITION 177
GEOGRAPHICAL DISTRIBUTION . . . 177
ETIOLOGY 177
Bacillus 177
Mode of Infection 177
Immunity 178
MORBID ANATOMY 178
PATHOLOGY 180
Serum Diagnosis 180
SYMPTOMS—
Signs of Invasion 181
Signs of Established State . . 182
Symptoms in Special Organs, e.g. *Intestine, Spleen, Kidney, etc.* . . 182
General Symptoms 185
Temperature 185
COURSE OF DISEASE 186
COMPLICATIONS AND SEQUELÆ . . . 186
RELAPSES 192
VARIETIES 193
TYPHOID IN CHILDREN 194
DIAGNOSIS 194
PROGNOSIS 197
TREATMENT 199
Preventive 199
General 200

See also ALCOHOL (*Indications*); APPENDIX VERMIFORMIS (*Appendicitis, Diagnosis*); APPENDIX VERMIFORMIS (*Typhoid Ulcers*); BLOOD (*Widal's Reaction*); BONE, DISEASES OF (*Osteomyelitis due to Typhoid Bacilli*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Thrombosis, Causes*); BRONCHI, BRONCHITIS (*Etiology*); CHOLERA, NOSTRAS; CHOLERA, EPIDEMIC (*Symptoms*); COLON, DISEASES OF (*Simple Colitis, Diagnosis*); DIAZO-REACTION; EPIDEMIOLOGY (*Seasonal Fluctuations*); FÆCES (*In Typhoid Fever*); FOOT AND MOUTH DISEASE (*Diagnosis*); GLANDULAR FEVER (*Diagnosis*); HÆMATOPORPHYRINURIA (*Etiology*); HÆMOGLOBINURIA (*In Fevers*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Endocarditis, Simple, Causes*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Malignant Endocarditis in Typhoid Fever*); HICCOUGH (*Etiology*); HIP-JOINT, DISEASES OF (*Pyogenic Diseases, Causes*); IMMUNITY (*Typhoid*); INFECTION (*Rules for Prevention*); INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties*); JOINTS, DISEASES OF (*Pyogenic, Causes*); LEUCOCYTOSIS (*Leucopenia in Typhoid Fever*); LUNGS, GANGRENE OF (*Etiology*); LUNGS, VASCULAR DISORDERS (*Embolism, Causes*); MALARIA (*Diagnosis*); MEASLES (*Complications*);

MELÆNA (*Causes*); MENINGES OF THE CEREBRUM (*Simple Meningitis, Causes, Diagnosis*); MENTAL DEFICIENCY (*Accidental, Post-Febrile*); MILK (*Pathological*); NEPHRITIS (*Etiology*); NERVES, PERIPHERAL (*Neuritis, Causes*); NERVES, MULTIPLE PERIPHERAL NEURITIS (*Etiology*); NOSE, DISEASES OF NASAL ORIFICES AND SEPTUM (*Perforation of Septum, Causes*); NOSE, ACCESSORY SINUSES, INFLAMMATION OF (*Etiology*); OVARIES, DISEASES OF THE (*Hæmorrhage, Causes*); PAROTID GLAND, DISORDERS OF (*Inflammation, Secondary*); PERITONEUM, ACUTE PERITONITIS, GENERAL (*Etiology*); PNEUMONIA (*Bacteriological, Pneumococcus and Typhoid Bacillus*); PNEUMONIA, CLINICAL (*Etiology*); PREGNANCY, INTRA-UTERINE DISEASES OF THE FŒTUS (*Infectious Fevers*); PURPURA (*Symptomatic*); SCROTUM AND TESTICLE, DISEASES OF THE (*Orchitis, Typhoid*); SEPTICÆMIA (*Diagnosis*); SEWAGE AND DRAINAGE; SNAKE-BITES AND POISONOUS FISHES (*Oysters, Typhoid*); SPINE, SURGICAL AFFECTIONS OF (*Typhoid Spine*); SYPHILIS (*Secondary, Typhoidal State*); THERAPEUTICS, SERUM-THERAPY (*Anti-enteric Serum*); TOXICOLOGY (*Shell-fish*); TROPICS, UNCLASSED FEVERS OF (*Bastard Typhoid*); UNDULANT FEVER (*Synonyms*); URINE, PATHOLOGICAL CHANGES IN (*Fevers, Typhoid, Albuminuria*); URINE, BACTERIA IN (*Typhoid Fever*); WATER (*Diseases due to Impure Water*).

SYNONYMS: *Abdominal Typhus; Enteric Fever; Gastric Fever; Seven-Day Fever, etc.*

DEFINITION.—A continued fever caused by the entry of a specific micro-organism—the bacillus typhosus—or of poisons arising from it—into the blood; anatomically characterised by hyperplasia, and usually ulceration of Peyer's glands, enlargement of spleen and of mesenteric glands; clinically manifested by some or all of the following symptoms:—Pyrexia, roseolous eruption, enlargement of spleen, evidences of general toxæmia, and symptoms due to local lesions situated for the most part in the intestinal and mesenteric glands.

GEOGRAPHICAL DISTRIBUTION.—It is endemic in Great Britain, but is lessening in frequency under the influence of improved hygienic conditions. It is met with throughout Europe, Asia, Africa, and America.

ETIOLOGY.—Discussion as to the proximate cause of typhoid fever has become superfluous since the discovery by Eberth in 1880 of the specific bacillus typhosus. This organism has been found in the spleen, the mesenteric glands, the lymphoid tissue of the intestines, the medulla of bones, the kidneys, the liver, the roseolous eruption, and in the blood of the dead and of living subjects of typhoid fever; it has also been detected in the placental blood, and in the organs of the new-born fœtus of a mother affected with the disease.

It abounds in the typhoid stools during the first fortnight of the malady, sometimes persisting throughout convalescence; it is to be found in a considerable percentage of cases in the urine after the first few days, frequently in the bile, and probably also in the sweat and the sputa. It is found in the pus from some, but not all, abscesses occurring in the course of the fever, and it may persist in such abscesses for even years after the disease.

Characters of the Bacillus.—The bacillus is 2-3 μ in length and 1-8 μ in thickness, longer and thicker than the typical bacillus coli, to which it bears a close resemblance; when stained shows ten to twenty-four wavy flagellæ, longer than the bacillus itself; the termination may show a glistening rounded body, really retracted protoplasm—in cultivations it is single; often in chains. Though readily distinguishable microscopically from the typical bacillus coli there are many forms of the latter greatly resembling the bacillus typhosus. Cultivations grow in the ordinary nutritive media, and rapidly develop in milk, beef-tea, or butter. The bacilli are killed when exposed to a temperature of 60° C. for even twenty minutes, but several observers have seen cultures of the bacilli in beef-tea alive for weeks at a freezing temperature, and they preserve their vitality in ice.

They usually soon disappear from ordinary water, but have been found in it, as in the Maidstone epidemic of 1897.

They usually soon die in typhoid stools, but may remain alive for months in sterilised stools; experiments of Andrews showed they rapidly die out if planted in sewage.

They retain their vitality under suitable conditions for months or years, even when dried, which supports the supposition that the disease may be transmitted by dust.

Mode of Infection is mainly through the alimentary canal; and the most common carrier of the poison is infected drinking water from wells or streams, or pollution of a general system of water supply. Instances of epidemics which were clearly traced to the water supply are numerous, the latest example on a large scale in England being the Maidstone epidemic of 1897, in which the water supply of a certain area was contaminated by surface drainage. Many so-called "milk epidemics" are due to water used to clean the cans or to dilute the milk.

Milk is a favourable medium for the growth of the bacillus, and many attacks due to milk infection are recorded.

Food.—Oysters eaten raw may certainly act as carriers of the poison, and the germs have been found in them derived from the water in which they are fattened; epidemics have been described due to other shell-fish; the disease may be conveyed by vegetables eaten uncooked. There is accumulating evidence that the disease

does affect other animals than man, and it is said that typhoid fever has been contracted through eating the flesh of animals suffering from typhoid fever.

Sewage Emanations.—It is probable that the gaseous emanations from cesspools or drains infected with the typhoid virus may, if inhaled, produce the disease; for though sewer gases rarely contain typhoid forms, which probably perish in the presence of the other organisms of sewer contents, there is no impossibility that under special circumstances the typhoid poison may be contained in drain effluvia, and give rise to the disease.

Contagion.—There is undoubted evidence that under favourable conditions the disease may be transmitted from one person to another. Sir William Jenner attributed typhoid occasionally to the inhaling of the patient's breath by students closely watching the rise of the mercury in the clinical thermometer in the patient's mouth in the days before the instrument was provided with an index; but it certainly does not in this country spread far through the medium of the air; possibly in countries where dust clouds are common and where typhoid is epidemic it may be so conducted to a distance.

The stools and urine containing the infecting organism may transmit it; attendants on typhoid patients and those employed in washing the soiled linen occasionally contract the disease, especially if insufficient care is taken in washing the hands, and inefficiently disinfecting excreta and nursing utensils, and bedding contaminated by excreta.

The disease is said to be conveyed by flies and other insects. Proofs of this are wanting.

The poison enters the body in nearly all cases by the alimentary canal, for even if inhaled it may pass from the pharynx into the digestive tract.

In many cases it is quite impossible to trace the source of infection.

Age.—It has been observed in infants even as young as 4½ months, but it is not common before the third year; it is apt to be mild in children, and probably it occurs in children more often than is generally stated. In the Maidstone epidemic 22 per cent of the subjects were children. The period of greatest susceptibility is from 15 to 25 years; half the cases occurring in these ten years; after this it gradually decreases in frequency, and is uncommon after the age of 50, but has been observed even at the age of 90 years.

Sex.—There is little or no difference in the susceptibility of the sexes.

Family and Individual Tendency.—There is a marked idiosyncrasy to contract typhoid fever in some families, or to suffer severely from it; on the other hand there is said to be individual immunity. Though an individual may possess immunity at any moment, there is no proof that such immunity is perpetual; and in typhoid, as in the other exanthemata, an individual may go unscathed for years while exposed to conditions most favourable for contracting it, and contract it at a later period when such risks appear much less. The condition of bodily health here as in other diseases is a factor in immunity.

Condition of Health.—Perfect apparent health is consistent with the contraction of typhoid fever, but in my own observation it has occurred with a frequency after injuries and accidents too great to ascribe to mere coincidence.

Season of the Year.—It occurs chiefly in the autumn, and has been called autumnal fever; this is noticeable in America as well as in England; the period of greatest mortality is in October or November, the minimum mortality is usually in April. In autumns succeeding a very hot summer it appears particularly common. The varying prevalence at different seasons has been attributed to the sinking of the level of the ground water from the surface of the soil.

Immunity.—As already said, some individuals appear to possess an immunity against typhoid fever, though such immunity may not be a perpetual one.

There is a general belief that the disease protects against a second attack; this belief is founded (*first*) on the existence of records of outbreaks affecting almost every one in the house or locality, who has not already had the disease, and sparing those who had been attacked in a visitation years before in the same locality; (*secondly*) on the fact that few patients coming under observation with typhoid fever admit having had a previous attack; (*thirdly*) on experimental proofs that typhoid protects against subsequent attacks founded on the investigations of Widal, Kitasato, and others; guinea-pigs which had been infected by cultures of typhoid bacilli were more or less immune to the action of a stronger virus, and the blood serum of animals thus rendered immune conferred immunity on other animals. Relapses, however, are very frequent in typhoid fever, which to some extent argues against acquired immunity. Many persons probably who get typhoid while children cannot remember this when adults, and as a matter of fact persons are known to have two or even three or more attacks of typhoid fever. In the Hamburg epidemic of 2000 typhoid patients, 14 had had previous attacks, a proportion which, though cited as a proof of immunity, is by no means inconsiderable compared with the incidence of typhoid in the population generally. In the South African campaign numerous individuals suffered from more than one attack of typhoid fever, and there is no reliable evidence that typhoid fever confers marked immunity against subsequent attacks.

Morbid Anatomy.—The specific lesions of

typhoid fever are mostly found in the intestinal track, mesenteric glands, and spleen.

Small ulcers may occur in the mucous membrane of the pharynx and the *œsophagus*.

The *stomach* may show catarrhal changes, and rarely small ulcers or hæmorrhages.

In the *small intestine* the changes are chiefly in the lower part of the ileum, but may be in its whole length, and even in the jejunum and duodenum. The solitary follicles and Peyer's patches become hyperæmic, and the mucous membrane becomes reddened with increased mucous secretion. The hyperæmia subsides, and the follicles and patches become turgid and prominent, constituting pale elevations above the general surface of the intestine. The swelling is due to a great increase in lymphoid cells which even infiltrate the adjacent mucosa. The cells are for the most part nucleated, and there are other larger multinucleated cells; the lymphatic vessels are dilated, and contain proliferating endothelial cells; the blood-vessels are compressed. In many of the swollen follicles and patches the swelling gradually subsides, the contained cells becoming granular or fatty and becoming absorbed. Some of the follicles, especially in severe cases, become the seat of sloughs, soft yellow, or grey, and varying in thickness; the sloughs may involve the mucous membrane only, or all the muscular coats of the intestine; they are formed of granules, fibrin, corpuscles, and micro-organisms; the sloughing process appears due to the action of the typhoid and other micro-organisms.

Ulcers result from this sloughing process; these ulcers may be minute and circular from sloughing of the solitary follicles, or oval with their long axes corresponding to that of a Peyer's patch, and consequently to that of the intestine. The depth of the ulcer depends on the thickness of the detached slough; sometimes it is quite shallow; usually it extends into the muscular coat or deeply to the peritoneal coat. The ulcers are mostly irregularly oval, corresponding to a part only of a Peyer's patch. The edges of the ulcer are thin, undermined, usually ragged, and float out in water. The base of the ulcer is usually smooth after detachment of the slough. Sometimes the ulcers are serpiginous. Healing occurs by the formation of granulations in the floor of the ulcer, and the epithelium grows gradually over the ulcer from the edge and from a smooth cicatrix sometimes depressed with little, if any, contraction, and is recognisable for years after the typhoid attack. In the hyperæmic stage crowds of typhoid bacilli may be seen by staining and in the lymphatic vessels of the follicles.

There may be only one ulcer usually near the ileo-cæcal valve, or the ulcers may be very numerous; and the greater part of the mucosa of the last foot of the ileum may be a brown-black eschar; or the terminal 6 or 8 inches of the mucous membrane of the ileum may form a large ulcer in which here and there are islets of mucosa. Often ulcers are seen in the lower part of the small intestine, and hyperæmic, and raised patches higher up in its length. Sometimes most of the ulcers have been healed up before death occurs from other causes.

In about one-third of the cases the cæcum and colon are affected. The cæcum may have very large irregular ulcers within it; in some cases the colon is the seat of ulcers along nearly its whole length—small circular ulcers which occasionally perforate: this may occur where there are no ulcers in the ileum. There may be no ulceration from first to last, even in cases of long duration which come to the post-mortem table, and it may fairly be inferred that this is even more likely to occur in slight cases which never come to the post-mortem table. The vermiform appendix may be inflamed or ulcerated.

Perforation of ulcers occurs from the thinned wall of the intestine rupturing at the seat of an ulcer, or from a sloughing process; occasionally it occurs in a partly-healed, or even in a completely cicatrised ulcer.

Mesenteric Glands.—The glands swell from hyperæmia and cell infiltration; those glands especially are involved most directly connected with the ileum, and so too are the retro-peritoneal glands. The swelling subsides as the infiltrated cells get fatty and become absorbed, but the glands may soften or actually suppurate and rupture, producing peritonitis, sometimes fatal; there is sometimes considerable gland enlargement with very little intestinal ulceration. The presence of typhoid bacilli in the mesenteric glands is almost constant.

The Spleen is nearly always enlarged, and may, in late stages, weigh as much as 20 ozs., and be 3 or 4 times the normal size; it is relatively most enlarged in children. The capsule is tense, and the pulp dark and soft with undue distinctness of the Malpighian bodies. Infarcts or abscesses may form in it which may give rise to rupture or to suppurative peritonitis. Typhoid bacilli are nearly, if not always, to be found in the spleen.

The Liver may be enlarged and pale or hyperæmic; the liver-cells may be granular. The biliary ducts are often inflamed, and also the gall-bladder, which may contain pus or its wall be gangrenous. Aggregations of typhoid bacilli are nearly always detectable in the liver, and the bacilli are often present in the gall-bladder.

The Kidneys.—Cloudy swelling of the epithelium of the cortical tubes, or signs of acute hæmorrhagic nephritis, or infarcts or microscopic abscesses may be present. The typhoid bacilli are found only exceptionally in the kidneys.

Heart.—The myocardium may be pale and

soft with granular or fatty changes, or the "myocardite segmentaire" of Renaut.

Endocarditis and pericarditis are rare. Typhoid bacilli have been found in the myocardium and in the vegetations on the valves.

Blood-Vessels.—Arteritis, and more commonly phlebitis, with typhoid bacilli in the walls of the vessels, occur and give rise to thrombosis, and may be gangrene.

Respiratory Organs.—The changes in the respiratory organs are indicated by the various affections described under the head of "Complications." The specific bacillus may be discovered sometimes in the lungs, but it is much more common to find other micro-organisms.

Nervous System.—The symptoms of nerve disturbance in typhoid fever are more often due to toxic blood conditions than to organic disease, but meningeal inflammation, cerebral softening, and peripheral or spinal cord changes may occur, and are described under the head of "Complications."

Muscles.—Hyaline degeneration of muscles occurs in typhoid as in other fevers, and it sometimes leads to rupture of muscles and consequent hæmatomata or abscesses.

Bones.—The changes in the medulla and periosteum of bones are not special to typhoid fever, but the typhoid bacillus is not infrequently found in the pus in abscesses in these structures.

PATHOLOGY.—The bacilli, for the most part, gaining entry into the body by the alimentary canal, invade the lymphatic tissues beneath the mucous membrane of the intestine, and pass through the mesenteric glands into the blood, spleen, and other organs. It is to the poison resulting from the bacilli, the exact constitution of which is not known, that are due most of the symptoms of typhoid fever. The poison is, for the most part, intracellular, and in the bacilli themselves, filtered cultures being of little power; but there is evidence that the toxins from the bacilli may diffuse into the culture medium or into the fluids of the body. The typhoid bacilli are themselves pyogenetic, but many of the local lesions and the toxic symptoms are producible by other micro-organisms, the growth of which becomes more active in presence of the typhoid bacillus. Various organisms take part in this mixed infection, chiefly the streptococcus, the staphylococcus pyogenes, the bacillus coli communis, and the pneumococcus. The chief poison produced by the typhoid germ appears to be an albuminoid, and has been called typhotoxin. Speculations have been published as to difference in the symptoms produced by the products of the typhoid, and of other bacilli in the blood; most often, probably, the symptoms are due to mixed toxæmia.

Dr. Alexander Paine, of St. Mary's Hospital, has kindly written on "The Serum Diagnosis of Typhoid," as follows:—

The action which the blood of individuals suffering from typhoid has on cultivations of the typhoid bacillus is a valuable aid in the recognition of that disease.

If the serum of the blood of a patient suffering from typhoid is added to a young broth-culture of the typhoid bacillus, the organisms lose their motility, aggregate in clumps (agglutination), and ultimately settle to the bottom of the liquid as a sediment, leaving the supernatant fluid clear. If the serum from a case other than typhoid is added, the bacilli remain actively motile, there is no sedimentation, and the fluid retains its turbidity.

This property of agglutinating organisms which the blood acquires in disease has been under consideration for many years. Its application to the diagnosis of this disease was made by Widal in 1896, hence it is known as Widal's Reaction.

The reaction appears as early as the seventh day, or even earlier; it lasts throughout the course of the disease, and may be obtained for an indefinite period after convalescence is established, often many months.

Other fluids of the body possess this property of agglutination—a reaction may be obtained from the milk, tears, and urine of typhoid patients, also from the fluid of blisters and other serous effusions. Drying of the blood does not destroy this power, though it diminishes it.

In a small percentage of cases (3 per cent) of individuals suffering from typhoid the serum fails to agglutinate the bacilli; but all cultures of the organisms do not react equally to typhoid serum—not only are there different species of the organism, but in the same species varying conditions of growth may alter its characters in this respect.

Since a reaction, though incomplete, may be given by the serum of a patient not suffering from typhoid—and even by the serum of a healthy individual if it is present in too large a quantity—it has been found necessary to dilute the serum when testing its agglutinative power. Low dilutions are not reliable for this reason; on the other hand, with an excessive dilution a typhoid serum with feeble agglutinative power may fail to give the reaction.

Various dilutions are used, but it is generally held that with a dilution of 1 in 30 there is a minimum of risk.

Application of the Test.—A small quantity of blood taken (with every precaution to avoid contamination) from the ear or finger of the patient is drawn into a capillary tube and allowed to clot. The serum which separates may be tested in the following manner:—

1. *Microscopically (rapid method).*—The serum is diluted with normal serum or normal saline by means of a graduated capillary pipette or a sterilised platinum loop, till half the required dilution is obtained.

A loopful of the diluted serum is added to a

loopful of a 24-hours-old culture of the typhoid bacillus in neutral Bouillon, and the mixture examined under the high power of the microscope by the hanging-drop method.

If the patient is suffering from typhoid the organisms will become motionless and adhere together in clumps. This may take place immediately, but it usually requires from a few minutes to half an hour for the agglutination to be complete. Under exceptional circumstances it may be delayed for one or two hours.

If the patient is not suffering from typhoid the organisms remain actively motile for hours, and do not form clumps.

2. *Microscopically (sedimentation test).*—This method, although it requires longer time for its completion, has the advantage that it can be observed with the naked eye.

The dilution of the serum and its addition to the bacterial culture can be most accurately made in the straight capillary tube suggested by Wright. The serum is drawn up to a fixed mark on the capillary tube and blown out into a glass capsule. Normal serum or normal saline is next drawn up to the same mark a number of times in succession, and mixed with the serum until half the number of dilutions is obtained.

Equal quantities of this mixture and a 24-hours-old broth culture of the typhoid bacillus is aspirated into a bulb at the upper end of the tube, and, after well mixing, is blown out in one long column into the capillary tube, the end of which is sealed off.

A control tube containing an equal quantity of the bacterial culture and normal serum or normal saline is made in the same manner.

The two tubes are kept upright for 12 or 24 hours—at the end of that time, if the serum is taken from a patient suffering from typhoid, the fluid becomes clear—the organisms forming a flocculent sediment at the bottom of the liquid. The fluid in the "control" remains uniformly turbid, and there is no sedimentation.

[*The Nature of Infection with the Typhoid Bacillus.*—It has recently been definitely shown that Eberth's bacillus is constantly present in the blood stream from the onset of an attack of typhoid fever. By using appropriate cultural methods it can be demonstrated in nearly all cases during the first week of the disease; during the later stages (third and fourth weeks) it is less frequently present, and it disappears as the temperature falls. If relapse occurs the bacilli reappear. The organism can always be detected in the blood before the Widal test can be determined. Two further points recently established are that in typhoid patients the organisms are excreted by the liver into the bowel along with the bile, and that they may enter the gall-bladder and go on growing there. Typhoid fever, therefore, should be regarded as a bacillæmia with secondary invasion of the intestines. This theory harmonises with the previously known fact that typhoid bacilli cannot be recovered from the fæces during the early period of the disease, as well as with the more recent discovery that they can sometimes be isolated from the blood of persons who are only in the incubation stage. Bacilli which enter the gall-bladder set up a certain degree of cholecystitis, which favours their perpetuating their existence in this organ, and after recovery from the disease patients may harbour virulent bacilli in their gall-bladders for months or even years. Förster has demonstrated bacilli in the motions of patients ten and twenty years after infection, and such cases are known as "chronic infection carriers." They number from 2 to 3 per cent of all cases of typhoid. That this prolonged infectivity of the bile may be very important from the public health point of view is shown by the claim of Blumenthal and his co-workers in Leipzig, that out of a series of 386 cases occurring during a period of eighteen months 77 patients had been in more or less close contact with "bacteria carriers." Similar observations have also been made in this country. It is obvious that these facts may explain the sporadic occurrence of typhoid fever in districts which have long been free of the disease. When bacilli carriers are discovered they should be warned of the danger they are to others, and instructed as to the necessity for cleansing the hands, care in cooking or handling food, and disinfecting the stools. No efficient means of disinfecting the gall-bladder is known; when it is diseased it is advisable to urge its removal, as there is some reason to suppose that typhoid cholecystitis predisposes to gall-stones.]

Symptoms

Incubation Period is the interval between the moment of infection and of the invasion. Each of these events is so often indefinite that in many cases it is not possible to exactly determine the duration of the incubation period; most commonly it is from eight to ten days, but it may be only one or two days (very rarely), or as long as twenty-three days.

There are seldom symptoms during the incubation period, but headache, loss of appetite, sense of fatigue, or sleeplessness may occur.

Symptoms of Invasion.—The invasion is usually gradual; the most constant and earliest symptom, in fact the symptom from which is usually dated the invasion, is *pyrexia*. The temperature rises first in the evening 1·5 to 2°, and on each succeeding day increases by 1°, the evening temperature maintaining the excess of 1·5 or 2° which it gained by commencing to rise before the morning temperature; after four, five, or six days of such step-like ascent the temperature stands at 101° to 103° in the morning and at 103° to 105° in the evening; the evening temperature then ceases to rise,

but the morning temperature rises one day later than the evening (having commenced a day later), and there results a morning temperature of 102° or 103° or 104° and an evening temperature of 103° or 104° or 105° according to the severity of the attack.

Exceptions to this gradual mode of rise occur. Not very uncommonly the rise of temperature is abrupt, reaching 103° or 104° in the first twenty-four hours. Sometimes, especially in children, the temperature may, though daily increasing, drop to normal during some part of each day, and in very rare cases the invasion has been without pyrexia, as has the whole course of the disease.

Headache usually comes on early, slight or of any degree of severity; usually frontal, rarely occipital or all over the head, or it may extend down the neck; a tensive dull pain with sometimes acute paroxysms; it usually lasts through the invasion period into the real period, but may cease earlier. *Convulsions* are very rare.

Epistaxis occurs in about half the cases, once or repeatedly, slight or considerable.

Chills rarely, rigors may occur; *deafness* may be an early symptom.

Discomfort, rarely pain, tenderness, and gurgling in the right iliac fossa may occur, and *laxity of bowels* is common. There is usually a feeling of languor and weakness, and the patient thinks he has "caught a chill" or got a "bilious attack."

Tonsillitis may be an early symptom.

The onset instead of being gradual may be very abrupt, with sudden pyrexia, nausea, repeated vomiting, and pains in the abdomen, simulating acute peritonitis or gastritis. Or it may begin with high fever, chills, profuse sweats, headache, and signs of severe bronchial catarrh, simulating acute tubercular phthisis.

Other cases begin with symptoms and signs—apical or basic—of acute croupous pneumonia. Some such cases are due to the bacillus typhosus, and have been called "pneumotyphoid."

Again, the initial symptoms may be severe headache, vomiting, high fever, retraction of head, vomiting, convulsions, and delirium. Usually these symptoms soon subside, being toxic only; but in others they persist and are due to actual meningitis, other signs of which, such as squint, paralysis, become evident later. The bacillus typhosus is accountable for some such cases which have been called "cerebral typhoid."

Occasionally the disease begins with acute maniacal delirium or with "confusional insanity," marking other evidences of the disease, and such patients have in consequence been sent to asylums.

In a few cases typhoid begins with symptoms of acute nephritis with blood and albumen in the urine, and other evidences of typhoid become marked later. These cases have been called "nephro-typhoid."

And cases are described by Salamon beginning with acute pleurisy due to infection of the typhoid bacillus—pleuro-typhoid.

Symptoms of the established disease are continuous with those of the invasion, gradual or abrupt as the case may be: the diarrhœa and headache continuing, the patient becoming more languid and weak, and eventually taking to bed at a date depending on the severity of the attack, the disposition of the patient, and the advice he receives or follows. When the onset is abrupt he may take to bed at once, but more often it is only after three or four days or a week, when the disease is suspected rather than diagnosed, sometimes much later, when he is quite exhausted, and in the "latent" form he may keep about even after perforation has occurred.

Alimentary Canal.—The tongue, at first moist and slightly furred, tends to gradually get more thickly coated, and after fifteen or sixteen days may get dry brown, baked, or even fissured, sometimes bleeding. If the temperature does not fall after about twenty-one days, tenacious exudation may collect around it, and it becomes difficult of protrusion; in general it tends to become dry much in proportion to the amount of diarrhœa. The tongue usually begins to clean and become moist as the temperature falls and convalescence comes on, but it may do so much earlier, cleaning entirely at the end of eight or ten days, though the disease runs on for the usual period. The tongue may be clean throughout even in fatal cases. Sometimes it becomes glazed and smooth.

Sordes are apt to occur on teeth and lips, which are often sore and bleed from picking.

Herpes on the lips or face is very rare without pneumonia or rigors.

The *pharynx and palate* may in severe cases become coated by a whitish exudation like thrush or diphtheritic membrane, and containing chiefly streptococci and staphylococci.

The breath has usually a *characteristic odour* even early in the disease; *thirst* is common, especially when pyrexia or diarrhœa or melæna is severe. *Anorexia* comes on early, lessening with defervescence, and before there is apyrexia the craving for solid food may become very great. Hydrochloric acid is deficient in the *gastric juice*; *nausea and retching* may, as already said, be initial symptoms, or they may come on later from peritonitis with or without perforation, from uræmia or other causes; the stomach is apt to become much *distended* from the liquid diet, recumbent position, and impaired digestive processes.

Tympanites is usually little marked during the first few days, and may be moderate throughout, or may become extreme, gradually or suddenly. Sometimes the tympanitic belly is tense and drum-like; in other cases is soft and doughy, muscle tonus being apparently lost

from toxæmia of the nerve centres. Tympanites is usually greatest when diarrhœa is severe (Hare finds the reverse). With subsidence of the disease the tympanites subsides; but the belly may become retracted when the patient is becoming exhausted (intestinal distension is often simulated by distension of the urinary bladder or of the stomach).

The "*bell sound*" may occur over parts of much distended colon. When general over the whole abdomen it indicates perforation of intestine.

The iliac fossa is often the seat of discomfort and tenderness, rarely of much pain. Sometimes *severe pain* is felt about the iliac region, indicating deep ulceration, and in many cases points to ulceration of the large intestine (severe pain may occur in the lower part of the belly from retention of urine).

Gurgling in the iliac fossa is common, but is not special to enteric fever.

Respiratory movements are often noticeably lessened over the lower part of the abdomen on the right side.

The *bowels* may be greatly relaxed, the motions being as many as twenty-four in the twenty-four hours, or there may be constipation throughout. Often they are relaxed at first from the administration of purgatives, and afterwards constipated, or the reverse may occur. Diarrhœa is certainly much less frequent now than Murchison's estimate some years ago of 95 per cent of cases. Osler found it in 30 per cent of his cases. In the Maidstone epidemic it occurred in half the cases. Hare finds constipation more often than diarrhœa. It appears to vary in different epidemics, and is certainly lessened by early and careful dieting. Murchison doubted any relationship between the amount of diarrhœa and the severity of the intestinal lesion, and certainly constipation may be associated with deep ulceration; but in general, when intestinal ulceration is slight or absent, diarrhœa is at its minimum, and when there is extensive ulceration there is almost always diarrhœa. The stools are unformed even though there is no diarrhœa. They may be at first dark, but soon become light yellow in colour (occasionally green, like chopped parsley), liquid, and offensive. They separate on standing into a deposit of debris of food, epithelium and biliary constituents, with a supernatant alkaline and albuminous clear fluid. The bacillus coli and bacillus of Eberth are found in them. At any time after the first eight or ten days they may contain shreds of larger portions of slough, sometimes with intestinal muscle fibre. Blood may be passed early from congestion around Peyer's patches, or later on from open ulcers (*vide* Complications). There is a tendency to involuntary passage of fæces, accounted for often by the liquid nature of the stools, and the abdominal distension, together with the mental hebetude; sometimes from complete unconsciousness or from paralysis of the sphincter.

The *spleen* may enlarge, sometimes very greatly, rarely sufficiently to ascertain clinically for the first week or so. The outline is sometimes easily felt, and in thin children may project the abdominal wall. It appears to vary in size from time to time, and its percussion dulness varies with the distension of the colon. Even when no enlargement can be detected its position is often tender on pressure. It may remain large some time after the temperature has fallen to normal, and it usually enlarges during relapses or exacerbations. Infarcts and abscesses may occur in the spleen (*see* Complications). The typhoid bacillus is obtainable from the spleen by puncture.

Under the name "spleno-typhoid" Eiselt describes cases in which the spleen is the chief organ affected, and intestinal symptoms are absent. In some of these cases the spleen becomes of enormous size, and there is pyrexia of a relapsing type.

The pulse is usually quickened from the first, being usually 85 to 100 per minute in males, and 100 to 120 in females (Dreschfeld). Its maximum in 100 cases of Murchison's was 110 to 120. It varies in frequency at different periods of the day, and is readily accelerated by slight causes, mental emotion, etc. Attacks of typhoid occur in which the pulse is slow throughout. As the temperature falls with the subsidence of the disease the pulse-rate falls also; but in bad cases, when the fall of temperature is only temporary, the pulse-rate often keeps up. The pulse is usually dicrotic and of low pressure from the first, but this character may be quite lost in late stages. In long lasting cases the pulse tends to get more and more feeble, and running or thready, and its beats become very rapid or uncountable (150 or 160 per minute). When hæmorrhage is about to occur it usually gets rapid, short, and irritable.

In late stages the pulse is apt to become irregular or intermittent, especially in children, and during convalescence it may become very slow, as is the case in other exanthemata, and influenza.

In some cases of typhoid the pulse is very feeble from the first, and there is blueness of finger ends.

There is nothing peculiar to typhoid in the character of the heart sounds. They tend to become weak as the heart progresses, and, it being a long lasting fever, with many conditions contributing to toxæmia and to drains on the tissues of the body, the action and sounds of the heart may become extremely weak; but typhoid mostly affects persons who are young and in whom the heart has reserve force enough to carry them through the longest and most severe illness.

Syncopal attacks, even fatal, may occur

during convalescence, and in some such cases little structural change has been found in the heart substance. They are probably attributable as much to want of blood in the body as to loss of heart power.

Blood in Typhoid Fever.—The red blood corpuscles in the late stages are much decreased, falling to even 2½ million per cmm., and the hæmoglobin is correspondingly lessened. The leucocytes also become much lessened in number, sometimes numbering only 1000 per cmm. (Huyens). This decrease is most marked in the late stages of the disease. According to Coe the fibrin of the blood is diminished in typhoid fever, the exact contrary of what occurs in other fevers. The blood plates are diminished in number.

All these results show the deterioration of the quality of the blood in typhoid fever, and there is in the disease a progressive anæmia. The actual volume of the blood becomes lessened; and especially when there has been much diarrhœa, sweating, and hæmorrhage, the vessels become extremely empty. In many cases, as will be found in opening veins for injection of salines, the veins are over full and the arteries quite empty. The tendency to bloodlessness in severe typhoid cases is an important point to be remembered in treatment. Fatal results attributed often to cardiac failure are really due to blood failure.

Epistaxis may occur not only during the incubation period, but later on, even as late as the twenty-ninth day (da Costa). It may then be a part of a tendency to general hæmorrhages from the mucous membranes (*see* Complications).

Respiratory System.—The respirations are accelerated by the pyrexia and blood condition. At the onset of the disease moist sounds are usually audible in the larger bronchial tubes, and later on there is a tendency to hypostatic congestion of the lungs. In some cases more severe affections of lung or pleura occur (*see* Complications). As already said, typhoid fever may begin with acute pneumonia due to the bacillus of Eberth (pneumo-typhoid) or more rarely with pleurisy (pleuro-typhoid).

Renal System.—The urine is lessened in quantity and febrile in character; during convalescence the quantity is often increased and phosphates deposited. Albuminuria occurs in about one-quarter of the cases, usually slight in amount, and due to pyrexia and to toxins in the blood, rarely to the typhoid bacillus; the urine may be very offensive.

Typhoid bacilli are detectable in the urine in about 25 per cent of the cases, not often before the twelfth day, sometimes in quantity sufficient to produce turbidity of the urine. It is probable that bacilli pass through the kidney into the bladder, and there multiply to produce this condition, and *retention* of urine, which is extremely common in typhoid fever, may favour this process. Other renal complications are described under that head.

Ehrlich's Reaction.—Ehrlich's test depends on the production of a pink colour and froth when the urine of a typhoid patient is treated with his solutions. These are two in number (1) acid hydrochloric one part, distilled water twenty parts, sulphanilic acid to saturation, (2) a half per cent solution of nitrate of sodium in distilled water. The test consists in adding to a specimen of the urine an equal quantity of liquid made up of the two solutions in the proportion of forty parts of No. 1 solution to one part of No. 2 solution, and then adding liquor ammoniæ to alkalinise the solution. In cases of typhoid fever the urine becomes of a deep ruby-red colour, and on shaking the test-tube a delicate pink froth is produced. It is not claimed this pink froth is produced only in the urine of typhoid fever, but it is very constant in such urine.

Nervous System.—The degree of affection of the nervous system varies with the character of the epidemic, the intensity of the poison, the idiosyncrasy of the patient, the condition of his secreting organs, and the associated secondary infection. It is rare for the patient to be suddenly prostrated by the onset of typhoid fever as by typhus fever or influenza, but this may occur especially in children. Most often the early nerve symptoms are headache, sleeplessness, and lassitude; the headache usually lasts the first ten days, but it may persist longer, sometimes coming on again late in the disease; the sleeplessness usually passes off with the headache and gives place to undue drowsiness. The mental condition may remain clear throughout even a long and fatal case, more often there is some degree of hebetude, with slowness of apprehension and delay in speech, though what is said is quite reasonable. In some cases the mind seems unusually active, the patient answering questions and volunteering information with great minuteness of detail. Often with a tendency to drop into heavy sleep, the patient is perfectly clear when roused. Delirium occurs in many cases, varying from a little "light-headedness" at night to loud, noisy, very active or low muttering delirium. Sometimes it resembles delirium tremens. Delirium does not often occur before the tenth day, that is, after cessation of the headache; the two together are rare; it may occur early, even on the very first day. Delirium may occur in cases with little intestinal disturbance, in fact in some cases toxic nervous symptoms predominate over all other symptoms. In cases which go on after the twenty-fourth day or thereabouts, delirium is rarely absent, in many cases it comes on with septic complications, especially pneumonia; in some is a symptom of local intracranial disease (*see* Complications). It may come on during convalescence, like acute mania; sometimes

acute maniacal attacks alternate with perfectly quiet intervals. In severe cases stupor or actual coma occurs, and in some cases there is coma vigil or coma with wide-open eyes and constant movements of hands or tremor of limbs.

A condition of trance may occur or of temporary aphasia; in some a dazed state due apparently to loss of memory of where they are or who is attending on them, and often when the disease is over there is no recollection of anything that occurred. The pupils may be dilated in typhoid fever, but they are very often not so, and may be contracted.

Deafness may be one of the earliest symptoms of typhoid, or come on at any time in the disease. Sometimes it is due to administration of quinine, sometimes to middle-ear disease, but most often appears to be a purely nervous affection.

Tremor of tongue is mostly present, often also of limbs; both may become extreme. Much tremor is often associated with deep ulceration, but it may occur from toxæmia only. Subsultus tendinum or "carphology" may occur in protracted and severe cases, and may become associated with coma vigil, the patient lying in a stupor with open eyes—a condition mostly fatal.

The deep reflexes are often but not invariably increased, and may be absent even without peripheral nerve changes. Convulsions may initiate the disease in children but are rare in adults except from uræmia or meningitis. (For other nervous phenomena, *see* Complications.)

General Symptoms.—There is nothing characteristic in the early period in the general aspect of the patient, but with the continuance of the fever there is a dull, heavy expression, especially when the headache is severe. The *decubitus* is usually dorsal, but children especially, and even adults, may lie on either side—in later stages the patient tends to sink deep into the bed. There is often a suffused look about the face, which may be pale or the cheeks may be flushed. In well-nursed cases and where diarrhœa or complications are absent, there may be little loss of flesh, but the emaciation in severe cases is extreme, with haggard face and sinking eyes. The daily loss of weight has been estimated at from two to seven ounces per diem. Children especially rapidly emaciate. The skin may be pungent, hot and dry, or there may be profuse sweats, even though the pyrexia is severe. It is said a peculiar semi-cadaverous odour emanates from the skin. I have noticed it only in cases with septic complications.

The eruption is present in a large proportion of cases. It consists of lenticular spots of delicate pink colour, about 2 to 4 mm. in diameter, very slightly raised, with margin shading into surrounding skin, fading on pressure. They may be slightly vesicular, they rarely appear before the eighth day of the disease. Each spot develops in a few hours, and lasts individually four or five days, fading gradually. They come out singly or in crops, varying in number from two or three to some hundreds. They chiefly appear on the back, or surface of chest or abdomen. Fading on pressure, they are not immediately visible on the back after the patient has been lying in the dorsal position, but become visible a few moments after he turns on his side. They are rarely seen on the limbs or face. In some cases the spots are deep purple in colour, though still disappearing on pressure. Sometimes they are actually hæmorrhagic; typhoid bacilli have been obtained from the spots. Very often before the true typhoid eruption or with it there come out numerous "typhoid blotches," carmine in colour, considerably raised above the surface, irregular in shape, and very much larger than true typhoid spots.

Chills or actual rigors may occur at the onset, or during the course of the fever (*see* Complications).

Temperature in Typhoid Fever.—In most cases the temperature rises a couple of degrees on the first evening, and for the next three or four or five days there is a daily increase of temperature of 1° compared with the temperature at the same time on the previous day. By this step-like ascent the temperature stands at about 102° in the morning and 104° in the evening of the fifth day, and the morning temperature continuing its increase a day later than the evening temperature, there results a temperature of 103° in the morning and 104° in the evening. Then ensues a period which has been called the fastigium, lasting till the commencement of the decline of the disease, in which the temperature continues at this level (sometimes rather less, sometimes more). The duration of this period is about twelve days, and the morning temperature then remits a degree or so, and falls back 2° below the temperature on the same evening; both morning and evening temperatures thus fall day by day each about 1°, till by the twenty-first day the normal is reached in the morning, and in a day or so later also in the evening. Then ensues a period of convalescence in which the temperature should be 1° or 2° below the normal for a few days, till with returning strength it again rises to normal.

The commoner variations in the course of the pyrexia are as follows:—

1. The temperature may rise abruptly with the invasion, reaching 104° or higher in the first twenty-four hours, and the fastigial stage begins at once.

2. The pyrexia may gain a degree daily, but may fall to normal at some time on each (or some) of these days.

3. The fastigium may continue much longer than the average of fourteen days, very fre-

quently another six or seven days, sometimes as long as fifty days.

4. The fastigium may be of much less than the average duration, the morning remission occurring after even four or five days, and apyrexia being attained by the twelfth or fourteenth day (abortive typhoid); in some cases the early termination may be by crisis.

5. There may be sudden, extensive, irregular rises or falls in the temperature; these may be due to complications, such as pneumonia, or hæmorrhage, or perforation, or septic absorption, or to slight excitement or to nerve disturbance. In some cases the irregularity in temperature is extreme, and without ascertainable cause other than to toxic disorder of the heat-regulating centres.

6. Hyperpyrexia may occur up to 110·3° in fatal cases, or to 107·8° consistently with recovery. It is in nearly if not all cases due to local complications and septic absorption.

7. The temperature may be intermittent throughout the disease.

8. It may remain normal or subnormal throughout the disease; such afebrile typhoid has been observed in epidemics, and is not less serious or free from the dangers of typhoid fever than other forms.

9. Occasionally the temperature may be higher in the mornings than the evenings.

10. The fall of temperature with cessation of the disease may be by crisis.

Course of the Disease.—The disease is very generally divided, for purposes of description, into periods or stages corresponding to epochs each of seven or of ten days' duration; but there is no anatomical or clinical ground for such division. The period at which ulceration of intestines begins varies within wide limits, and even if it did not, its commencement is unmarked by any recognisable symptom. The cutaneous eruption appears at very varying dates in different cases. The period of continued fever or fastigium is no constant duration, and there is no special change on the seventh day or on any multiple of the seventh day, nor is there any crucial day on which the disease is apt to end, though this often occurs about the twenty-first or twenty-eighth days. Such an idea appears to be a vestige of the time when typhoid and typhus fever were regarded as one disease, and spoken of as seven-day fever.

By a consideration of the history of the illness taken with the symptoms we can usually tell approximately about how many days the disease has lasted, and divisions into weekly stages are artificial and useless.

It is convenient to describe the invasion symptoms separately from those of the established disease, but as already noted there is no defined line between these periods. As the disease progresses the condition of tongue becomes worse as a rule, the pulse-rate quickens, the patient weakens and loses flesh, and the mind usually becomes more confused; symptoms, too, may be added due to complications.

The great majority of cases recover, the temperature gradually falling to subnormal, the stools becoming formed, and other symptoms subsiding till convalescence is established.

The duration of the disease varies considerably. As already mentioned it may abort, the symptoms and pyrexia subsiding after about ten days, possibly even earlier; but the diagnosis of a case of typhoid which aborts before the period at which the characteristic symptom appears must necessarily be open to doubt.

The average duration of 200 cases, which recovered, collected by Murchison, was 24·3 days, and over three-fourths of them lasted more than three weeks; there was no evidence of any critical day on which cessation most often occurred, though it was often *about* the twenty-first or twenty-eighth day. In exceptional cases the duration may be as long as sixty days.

In a minority of cases death occurs—from asthenia and bloodlessness, from toxæmia, from heart-failure, or as a result of the many complications which are apt to occur.

Though we may thus separate the causes of the fatal issue, they are not infrequently combined in practice; and many of the deaths resulting from typhoid fever are not due to the typhoid poison but to septicæmia consequent on infection by other organisms.

Statistics show that about 35 per cent of the deaths are due to local complications, the most important of which are melæna and perforation of intestine; the remaining 65 percentage of deaths are attributable to more general causes.

When the disease is long protracted the temperature is apt to become irregular in the late stages, the circulation to become feebler, and the patient to sink gradually from toxic coma, with faint, flickering pulse; extreme tremor of limbs, face, and tongue muscles; blueness of extremities, and rambling delirium, with all the evidences of the typhoid state and extreme irregularity of temperature range. In other cases the mind may remain fairly clear and the heart force may fail, and death occur from syncope—a very rare termination in typhoid. When death is due to local complications the special symptoms arise described under that head.

Sudden death may occur from myocarditis and unexplained causes.

Death rarely occurs in typhoid fever before the fourteenth day—the average duration of Murchison's *fatal* cases was 27·6 days. Hoffman found it to be 28·9 days.

Very rarely death occurs quite early, even on the second day.

Complications and Sequelæ. — *Digestive System.—Ulcers occur on the pharynx* (Bouvert);

sharp-cut ulcers may also occur on the palate and on the pillars of fauces, due to the streptococcus and staphylococcus.

Acute glossitis; also *gangrene of tongue* may occur.

Cancrum Oris is very rare; said to be commonest in children. It may invade the cheek, gums, and alveolar process of jaw, and is a serious but not necessarily fatal complication.

Parotitis is uncommon, is usually unilateral, occurs as a late complication or as a sequela, attributed to spread of inflammation along Stenon's duct or to metastatic inflammation; rather over half the cases are said to suppurate, and pus may escape through the meatus auditorius externus; the specific bacillus has been found in the pus. The entire gland may slough out. Thrombosis of the internal jugular vein may be produced and pyæmia. It is a serious but by no means fatal complication; the symptoms are those of parotitis from other causes, and the treatment is the same.

The œsophageal mucous membrane may be inflamed, and ulcers may form leading to stricture; submucous abscesses may occur.

Hæmatemesis is very uncommon, and probably arises from small ulcers or erosions of the mucous membrane; it may be fatal, as in a case under the author's observation in a young child.

Hiccough is sometimes obstinate; it may be due merely to flatulence, but if severe and persistent, suggests peritonitis or gangrenous changes in the intestine.

Peritonitis may result from deep intestinal ulcers without (or oftener) with perforation, from rupture of softened mesenteric glands, from splenic infarcts or abscesses, from rupture of gall-bladder, or of abscesses in the abdomen. It is indicated by its usual symptoms, preceded by symptoms special to the cause in the individual case. It may be localised and subside, or be rapidly spreading and soon fatal: the symptoms may be quite latent.

Perforation occurs in about 3 per cent of cases, accounting for about 18 per cent of the deaths from typhoid. It is most apt to occur in protracted cases with much diarrhœa or tympanites; melæna and pain in the abdomen, pointing to deep ulceration, often precede perforation. It is predisposed to by purgation, enemata, vomiting, sudden movements of the patient, by fighting the disease without going to bed, and by taking solid food too early.

It occurs as early as the eighth or as late as the hundred-and-tenth day, is uncommon before the fourteenth or sixteenth day, and is commonest between the twentieth and twenty-eighth days. It may occur during a relapse, during convalescence or long after convalescence appears completed. The perforation is usually in the ileum, rarely in the jejunum, occasionally the colon or the vermiform appendix; it may be a mere pin-point hole in the thinned intestinal wall or an actual sloughing of the floor of an ulcer. Usually there are ulcers in the intestine which are nearly or actually perforated, as well as that which has actually perforated.

The symptoms of perforation are equivocal in some cases, being those producible by other complications likely to happen in typhoid, and on the other hand they may be so slight that perforation is not even suspected.

There may be sudden acute abdominal pain with subsidence of previous tympanites, sudden fall in temperature and pulse force, faintness, pallor, vomiting, clammy sweats; death may very quickly occur; more often the patient rallies and peritonitis sets in, with shivers or rigors, recurrence of pyrexia, vomiting, increased distension of abdomen, rapid pulse, followed by hiccough, rambling delirium, sunken features, sipping breathing, and death from exhaustion and sepsis in two to twenty-one days, usually in four or five days.

When the intestine gives way by some small pin-point hole, the mutual pressure of distended coils of gut may prevent the escape of intestinal contents, except perhaps a little oozing. In rare instances local peritonitis may seal up such perforation and lead to recovery; more often local abscess forms with septic absorption and fatal results. In cases of perforation such as these, the symptoms may be of the slightest: merely a slight fall in temperature and a feeling of faintness; later on rigidity of abdominal muscles, pain, and symptoms of peritonitis, but the more prominent symptoms are usually those of septic absorption, rigors, high fever, hiccough, etc.; such cases may last longer than those in which there is diffuse peritonitis, but eventually die of toxæmia.

Intestinal Hæmorrhage occurs in a proportion of cases variously estimated at from 3 to 9 per cent. The statistics of the Hospitals of the Metropolitan Asylums Board provide by far the greatest total of cases from which an average can be deduced and place it at about 10 per cent.

It may occur as early as the fifth or sixth day from congestion of the vessels around a Peyer's patch, but *severe* hæmorrhage does not occur till ulcers have formed, *i.e.* before the tenth day or thereabouts. It may occur at any time after this, most often between the twentieth and twenty-eighth days (or, according to Osler, between the fourteenth and twenty-first days); it usually arises from an opened arterial twig, sometimes from a vein.

The quantity may be a mere streaking of the motions or as much as several pints, bright red or dark coloured, in sudden rushes or gradual oozing, with or without clots; is often repeated again and again.

It is commoner in cases where there has been diarrhœa than where there has been constipation, and in cases where pain has been complained of. It may occur in apparently slight, but is more common in severe, and especially in protracted cases.

There may be no warning of even severe melæna; a sudden drop in temperature may precede it, or accompany it, or follow it. The fall in temperature may precede the melæna, probably occurring at the time of the escape of blood from the vessels, though this blood may not be voided for some hours later. Copious hæmorrhage may occur without any remission of pyrexia, and in any case it usually soon rises again after the hæmorrhage. Repeated or copious bleedings are followed by anæmia, prostration, pallor, and quickened pulse-rate.

The mortality in cases of hæmorrhage has been estimated at from 30 to 50 per cent of the cases. Even when not the direct cause it is a powerful contributory cause of death, by draining away blood from the patient and predisposing to toxæmia.

Mesenteric Glands may soften and burst, setting up peritonitis; recovery or death may follow. The symptoms are often indistinguishable at the onset from those of perforation of the intestine, and probably some cases of supposed perforation and recovery were of this nature.

Fungel records perforation of the superior mesenteric artery by a suppurating mesenteric gland.

Splenic Infarctions are not very common, they may lead to abscesses which extend to the surface, and with or without bursting set up peritonitis and septicæmia. *Rupture* of spleen is recorded. *Hypertrophy* of spleen may follow typhoid.

Ascites I have seen in one case; it cleared up.

Appendicitis.—The cases in which a true typhoid ulcer has been found in the appendix are few, but occasionally appendicitis does occur in typhoid fever; and Hof found 8 cases of perforated appendix in 808 fatal cases. Sometimes abscess occurs around the appendix, and bursts externally.

There is some, though not very certain evidence, that typhoid fever may account for appendicitis occurring long afterwards.

Persistent Dysenteric Diarrhœa may be a sequel of typhoid, sometimes it is due to serpiginous ulcers round the intestine.

Cicatrisation of Ulcers with constriction of the gut is a rare sequela of typhoid.

Liver.—A few cases of solitary abscess of liver due to bacillus coli, rarely to bacillus of Eberth, are on record, even fewer cases are known of multiple abscesses; these are of pyæmic origin from abscess elsewhere. Cases of suppurative pylephlebitis are recorded.

Cholecystitis.—The bacillus coli and the specific bacillus are frequently to be found in the gall-bladder, the latter Chiari, indeed, believes rather as the rule than the exception; it has been found in the gall-bladder as long as fourteen years after the typhoid. It may give no symptoms, but sometimes cholecystitis results and empyema of the gall-bladder, forming a large tumour with or without jaundice, which may perforate and prove fatal; three such cases have been operated upon successfully. It is believed that the bacilli of Eberth in the gall-bladder may be the starting-point of the formation of gall-stones.

Jaundice may arise in typhoid from catarrh of the bile ducts, or from toxic changes in the liver-cells of the nature of acute atrophy, or from cholecystitis or cholangitis; the streptococcus pyogenes and the bacillus coli have been found in the biliary ducts.

Circulatory System.—Endocarditis, with the specific bacilli in the vegetations occasionally, pericarditis still less commonly occurs. The *myocardium* is commonly soft, friable, and pale yellow, with fatty or parenchymatous change. Extreme softening of heart muscle is rare, and even in cases in which death has been attributed to syncope, the heart fibres may show little change; some such deaths are due to *intracardiac thrombosis* or to thrombi or emboli in the coronary arteries; some are due to extreme bloodlessness of the patient.

Hæmorrhages from the mucous membranes, hæmoptysis, gingivitis, hæmatoma, menorrhagia, and epistaxis may occur, and occasionally purpuric blotches in the skin. Such cases are grave, the bleeding arising from disorganisation of the blood.

Thrombosis may occur in the heart, veins, or arteries. Intracardiac thrombosis may cause sudden death, and is a not uncommon source of infarcts in the viscera (Keen).

Venous Thrombosis is not uncommon during convalescence, most often in the veins of the left lower limb, especially of the calf. Typhoid bacilli have been found in the walls of the vein and in the clot, and probably cause the thrombosis, but it is predisposed to by the feebleness of circulation during convalescence, and occurs mostly in cases which are not very severe, but in which a slight persistent pyrexia delays an increase of patient's diet. The symptoms are the same as thrombosis produces apart from typhoid fever, and usually gradually clear up, but the clot may spread up into large veins, even up the inferior vena cava. Death may result from pulmonary embolism or pyrexia. Occasionally several attacks of thrombosis occur each with rigors, great pyrexia, and profuse sweats.

Thrombosis may occur in the intracranial veins.

Occlusion of arteries is much less common than once thought. Keen collected records of

46 cases, Ferard and Arden 23 and 17 others respectively. Six cases have occurred in my own observation. The obstruction is sometimes from thrombosis, sometimes embolic. Bacilli have been found in the clot and in the arterial wall, and thrombosis appears due in most cases to bacillary invasion of the arterial wall. The arteries of the lower limb are those most often affected. It is manifested by sudden pain in the limb, with lessened or no pulsation in the distal part arteries, coldness, or in most cases rise of temperature, in the local tenderness of the artery at the seat of occlusion, and sometimes loss of power in the limb; also pyrexia, or sudden increase of previous pyrexia. It occurs most often in the third week of the disease, but may be as late as the seventh week. It may entirely clear up, or the limb may remain weak and swelled for months after, or gangrene may result, usually dry (unless both vein and artery are occluded), and death may occur, or recovery after operation.

Gangrene has occurred in the feet, ears, labia, penis, cornea, etc.

Thrombosis may occur in the cerebral arteries and produce convulsions or paralysis.

Respiratory System.—*Lobar pneumonia* may occur at various periods in the disease. In a few cases it has been proved due to the bacillus typhosus.

1. Typhoid fever may begin with the symptoms and signs of acute lobar pneumonia (the initial rigor and rusty sputa are not always present), which quite obscure all other evidences of typhoid fever. After about six days the pneumonia subsides by lysis or by crisis, but the temperature immediately rises again, and there become manifest other and more usual symptoms of typhoid fever, which runs through its usual course. The term pneumo-typhoid has been applied to these cases, but it is seldom that the pneumonia is proved to have been due to the specific bacillus.

2. An attack of pneumonia may occur ending by crisis, but three or four days later an attack of typhoid may commence and go through its usual course. In such cases there is probably a simultaneous mixed infection, the pneumonia developing and completing its course before the typhoid, with its longer incubation period, develops.

3. At any time during the course of typhoid fever pneumonia (at apex or base of the lung) may occur as a complication, with well-marked physical signs, and with a considerable increase in the previous pyrexia, and may subside by crisis, just as in non-typhoid cases, producing sudden collapse symptoms. After the crisis the temperature of the typhoid reasserts itself, and the latter disease continues its course. Sometimes such an attack of pneumonia may come on just as the typhoid is passing away, constituting, as it were, a relapse.

4. Hypostatic pneumonia of insidious character, with percussion dulness and fine true crepitations, but without tubular breathing, may come on at any time in typhoid, most usually in late stages, and may gradually invade increasing portions of lung or lungs. Such cases are probably septic in origin, and are very fatal.

Bronchitis.—In nearly every case catarrhal sounds are audible in the larger bronchial tubes at the commencement of typhoid fever, and they often persist throughout its course, and are of no serious import. But *acute bronchitis* may occur at any time in the disease Sometimes it comes on with the invasion, and, with the headache and high fever, simulates acute tuberculosis. In such cases it usually soon passes off, but when it comes on late in the disease and spreads into the smaller tubes, and is associated with congestion of the lungs, it is a serious complication, and accounts for a good many deaths.

Pleurisy is less common than bronchitis or pneumonia. The typhoid bacillus has been found in the pleura in only a few cases. Typhoid pleurisy may remain "dry" or result in clear or purulent effusion; according to Osler usually the latter when it occurs in convalescence. Empyema may result from abscesses in the lung following pneumonia.

Pulmonary abscesses, sometimes multiple, may result from pneumonia, and may lead to empyema or to pneumothorax.

Broncho-pneumonia was found by Hoffmann in 38 of 250 autopsies in typhoid. It comes on usually in the later stages of the disease.

Hæmorrhagic infarctions occur in the lungs, probably arising from detached thrombi in the right heart, and sometimes from septic collections in other organs.

Gangrene of lung may occur as a sequel of pneumonia.

Hæmoptysis may occur with no discoverable lesion post-mortem. It is sometimes part of a general tendency to a hæmorrhage.

Pneumothorax has occurred from pulmonary abscesses, or abscesses behind the larynx bursting into the pleura.

Tubercle and Typhoid Fever were formerly said to be mutually antagonistic, but typhoid may occur in the subject of acute or chronic phthisis; moreover, the latter may first become evident, if not actually develop, during convalescence from typhoid. Typhoid fever is frequently recovered from when it occurs in phthisical patients, but it tends to increase the activity of the tubercular disease.

Laryngeal Complications. — Simple catarrh, with cough and husky voice, is not rare. Ulcers of the larynx, especially about the arytænoids and base or margin of the epiglottis, may occur during convalescence. Hoffner found them in 28 of 250 autopsies of typhoid cases, and they seem more frequent in Germany than in England. Some ulcers of the larynx are due

to other pyogenic organisms than the bacillus typhosus, but the latter is sometimes found in them.

Œdema of the glottis may result from ulceration, or perichondritis may do so, with expectoration of pus and parts of the laryngeal cartilages. Abscesses may also form in the subcutaneous tissues outside the larynx.

Paralysis of the vocal cords may occur during convalescence, with loss of voice, or dyspnœa, requiring tracheotomy. It is mostly due to peripheral nerve changes, and is occasionally a part of general peripheral neuritis.

Urino-genital.—Albuminuria, slight in amount and without symptoms, can scarcely be regarded as a complication, nor can the presence of bacilli in the urine in small numbers. But actual nephritis, with hæmaturia and casts, occurs occasionally. And cases are described and termed "nephro-typhoid" in which the disease begins with all the symptoms of a very acute nephritis, followed by other more characteristic signs of typhoid fever. Some such cases have even proved fatal by perforation of the intestine.

Hæmaturia may occur as part of a general hæmorrhagic tendency. In a case of my own it was fatal, and the autopsy showed it to arise from the mucous membrane of the bladder.

In some cases hæmaturia results from renal infarcts, in which are found the specific bacilli.

Cystitis may occur. Sometimes the colon bacillus or the streptococcus pyogenes or the staphylococcus is found in the pus, more often the typhoid bacillus itself; and probably the cystitis is due in such cases to a rapid multiplication of the bacilli in the urinary bladder, no doubt favoured by the difficulty in emptying the bladder, or the actual retention of urine so common in typhoid fever.

Renal abscesses and pyelitis may occur, also orchitis and epididymitis, sometimes going on to suppuration or sloughing of most of the testis; orchitis and parotitis have been observed together.

Ovaritis may occur, and the specific bacillus has been found in some such cases which suppurated.

Nervous System.—As already said, the nervous symptoms simulating meningitis, which come on with a typhoid attack or during its course, are usually toxic in origin, and not due to actual inflammation. But actual meningitis may occur in typhoid. It may initiate the disease with severe headache, vomiting, squint, retraction of head, etc. Such cases are very rare, still more rarely are they due to the presence of the specific bacillus. They have been spoken of as "cerebral-typhoid." As a complication of typhoid in its later stages meningitis is also very uncommon. It occurred in 11 of 2000 cases at Munich. Sometimes it is started by middle-ear disease. The streptococcus and pneumococcus, rarely the bacillus typhosus, have been found in the meningeal effusion. Meningitis in typhoid produces its usual symptoms, and often hyperpyrexia. Kernig's sign is useful in the diagnosis. It is quite possible some cases, even of meningitis, may recover, but it has been suggested that those cases which clear up are due to thrombosis of the cerebral vessels.

Hemiplegia, with or without convulsions, may result from thrombosis of cerebral vessels.

Aphasia may be a sequel or a complication, and is usually temporary only; it is said to be commonest in children.

Hysteria may follow typhoid; also a state like *trance*.

Epilepsy.—Typhoid in epileptics runs its usual course; the fits may occur during the illness, more often during convalescence.

Mental Defects.—After the attack the mind may be completely blank of all that occurred during a considerable period of the disease, and may remain so for ever. Sudden acute mania may occur in convalescence; chronic mania, melancholia, general mental weakness, various forms of delusion, may result from typhoid. They usually pass off in weeks or months.

Ocular Complications.—Catarrhal and phlyctenular conjunctivitis are the most common; suppurative keratitis, iritis, or cyclitis are occasional. Double optic neuritis and subsequent atrophy are recorded. Amblyopia has followed severe hæmorrhages, even without any ophthalmoscopic changes

Keen believes retinal hæmorrhages in typhoid are more common than thought. Cellulitis of orbit and paralysis of the extra-ocular muscles may occur.

Aural.—Otitis media may occur with its usual symptoms; the bacillus typhosus has not been found in connection with it.

Peripheral Neuritis is a not uncommon complication in the late stages: the symptoms had been noticed by many, but it was Nothnagel who first attributed them to peripheral nerve changes. The manifestations of peripheral neuritis in typhoid are sensory rather than motor disturbance, sometimes merely persistent numbness of the upper or lower extremities; in other cases intense, even agonising pains in the area of distribution of the sciatic, peroneal, ulnar, or other nerve.

The numbness or pains may last for weeks, and may be succeeded by anæsthesia, in one case lasting for eight years. Sometimes paralysis and atrophy of muscles occur, chiefly in the forearms. Contractions may follow: the palate has been paralysed, and the motor and sensory roots of the fifth nerve (Dreschfeld). Laryngeal paralysis may occur, necessitating tracheotomy.

Optic neuritis is excessively rare. Wednenske records a case in which symmetrical gangrene due to degeneration of peripheral nerves

occurred after typhoid. It is probably to peripheral neuritis that most cases of *paraplegia* after typhoid are due, but anterior cornual myelitis has been shown by post-mortem examination sometimes to follow typhoid.

"*Typhoid Spine.*"—Severe pain in the spine and the legs, especially on movement, may occur during convalescence; it seems to be a neurosis.

Tender Toes.—The tops and pads of the toes may become intensely sensitive; this passes off in a few days.

Tetany has been recorded.

Hyperpyrexia is very rare in typhoid fever. Of 200 consecutive cases at St. Mary's Hospital it occurred only twice; the same percentage was found by Bryant in 608 cases collected from *Guy's Hospital Reports;* Ord, Osler, Fagge, and the statistics of the Metropolitan Asylums Board all show the rarity of its occurrence. But rare as it is in typhoid fever, it is infinitely rarer as a complication in itself; it is nearly always due to some local affection. In one of my cases pneumonia, in two perforation and peritonitis, in one suppurative meningitis, and in one rupture of the gall-bladder, were found post-mortem. It should, then, never be assumed that hyperpyrexia is a complication in itself without a thorough search for some local affection to account for it. This may be of import in treatment, as the cold bath, which is the most efficient means of reducing temperature, might do harm if it were due to perforation or peritonitis; in such cases treatment must be directed to the local condition and the temperature meanwhile lowered by sponging, quinine, etc.

Rigors occasionally inaugurate the disease, and they are not very infrequent in its later stages. They occur from various causes, from peritonitis, with or without perforation of intestine, or from the onset of pneumonia; they occur repeatedly with repeated melæna in some cases, or with indications of thrombosis or embolism of vessels. In some cases they appear due to septic poisoning from insanitary conditions, and cease when the patient is removed to another dwelling, and in many cases they are probably due to septic absorption from some source. They have been attributed to constipation. In many cases they occur with increase of pyrexia which is quite evanescent and without constitutional disturbance; probably in these cases (mostly females) they are due to some slight disturbance acting on a heat-regulating centre, which has become unduly sensitive from toxic and febrile changes.

The significance of rigors as regards prognosis is nil; and the prognosis depends not on the rigors but on the gravity or unimportance of their cause.

Cutaneous System.—Lineæ albicantes on the abdominal wall may result from typhoid fever.

Alopecia is common and may not commence till after convalescence; the hair usually grows again.

The undermentioned cutaneous eruptions may occur:—

1. A uniform scarlet-red rash may appear in the early stages of typhoid. It is usually more marked on the limbs and shoulders than on the trunk, and is apt to form coalescent patches which point to its being an erythema.
2. Typical erythematous large plaques especially on the forearms.
3. *Tâches bleuâtres.*—Large maculæ of a purple colour—these are said to be always due to pediculi.
4. Purpura may come on, usually in late stages of severe attacks, but not necessarily.
5. Miliaria and sudamina occur here as they do in other diseases, especially if there be much sweating.
6. A morbilliform rash and urticaria have been observed.
7. Herpes is uncommon, and mostly indicates pneumonia as a complication.
8. Boils or carbuncles may occur during convalescence.
9. Occasionally pus vesicles like varicella or large bullæ.
10. Drug rashes from quinine, antipyrin, sulphonal, trional, etc.
11. Desquamation occasionally follows typhoid fever.

Gangrene may occur from occlusion of veins or arteries as already described, but it may occur without this, or at any rate without blocking of vessels of any but very small size; and is attributed by Keen to stasis of blood in the capillary circulation, perhaps also to fatty degeneration of the smaller arteries. In this way the penis, labia, or ear may become gangrenous; it usually comes on in early convalescence, and the pain is not so marked as in embolic gangrene, and a blue line of demarcation soon forms. Some such cases may be of peripheral nerve origin.

Bed-sores may occur, but rarely in well-nursed patients; they may destroy the ligaments of the sacrum and coccyx, and set up spinal meningitis.

Rupture of muscular fibre has been studied by Zenker. The muscle changes are not peculiar to typhoid fever, being a granular and waxy change, the fibre coming to resemble the flesh of fish; rupture may easily occur from cough or sudden effort. Ecchymoses and hæmatoma may occur, and the latter may suppurate. The muscles which most often rupture are the abdominal muscles, the adductors of the thigh, and, as Hoffmann showed, the diaphragm. There may be no symptoms, but sudden, intense pain may be set up, and when the abdominal muscles are torn peritonitis may be simulated.

Periostitis and Osteitis may affect any of the bones, most commonly those of the lower limbs,

sometimes the lower jaw; abscess may form in the medulla or beneath the periosteum. The streptococcus or staphylococcus is most often found in the pus, but Eberth's bacillus has been discovered in it, even years after the typhoid fever.

Necrosis, usually in the bones of the lower limb, may occur, attributed by Keen to thrombosis of the veins in the cavernous tissue of bones.

Arthritis may occur, sometimes rheumatic, affecting several joints, or septic, with other evidences of pyæmia. It may also arise from spread of osteitis into a joint, and there may be suppuration. Keen describes also a monarthritis most common in the lower limb, especially the hip; it usually arises spontaneously, sometimes from periostitis or osteitis extending into the joint. It comes on during convalescence insidiously with subacute synovitis; it usually subsides, but may go on to extreme distention of the joint and even to dislocation, especially in the hip in children.

Abscesses may form during typhoid fever in any part of the body—in the soft tissues, serous cavities, bones or joints, and even in the thyroid gland. Subphrenic abscess is recorded resulting from typhoid ulcers in the intestine, or from peritonitis or suppuration of mesenteric glands or from cholecystitis. The abscesses are mostly due to other pyogenic organisms than the typhoid bacillus, and in such cases must be looked on as of septic origin, but in the pus of some abscesses occurring in typhoid fever the specific bacillus may be found, especially in abscesses connected with the bones.

Pyæmia.—Cases of undoubted pyæmia, setting in with rigors, sweats, and other characteristic symptoms, occur, and with metastatic abscesses as a result of typhoid. And many of the dangers and complications of the disease are due to pyæmic infection, and not to the typhoid poison.

Marasmus.—Cases occur in which the patient, though taking food well, wastes after typhoid fever from non-assimilation. This is supposed to be due to atrophy of the ileum and the mesenteric glands; the patient may die after some months or live for years in this wasted state.

Obesity sometimes results after typhoid.

Anasarca may occur during typhoid without albuminuria.

Relapse.—Authors differ as to what constitutes a relapse. Murchison insists on a fresh eruption and fresh bout of pyrexia unaccounted for by any local conditions. Osler writes that two of the three symptoms—step-like ascent of temperature, roseola and enlarged spleen—should be present to justify the diagnosis. But there seems no reason to require for a relapse symptoms not considered essential for a primary attack, and just as the latter may occur without roseola, ascertainable enlargement of spleen or step-like pyrexia (or indeed any pyrexia at all), so may the relapse.

A relapse may be manifested by a period of pyrexia with diarrhœa, tympanites, headache, or other grouping of symptoms such as sometimes constitute a primary attack, and the diagnosis of such a relapse is always aided by the fact of its succeeding an attack of typhoid fever.

There is ambiguity also as to the time of occurrence of a relapse. Murchison speaks of it as occurring after convalescence is fully established, but opinions may differ as to what indicates complete convalescence. Osler's dictum that a relapse can only take place after complete defervescence must not be taken too strictly if it implies that the temperature has been absolutely normal before it occurs, for what can hardly be denied the name of a true relapse may occur some days after all symptoms of the first attack have subsided, except that the temperature has on some days slightly exceeded the normal. Irvine believed "intercurrent relapses" may occur without any apyretic interval, and Murchison regarded it as possible. It is certain there may be no *long* apyretic interval between attack and relapse; cases are recorded after forty-eight hours' interval, and if so why not after twenty-four hours? And one must admit a relapse may occur *immediately* after the subsidence of the primary attack; but this subsidence must not be judged only by temporary fall in the temperature, a temporary drop of temperature often occurring about the twentieth day with no amelioration of symptoms.

A relapse may be defined as a re-access of continued pyrexia, and of other symptoms of typhoid fever, coming on immediately or some days after the subsidence of the pyrexia and symptoms of the primary attack (or previous relapse).

This definition may perhaps allow some latitude, but not more than the varying phenomena of typhoid and the insufficiency of our knowledge require.

Frequency of Relapse.—Estimates vary from 3 to 18 per cent of cases, partly owing to the discrepancy in what is regarded as a relapse, partly owing to the varying nature of the epidemic. In some years the frequency of relapse is much greater than in others.

Among 6879 cases treated in the hospitals of the Metropolitan Asylums Board, the relapses averaged 8 per cent.

Relapse is said to be least common in children; to be predisposed to by constipation. Early administration of solid food is generally believed to predispose to relapse, and though this is denied, one sees a fresh access of fever, diarrhœa, quick pulse, tympanites, and general symptoms after giving solid food, too often to ascribe to mere coincidence. Such is specially the case where there has been constipation when

solids are first started. Chiari suggests dietetic indiscretions may lead to the discharge of specific bacilli from the bile ducts into the intestine. The cold-bath treatment is said to increase the frequency of relapse, but the curious epochs in which relapses become unusually frequent, point strongly to the character of the epidemic exerting an important influence.

Course of the Relapse.—It may begin with all the typical symptoms of typhoid fever, but the temperature more often ascends abruptly than in primary attacks, and rash is less often present, probably because a good many relapses dwindle away by the period at which eruption is due. A relapse may last five days or be long protracted, longer even than the primary attacks; in Murchison's 53 cases the average duration was fifteen days.

The fever and symptoms usually slowly subside, but hæmorrhage or perforation or pneumonia may occur. Flint regarded a relapse as more serious than the primary attack; the contary is the general experience; often complications which are septic set up pyrexia and simulate relapses.

Period at which Relapse occurs.—An analysis of 53 cases of relapse by Murchison showed the average period of intermission to be just over eleven days.

Cases are recorded of relapse forty-eight hours after the subsidence of the first attack; probably they may occur immediately after its subsidence. The period of intermission may be, on the other hand, very long; in one of my own cases it was twenty-six days, in another sixty days, and Goltdammer records an interval of ten weeks.

Multiple Relapses.—A second relapse is not very uncommon, and records exist of three, four, or even five relapses; each relapse is usually less severe than the preceding one.

Cause of Relapses.—Autopsies show that during a relapse fresh intestinal disease associated with the original older disease of the intestine. The part of intestine affected in the relapse is higher than that first attack. The mesenteric glands and spleen become enlarged again. The relapse can only be ascribed to re-infection from within, an occurrence conflicting to some degree with the belief that typhoid fever confers immunity against a second attack.

They have been attributed, with considerable reason, to a fresh absorption of toxins from the alimentary canal.

Recrudescences, exacerbations, spurious relapses, intercurrent relapses, are spoken of and much confused together.

A Spurious Relapse is a rise of temperature occurring during convalescence without the symptoms of a true relapse, and is due usually to nervous excitement or fatigue, or some slight local complication acting on a heat-regulating centre which is in a condition easily disturbed from the effects of the attack of typhoid.

An Exacerbation or Recrudescence.—After an improvement in temperature and symptoms, but without a fall of temperature to normal, the temperature ascends again, and the symptoms become more severe and sometimes more so than throughout the attack. Irvine regarded these as genuine relapses, the relapse overlapping the attack, and spoke of them as "intercurrent relapses.'

Forms of Typhoid Fever

The Mild Form.—There is pyrexia, usually commencing gradually and of moderate degree, and the symptoms generally are not severe. It may run a full course of twenty-one to twenty-eight days, or one of only ten or twelve days. The mildest cases are seen in children. It is to be borne in mind that a "mild" may develop into a serious case as it goes on.

The Severe or Grave Form may be severe from various causes: from the height of the fever, the severity of the intestinal lesions and symptoms, or from profound toxic effects, from the gravity of the complications, or from any combination of these causes. The grave form has been subdivided into many varieties—the hæmorrhagic, adynamic, bilious, ataxic, etc.—but however numerous we make these varieties, they will not cover all the combinations of types which occur in practice. Much of the gravity of many cases is due to other micro-organisms than the typhoid bacillus.

The hæmorrhagic form is not often seen now in England, and is characterised by hæmorrhages under the skin, and from mucous membranes. It is a most dangerous type of the disease.

Abortive Form.—Cases beginning gradually or abruptly (Turjensen says usually abruptly) may end prematurely with a gradual or critical fall of temperature at any time after the tenth day. It is said this may occur even earlier than the tenth day, but before this date the characteristic eruption can hardly have appeared, and the diagnosis can scarcely have been a certain one. (It has been said that in these cases the roseolous eruption occurred on the second to fifth day, but more probably the date of commencement of the disease had been determined wrongly.) These abortive attacks of typhoid may be followed by a relapse.

Ambulatory or Latent Form mostly occurs in the labouring classes who continue their occupation in defiance of symptoms throughout the whole course of the disease to recovery, or till some complication, such as melæna or perforation of intestine, compels their giving up. A young man presented himself in my out-patient department, with extreme tremor of lips and limbs, with extremely feeble pulse, and subnormal temperature, but quite clear in mind.

For some days he had felt weak, and on the previous night he had been delirious. He died in the hospital the next morning, the autopsy disclosing typhoid ulcers in the intestines, one of which must have perforated two or three days before death.

Apyrexial Form.—Cases are recorded which have run their whole course to recovery or to death without any pyrexia. Some such cases have been followed by relapses as in other forms of the disease.

Sudoral Type.—Jacqoud has described a form of typhoid under this title, commencing suddenly with severe headache, chills, and sweats; these symptoms may persist a long time, but the disease is usually of a mild type. Jacqoud believes such cases independent of malarial infection.

Typhoid Fever of Non-Enteric Form.—There is no doubt that typhoid fever may occur without any intestinal ulceration, and that the decrease must be regarded as a blood infection with local lesions, of which those in Peyer's glands are the most constant.

As already said, cases of typhoid fever occur commencing with acute pneumonia, or acute nephritis, or acute meningitis, or affections of other internal viscera. In some of these cases the local affection has been demonstrated to be due to the specific bacillus of typhoid, and the term pneumo-, or nephro-, or cerebral-typhoid has been applied to denote such attacks.

Such a nomenclature does not appear necessary or even descriptive of the phenomena that occur, for in such cases the disease does not manifest itself only by these local symptoms, but, as they subside, the more characteristic symptoms of typhoid become manifested, such as large spleen, diarrhœa, and other evidences of intestinal lesions of typhoid fever.

Typhoid Fever in Children.—As mentioned under the head of etiology, the disease is not uncommon even in young children. Of the symptoms of onset, *epistaxis* appears to be less common than in adults, *headache* is nearly always present, marked *somnolence* is more common at the onset than in adults, convulsions and nervous symptoms generally are more prominent, and delirium is often one of the earliest symptoms. It is said, but my own experience is to the contrary, that the temperature usually rises more abruptly than in adults. Its range tends to be higher, and it tends to greater remissions, even intermissions, on each day than it does in adults. The decubitus is often on the side. The disease more often runs a short course than in adults. The splenic enlargement is greater and more easily detectable than in adults, often protruding the abdominal wall. According to Hare, constipation specially occurs in children: this is not the author's experience. The rash is usually slight. Perforation and melæna are less common, but by no means unknown in children's typhoid, and the same is true of septic complications. Emaciation progresses faster in children than in adults.

Pneumo-typhoid is certainly commoner in children than adults, so also are joint affections as sequelæ, while laryngeal affections are rare.

The prognosis is less grave in children than in adults.

Typhoid Fever in the Aged.—It gets rapidly less common after the age of forty years, but has been seen at the age of seventy-five years. The symptoms are less well marked than in younger persons, and the intestinal lesions probably less severe, but there is a tendency to hypostatic pneumonia and to asthenia, which renders the prognosis more grave than at an earlier period of life.

Association of Typhoid with other Conditions.—*Pregnancy* confers no immunity. In the early months typhoid usually leads to abortion, but even when the typhoid occurs in the fourth month, pregnancy may go on to full term, and when occurring in the late months it usually does so and the child is born living and healthy. Freund and Levy record a case of spontaneous abortion at the fifth month, and the typhoid bacillus was found in the placenta, and in the spleen and heart of the fœtus. In a case of delivery at the eighth month, the child lived five days, and the bacilli were found in nearly all the viscera. Other similar cases are on record.

Lactation.—In a case of my own, a mother suckled her infant through the first twelve days of typhoid without ill result to the child.

Other Exanthemata.—There is no immunity conferred to other exanthema. On the contrary, a patient with any of the exanthemata appears to be in a condition particularly susceptible to contract any of the others, and typhoid may be in any of its stages associated with scarlet fever, either coming on first, or the rashes of both being present together. Diphtheria or measles may be contracted by the typhoid patient. Influenza and typhoid are commonly associated.

Surgical Operations and Injuries.—I have noticed typhoid fever in such cases too often to account for it by mere coincidence.

Diagnosis.—The most characteristic evidences of typhoid fever—the roseolous eruption, the tympanites, the recognisable enlargement of spleen, and Widal's reaction—are not available for diagnosis till after about eight or ten days of the disease have elapsed; the diagnosis at that period is seldom a matter of difficulty, the gradual establishment of a persistent continued pyrexia with the above indications being all that is required.

But the diagnosis is often important at an earlier period than this. It must then be based on the step-like ascent of pyrexia, the headache,

epistaxis, and looseness of bowels with moist sounds audible in the bronchial tubes, a dicrotic pulse, and the smell of the breath. Some of these symptoms may be wanting. The temperature chart from the commencement of the disease may not be forthcoming, and even if it is available, the pyrexia may have begun suddenly, and may, contrary to what is customary, have touched the normal on some of the early days. The bowels too may have been constipated, and epistaxis and headache may not have occurred.

Under such circumstances the diagnosis during the first eight or ten days may be impossible, but continued fever without ascertainable local cause should always raise a suspicion of typhoid fever, especially if inquiry shows other cases to have occurred recently in the locality.

The differential diagnosis from the several conditions most simulating typhoid fever must be discussed seriatim.

From Influenza.—Both diseases may begin with headache, pyrexia, and epistaxis. The suddenness of onset, the rapid ascent of temperature, the pain in the back, and the great prostration, usually suffice to distinguish influenza from typhoid fever. But typhoid may begin as abruptly as a typical influenza, and influenza on the other hand may come on as gradually as a typical typhoid attack, with gradually ascending pyrexia, headache, furred tongue, disturbed bowels, and bronchial catarrh. In such cases the diagnosis rests upon the enlargement of spleen, the dicrotic character of the pulse, the roseolous eruption, and Widal's reaction in typhoid, but it is sometimes impossible to make the differential diagnosis on these grounds without waiting eight or ten days.

The mere persistence of continued fever for many days makes typhoid more probable than influenza, but is not an absolute sign, for the pyrexia of the latter may last for weeks.

Both typhoid and influenza may begin with symptoms of meningitis or encephalitis, and the differential diagnosis rests on the Widal reaction and the appearance of the cutaneous eruption.

From Pneumonia.—The rigor, abrupt onset, great dyspnœa, rusty sputa, and the physical signs usually serve to distinguish pneumonia from typhoid, but pneumonia may come on gradually after a few days of "cold" or catarrh, and the physical signs may be late in developing. The occurrence of herpes and the ultimate development of the physical signs of pneumonia, and the Widal reaction in typhoid, will usually enable the diagnosis to be made in such cases.

A more common difficulty is to decide whether an undoubted pneumonia is the primary disease or whether it is only a part of typhoid fever; the diagnosis of such cases is often impossible until some days have elapsed, when other symptoms of typhoid fever may appear.

In children especially, "creeping pneumonia" is apt to occur, successive patches of pneumonic consolidation occurring with long, protracted pyrexia; this closely resembles a typhoid attack; the diagnosis from typhoid rests then on the course of the pyrexia, the physical signs, the absence of splenic enlargement or of a rash, and the Widal reaction.

From Tubercular Disease.—Tubercular disease may begin in various ways according to the organs which it mainly affects: it may like typhoid be evidenced for the first few days mainly by pyrexia, with scarcely any sign of visceral lesion; or it may commence with signs of meningitis, closely resembling the onset of those cases known as meningo- or cerebro-typhoid; or commencing by pulmonary symptoms, it may resemble for a time those cases of typhoid commencing by pneumonia or by severe bronchial catarrh.

Again, tubercular disease of the peritoneum may resemble typhoid fever; and data for the differential diagnosis between these various forms of tubercular disease and typhoid fever can only be derived by considering them individually.

Diagnosis between Typhoid and Acute Miliary Tuberculosis.—In both there may be much pyrexia, wasting, diarrhœa, bronchial catarrh, delirium, headache, and abdominal distention.

Typhoid Fever	*Miliary Tuberculosis*
1. Step-like ascent of temperature with subsequent continued pyrexia.	1. Rapid rise of temperature with irregular or regular intermissions or remissions.
2. Cutaneous eruption.	2. No cutaneous eruption.
3. Splenic enlargement.	3. No splenic enlargement.
4. Stools characteristic and usually diarrhœa.	4. Stools not characteristic; often no diarrhœa.
5. No optic neuritis.	5. Optic neuritis common.
6. Rigors.	6. Rigors common.
7. Prostration gradual.	7. Prostration much more than in typhoid.
8. Widal's test positive.	8. Widal's test negative.
9. No signs of cranial nerve paralysis.	9. Often signs of cranial nerve paralysis.
10. No tubercle bacilli in sputa.	10. Tubercle bacilli in sputa, if any.

Diagnosis of Cerebral form of Typhoid Fever from Tubercular Meningitis.—In both there occur pyrexia, headache, vomiting.

Typhoid Fever

1. Gradual ascent of temperature.
2. Generally drowsiness.
3. Vomiting rare.
4. Optic neuritis very rare.
5. Abdomen tumid.
6. Spleen enlarged.
7. Cutaneous eruption.
8. Cranial nerve affections very rare.
9. Widal's test positive.
10. Knee jerk persistent.
11. No signs of tubercular deposit elsewhere in the body.

Meningitis

1. Rapid rise of temperature.
2. Generally cerebral irritability.
3. Vomiting common.
4. Optic neuritis often.
5. Abdomen retracted.
6. Spleen not enlarged.
7. No cutaneous eruption.
8. Cranial nerve paralysis common.
9. Widal's test negative.
10. Knee jerks may disappear and reappear with a few days' interval.
11. Often evidences of tubercle in lymphatic glands or lungs, etc.

Diagnosis between Typhoid and Tubercle of Peritoneum.—In both conditions there occur pyrexia, diarrhœa, abdominal distention, and emaciation.

Typhoid Fever

1. Temperature rises gradually and is subsequently of continued type.
2. Splenic enlargement.
3. Cutaneous eruption.
4. Character of stools distinctive.
5. Widal's test affirmative.
6. Local tenderness in right iliac region.
7. No ascites.
8. History of exposure to contagion.
9. Previous health good.

Tubercle of Peritoneum

1. No step-like ascent of temperature, which is often remittent or intermittent.
2. No splenic enlargement.
3. No cutaneous eruption.
4. No distinctive character of stools.
5. Widal's test negative.
6. Tenderness not limited to right iliac region.
7. Often ascites.
8. Often evidences of tubercle in lymphatic glands, etc.
9. Often precedent loss of flesh.

Diagnosis between Typhoid Fever and Acute Pulmonary Tuberculosis.—In both there occur high fever, cough, dyspnœa, expectoration, and the "physical signs" of acute pulmonary catarrh.

Typhoid

1. There may be step-like ascent of temperature, but often the rise is abrupt.
2. Epistaxis common.
3. Splenic enlargement.
4. Pulse dicrotic.
5. Prostration moderate.
6. Roseolar eruption after tenth day.
7. Widal's test positive after tenth day.
8. No evidences of consolidation at apices of lungs.
9. No tubercle bacilli in sputa.
10. No evidence of tubercle in body.

Acute Pulmonary Tuberculosis

1. Abrupt rise of temperature.
2. Epistaxis absent.
3. No splenic enlargement.
4. Pulse not dicrotic.
5. Prostration severe.
6. No roseolar eruption.
7. Widal's test negative.
8. Sometimes evidences of apical consolidation.
9. Tubercle bacilli in sputa.
10. May be tubercular deposits in glands, etc.

From Typhlitis. — In typhoid fever there usually is typhlitis, and the problem to be solved is not, Is typhlitis present or not? but Is the typhlitis present a typhoid or a simple typhlitis?

In typhoid fever constitutional symptoms usually precede the local ones, and the latter are not nearly so severe as in simple typhlitis, there being rarely much pain or vomiting; in typhoid too the stools may be characteristic, and the rise of temperature is gradual and accompanied by headache. The cutaneous eruption of typhoid appears too late to be of use in the diagnosis, and too much stress must not be laid upon it if enemas have been given, as they are capable of producing a similar roseolous eruption.

From Gastro-Enteritis.—Typhoid fever begins sometimes with repeated vomiting, diarrhœa, headache, abdominal pain, and fever simulating and no doubt being associated with gastro-enteritis. The temperature chart, enlargement of spleen, and eventually the rash will clear up the diagnosis, but it is not always possible to

diagnose such aberrant cases of typhoid for some days.

From Ulcerative Colitis.—Colonic distention, diarrhœa, melæna, and pyrexia occur in both diseases. Colitis is distinguished from typhoid by the absence of skin eruption, of enlarged spleen, of step-like temperature rise, and by the character of the stools, and the pain in the course of the colon or sigmoid flexure.

From Syphilitic Fever.—Secondary or more rarely tertiary syphilis may give rise to persistent, continued (or intermittent) fever with no ascertainable local lesion. When associated with headache and enlarged spleen as it sometimes is, typhoid fever may be closely simulated. When other evidences of typhoid are long absent, and there is a history or evidence of old syphilis, this should be borne in mind.

From Pus Collections in inaccessible Situations.—Pent-up pus may give persistent pyrexia, and when it is so situated that the local signs of its existence are unobtainable typhoid fever may be simulated; especially is this likely to be the case when the abscess is within the abdomen. Thus a right-sided perinephritic abscess in a child produced chills, pyrexia, abdominal swelling, tenderness in the right flank, and was quite impossible to diagnose from typhoid fever till it produced a localised fluctuating swelling. And a duodenal ulcer, with abscess in the liver, produced tension of the abdominal muscles and pain, with chills, continued pyrexia, melæna, and wasting—a combination for a time greatly resembling the symptoms of typhoid fever. Subdiaphragmatic abscess, again, may produce *apparent* enlargement of spleen, with pyrexia and tympanites, resembling an attack of typhoid. And a loculated empyema may do the same.

From Malarial Fever.—The diagnosis is not often required in this country. As a rule the rigors and temperature range serve to distinguish between the two diseases, but cases of malaria occur in which the pyrexia may be nearly continuous, rigors absent, and diarrhœa present. This combination of continued pyrexia with enlarged spleen and diarrhœa greatly simulates typhoid, from which it can only be distinguished by examination of the blood for malarial parasite, or by the appearance of the typhoid roseola.

From Pyœmia and Ulcerative Endocarditis.—These may give pyrexia and enlargement of spleen and pink cutaneous spots. The irregularity of the pyrexia, the absence of the characteristic stools, Widal's reaction, and, in the case of ulcerative endocarditis, the production of cardiac bruits will assist in diagnosis.

Prognosis.—The fatality of typhoid fever is very variously stated by different authors, and it depends greatly on the character of the epidemic, the age of the patient, and the conditions under which he is placed. Thus in military campaigns the mortality is usually high. Again, percentage mortality varies with the inclusion or exclusion from statistics of those numerous cases of continued fever which cannot with certainty be diagnosed as typhoid. Under the cold bath treatment the mortality is 8 or 9 per cent. In the Maidstone epidemic it was only 7·5 per cent. Hare of Philadelphia estimates it as 10 per cent.

By far the largest body of statistics available are those from the returns of the hospitals under the Metropolitan Asylums Board of London, and the returns of the Registrar-General.

In the former, of 13,552 cases treated from 1871 to 1899 inclusive, 2331 died, a mortality of 17·2 per cent., and of 35,442 cases notified to the Registrar-General as occurring in London during the ten years 1891 to 1900 there were 6011 deaths, a mortality of 16·9 per cent.

This may be taken as the average mortality of typhoid fever in London, and it stands at much the same point as it was computed to stand by Murchison thirty years ago. It must be remembered that a case, however slight it may at first appear, may become serious from toxic effects, or from perforation or melæna.

Age.—The chances of recovery are generally admitted to be greater in children than in adults, but they may have the disease severely, and are not exempt from any of its dangers. Griesinger thinks the prognosis bad in very young children.

Sex.—Is rather more favourable in males than in females.

Obesity is unfavourable.

Idiosyncrasy has probably much to do with the severity of the attack, as it has in the exanthemata generally, some families and some individuals being predisposed to contract and to suffer severely from typhoid.

Season of the Year.—Dreschfield regards it as most fatal in the warm months.

Epidemic Character.—The mortality is great in certain localities and in certain epidemics. Murchison points out the resemblance cases occurring in the same house bear to one another. Surrounding insanitary conditions gravely affect the prognosis.

Previous Health.—Robustness of health confers no immunity or exemption from the chief dangers of the disease, but visceral disease, or diabetes, or privation, affects the prognosis very unfavourably.

Early Treatment.—The prognosis becomes unfavourable the longer the taking to bed is resisted. Moving the patient from the place where he has been lying ill to hospital, etc., is prejudicial in late stages of the disease, but may be less so than stopping in an insanitary dwelling.

Cutaneous Eruption.—There is no relationship between the amount of rash and the severity of the disease. Still, where profuse eruption repeatedly recurs the prognosis is less favourable than where the eruption is short-lived and slight.

Profuse sweating does not affect the prognosis.

Spleen.—There is no relationship between the degree of enlargement of the spleen and the severity of the attack.

Vomiting at the onset of the disease does not affect the prognosis. Coming on late in the disease it is unfavourable, often indicating peritonitis, perforation, or other local lesion or renal failure.

Condition of the Bowels.—Where there is deep ulceration there is generally severe diarrhœa, and though the converse is not the case, there is a general agreement that cases with severe diarrhœa are less favourable than those with constipation. Diarrhœa is especially bad when it comes on late in the disease, and at all times it tends to dry up the tongue and to drain the tissues of fluid. It also prevents the absorption of nutriment from the alimentary canal, and contributes to exhaustion. The best indication of commencing convalescence is the passing of "formed" motions.

Melœna.—Though no amount of melæna renders recovery impossible, and Trousseau and others regarded it as a favourable event, when it occurs in any quantity it is a most serious symptom, not only threatening death from loss of blood from one or repeated bleedings, but because it implies deep ulceration, and therefore often the precedent of perforation. Likewise the anæmia it produces increases the tendency to exhaustion and asthenia.

Perforation may be recovered from, as proved by Murchison's cases, where a pin-point perforation had all but healed, when erysipelas carried off the patient. But the frequency of such an occurrence must not be estimated by the many reports of cases of recovery from perforation inferred from symptoms compatible with it. Other conditions produce similar symptoms as operative procedure has several times conclusively proved. And though it is possible a pin-point perforation, with but little oozing of intestinal contents, may be recovered from, it is almost certain that any case of perforation with evidences sufficient to diagnose it will allow escape of intestinal contents, and that fatal peritonitis will ensue.

Tympanites.—Sudden supervention of marked tympanites is of bad augury, and great distention is unfavourable under any circumstances; but above all, it is so when the belly is not only distended, but is also soft and doughy, indicating complete loss of nerve tone.

Abdominal pain often indicates *deep* ulceration, especially of the colon, and is unfavourable, and often followed by malæna or perforation.

Incontinence of Fœces is not so grave as it is in other diseases. In them it is usually indicative of profound affection of the nerve centres, but in typhoid local conditions—tympanites, diarrhœa, and the liquidity of the motions—are often the main factors.

Respiratory System.—"Sipping" rapid breathing, with cyanosis and subjective dyspnœa, but without physical signs of lung affection, is of very fatal augury, being due to blood-poisoning. *Pneumonia*, with the ordinary marked physical signs, occurring at the commencement of, or at any time during the attack, is usually recovered from, but pneumonia coming on insidiously, with dulness on percussion, and crepitations, but without tubular breathing, especially on both sides of the chest, is apt to gradually invade increasing portions of lung. It is probably due to septic rather than typhoid infection, and is very fatal.

Acute general bronchitis is a very serious complication. *Pleurisy* is often recovered from if non-suppurative. *Empyema* may result from small abscesses in lung arising from septic pneumonia, and may be recovered from.

Pulse.—Rapidity of pulse is an unfavourable sign; the deaths progressively increasing as the pulse-rate rises over 120 a minute (Murchison). A pulse of 130 per minute is serious, of 140 means grave danger, and a pulse of 150 is rarely recovered from, especially in men. Death may occur when the pulse has never been over 90 a minute. Irregularity or intermittency of pulse is unfavourable, except after the pyrexia has quite subsided, and in children, in whom it is common. The pulse-rate is the most important guide we have in the prognosis of typhoid fever.

A persistence of quick pulse though the pyrexia falls foretells a continuance or exacerbation of the disease.

Anœmia.—There is often great emptiness of vessels and want of blood in the late stages, especially of long attacks, and the asthenia resulting from this is of grave import.

Pyrexia.—A temperature of 107° has been recovered from, and cases with normal or subnormal temperature throughout have been fatal; nevertheless, the *temperature range* is to some extent a gauge of the severity of the attack, and a temperature at any time of 105° or over indicates a severe attack. Of cases where the temperature rose to 105·8°, more than half died.

A persistent high temperature without some amount of remission each day is less favourable than where remission occurs, and a well-marked remission each day of 2° is a good omen; but *irregular* sudden rises and falls are of exceedingly bad omen, indicating either local complications or absorption of septic matter, or that the heat-regulating centres are so profoundly affected by toxic influences that they are thrown out of gear. A sudden fall may indicate intestinal hæmorrhage (though the actual discharge of blood per rectum may be delayed some hours), or it may indicate perforation or the crisis of an intercurrent pneumonia. A sudden rise may be produced by the supervention of lobar pneumonia or of peritonitis.

A slight fall in temperature when the rash comes out is favourable and often persists.

A high temperature of 105° is much less serious when it occurs early in the disease than late.

A temperature is often raised by retention of urine.

An early tendency to considerable morning remission is favourable.

Persistence or increase of pyrexia after the twenty-first day or thereabouts is unfavourable, and especially if the temperature rise higher than before.

A fall in temperature without a fall in the pulse-rate is mostly followed by a rapid re-rise in temperature and increased severity of symptoms.

Obstinate resistance to antipyretic measures indicates usually a severe case. Hyperpyrexia is always grave, but is nearly always due to some local complication or to septicæmia.

Rigors do not affect the gravity of a case in themselves, but when they arise from severe complications or septic absorption, the prognosis is necessarily serious.

Delirium, etc.—Liebermeister found only 3·5 per cent of cases without noteworthy brain symptoms died; 19·8 per cent of cases with mild delirium died, and 70 per cent of those with stupor or coma.

Jenner asserts that 50 per cent of those with severe delirium die. As Hare observes: "Much depends on individual tendencies and on the febrile movement, and he adds that the mental state is a fair index of the severity of the malady." But too much reliance must not be placed on this. Severe delirium is often recovered from, and *without other grave symptoms* is not of serious import. And cases may die of asthenia with the mind clear to the end.

Severe tremor was regarded by Jenner as indicative of deep intestinal ulceration; it is also producible by severe toxæmia; in either case it implies gravity of the case.

Albuminuria of more than slight amount is unfavourable, so also is glycosuria. Nephro-typhoid is said to be always serious; the one case observed by the author recovered.

Retention of urine is of little import unless neglected, when toxic symptoms may result from it which, added to those of typhoid fever generally, may be fatal in effect.

Pregnancy and the Puerperal State.—Typhoid in early pregnancy usually leads to abortion, but after the sixth month pregnancy usually continues if the typhoid is recovered from. Pregnancy adds to the danger of the patient, but the author's experience agrees with that of Murchison that it is a less serious complication than might be imagined; in the early months abortion is often recovered from, and in the late months pregnancy continues and the child is born alive. The specific bacilli have been found in the fœtus. In the puerperal state the fatality is ·50 per cent.

Hæmorrhage.—Epistaxis in the early stage is not unfavourable, but, coming on late, it or any other hæmorrhage (apart from melæna) indicates grave blood deterioration and a severe type of case.

It has been suggested that the prognosis may be based on the intensity or reverse of Widal's serum reaction, but this is not practicable or of any service.

Toxæmia is one of the chief dangers in typhoid fever. In some cases the well-known signs of the typhoid state terminate in death, but may be recovered from; in others, especially in protracted cases, the tongue does not become dry nor the temperature high, but the patient drifts into a condition of anæmia, great tremulousness, rapid pulse, clammy sweats, sunken belly, and pinched face, with blueness of extremities, the mind remaining clear throughout, or there may be rambling. Such conditions are very unfavourable in prognosis, death occurring from bloodlessness and exhaustion.

Treatment.—Prophylaxis of typhoid fever consists in measures for ensuring the purity of water supply, the perfection of systems of drainage or of other methods of disposal of excreta, and the improvement of sanitary conditions in general, details of which cannot be dealt with here. To the attention given to these points is mainly due the lessened mortality from typhoid fever in England, the returns of the Registrar-General showing a diminution from 390 persons in each million living in 1869, to 173 per million living in 1899.

The *preventative treatment by inoculation* has received extensive trial in the South African campaign, and is said to lessen the susceptibility to the disease and to lighten its severity. Reliable statistics on these points are not available.

In any attack of typhoid fever in a private dwelling (or elsewhere) inquiry should be made as to its sanitary condition, which may, if defective, be prejudicial to recovery and increase the danger of infection of other persons in the house. Defective drains become much more dangerous when a case of typhoid occurs.

The danger of contagion being imparted to others is small if those who attend upon or come in contact with the patient or his excreta be careful to secure ample ventilation, to immediately disinfect all excreta and soiled linen by carbolic acid, and to thoroughly and frequently wash their hands with disinfectants.

In treating typhoid fever it is desirable to know what are its chief dangers. Perforation of intestine causes about 18 per cent, melæna about 15 per cent of the deaths. Other local complications, pneumonia, etc., may bring the deaths from local causes to at the most 50 per cent, so that at least 50 per cent of deaths in typhoid occur from general conditions, asthenia,

hyperpyrexia, or by typhoid or other septic poisoning.

It is agreed to by all that the patient with typhoid should keep to bed from the earliest possible moment, and that there shall be no necessity to move from it, the use of the bed-pan is necessary. All excreta should be passed into vessels containing disinfectants.

The Diet must be such as will nourish the patient until the disease has run its course, must be easily digestible and assimilable, and such as that little residue shall pass from the stomach. Milk should be the staple article of food, diluted, so as the more to relieve thirst and the less to form solid clot; the diluent may be lime or barley or an aerated water. The quantity of milk should be not less for an adult than two pints in each twenty-four hours; three pints are often well taken. Typhoid patients' appetites and requirements for food vary, and as much milk may be given as the patient can take without producing curds in the stools, or diarrhœa, or distension of stomach or intestines. The milk may be given warm or cold as the patient prefers.

If diluted milk is ill borne it may be peptonised, or tried with the addition of a little tea or coffee. A few patients cannot take milk in any form, and whey, butter-milk, koumiss, or albumen of egg in water may be substituted.

Beef tea and soup, strained clear and free from fat, may be given; ten ounces in twenty-four hours is usually sufficient to supplement the milk, except in cases where the latter is not well taken.

There is no evidence that any of the innumerable artificial foods recommended as superior to milk are preferable to it, and they may be reserved for cases where milk cannot be taken, and for additions to the dietary in early convalescence. No general rule can be laid down for the dieting of those rare cases where cow's milk cannot be taken in any form; they must be treated on the principles adopted in the hand feeding of infants, sometimes substituting for a time veal-tea or beef-tea or white wine whey. Additions to the diet should not be delayed longer than is necessary, many complications being favoured by debility and feebleness of circulation. On the other hand there is good evidence that too early giving of solid food may set up relapses, or melæna, or perforation. It is possible that solid food may act injuriously by exciting increase of peristalsis, or by fermentation in the intestine lead to fresh absorption of toxins.

It is inadvisable to give solids while the tongue is coated; the converse cannot be said, for the tongue may entirely clean up early in the course of an attack, subsequently proving severe. Nor should solid food be given until the bowels, if constipated, have been well relieved by enemata or otherwise.

The appetite of the patient has been suggested as a guide for his diet, but patients often crave after, and sometimes surreptitiously eat, food which produces very evil results.

The temperature chart is generally taken as a guide to the commencement of solid food; some withhold it until seven or even ten days of complete apyrexia have expired; others give solid food without reference to a persistence of pyrexia, often with complete impunity. No doubt in those cases of typhoid in which intestinal ulceration never occurs or is at a minimum, solid food may be given without danger, and even with advantage at a very much earlier period than in other cases in which there has been severe intestinal ulceration. It is not, however, always possible to determine with certainty if ulceration has been slight in an individual case, but where there has been no great tremor, nor melæna, nor much tympanites, nor diarrhœa, and where the motions become "formed" early, ulceration of intestines has probably been slight, and solid food may be tried early. In cases where there is reason to believe that there has been deep ulceration there is a danger, quite out of proportion to any advantage obtainable, in giving solid food before pyrexia has subsided for at least three complete successive days.

Bread and milk, fish, minced chicken, vegetables, may be gradually added to the diet, and persisted in even if a slight rise of temperature occurs, so long as there is no diarrhœa or tympanites set up, or acceleration of pulse.

Great Thirst may be appeased by drinking cold water or toast water in moderate quantities; large hot-water enemata are also very useful. Saline enemata increase the thirst.

Alcohol is not required in cases of typhoid which progress well, but patients exhausted by combating the disease without taking to bed often require it as soon as they come under treatment. During convalescence a little alcohol is often useful. The general rules which are guides to administration of alcohol in other diseases are applicable in typhoid fever, a dry brown tongue, muttering delirium, failing pulse, and weak heart sounds indicating its administration, but it should not be given more freely than is absolutely required.

Diffusible Stimulants.—Ether, ammonia, sumbul, and black coffee are often useful, and *strychnine* hypodermically administered is of great use when the heart is acting feebly or the nervous system much depressed. Caffein is also useful, especially combined with strychnine.

The Antiseptic Treatment is based on the endeavour to lessen the virulence of the toxins in the intestine before absorption, and to limit the action of the micro-organisms in the alimentary canal. It has been claimed also that antiseptics may *abort* the disease, but this is not substantiated. The antiseptics which have been advocated are innumerable. Charcoal was used by

Sir W. Jenner, but is bulky to take; salol is one of the best intestinal antiseptics. B. naphthol is equally useful. The internal use of mercury in typhoid fever was condemned by Murchison, but has been recommended by Broadbent, and the author's experience agrees with his, that it often lessens tympanites, lowers the pyrexia, and appears to lessen the toxæmic symptoms. It may be given as bichloride grain $\frac{1}{16}$ ter in die or as calomel in $\frac{1}{4}$-grain doses. If there is constipation, the calomel is often useful as a laxative as well as an antiseptic. The bichloride is best abstained from when there is melæna. Burney Yeo advocates the internal administration of chlorine water. The carbolic acid treatment readily produces carboluria.

During convalescence the patient should be given as strengthening a diet as he can take, and some alcohol is useful as well as dilute acids and bitters. He should sit up in bed as soon as he can do so without becoming tired, and get up when strength allows. It must always be remembered relapses may occur, and, when they commence, liquid food should be at once returned to. As early as possible the patient should be got into fresh air.

Antitoxin Treatment.—An anti-typhoid serum has been used, but is not of any proved efficiency.

Diarrhœa.—Measures should always be taken to check diarrhœa if the motions are more than two (some say more than four or even eight) in twenty-four hours. Diarrhœa is often due to milk being given too freely or in an unsuitable manner, and lessening the quantity or giving smaller quantities more frequently, or changing the diluent will terminate the diarrhœa; sometimes beef-tea, etc., has to take the place of milk for a time. If changes in diet are not effective, nothing is so useful as an enema of starch with five to ten minims of laudanum. The drugs ordinarily used in diarrhœa are of service in typhoid; salicylate of bismuth in 20-grain doses, with or without a small quantity of Dover's powder, is very efficacious. Intestinal antiseptics sometimes relieve diarrhœa.

Constipation.—Some consider a purge, or a series of purges of calomel to be useful at the commencement of the disease, and a purge is often given before the diagnosis has been made, and appears to do no harm, but there is no reliable evidence that they do any good. Later on purgatives may do serious harm, and constipation must be dealt with by simple enemata, and it is well to add to them some disinfectant, such as sanitas. During convalescence, belladonna and aloes in pill is a useful laxative.

Peritonitis, from any cause except perforation, is best treated by opium, and possibly, even when not due to perforation, laparotomy would offer the best chance of recovery.

Vomiting may be due to uræmia or to some local abdominal complication, peritonitis, etc. When not due to these it may be treated as is usual in other conditions.

Tympanites may be often lessened by attention to the quantity or character of the diet; apart from this, hot fomentations with turpentine or belladonna are the best means of lessening it. Oil of turpentine enemata are recommended by Murchison, or 10-minim doses of oil turpentine by the mouth. Some adopt the ice poultice on the abdomen. The insertion of a hollow tube into the rectum, with attached drainage tube passing into a bucket of water, often allows the escape of flatus, but is apt to lead to urinary retention.

Pain in Abdomen is best treated by fomentations with opium.

Perforation is so rarely spontaneously recovered from that operation affords the best chance of recovery; the difficulty rests in deciding if perforation has occurred; in many cases operation has been performed for perforation when the symptoms resulted from some other cause, and in many other cases perforation occurs with scarcely any symptoms to indicate it.

Melæna.—Opium should be given (in pills, if such can be swallowed), and pushed to the point of contracting the pupils; tincture of hamamelis in 5-minim doses every hour is of great service. Oil of turpentine (a drachm daily in 10-minim doses).

Ergot, gallic acid, and even ergotine seem of little use. The patient must be kept absolutely quiet to lessen the probability of recurrence of bleeding, and the food for the time should be diminished in quantity, but water may be given to allay thirst. The ice bag or ice poultice to the abdomen has some restraining effect in melæna, but must be removed after twenty-four hours, or the tissues become frozen and their vitality impaired. Opium enemata are recommended. Small quantities of alcohol may be required, but not sufficient to excite the heart's action.

Often the pulse becomes rapid and full, threatening recurrence of melæna; this is best met by opium.

Murchison strongly recommends the internal administration of spiritus terebinthinæ ♏xv, tinct. opii ♏x, and tannic gr. x, with mucilage every two hours.

Ether subcutaneously may be required after profuse melæna, and if collapse be great, saline intravenous or subcutaneous injections.

Sleeplessness is due often to headache, and ceases when that is relieved. Trional and paraldehyde are good hypnotics; sulphonal takes some hours to act and may produce a rash. Morphia may be given if thought necessary, but once begun will be asked for through the whole course of the disease.

Headache may be best relieved by 3 grains of acetanilide, repeated from time to time.

Delirium requires careful watching of the

patient, for he may suddenly get up from bed and catch a chill, or determine hæmorrhage or perforation, or may attempt suicide or injury to others. Low muttering delirium requires alcohol; the liability to it appears lessened by the administration of intestinal antiseptics, commenced early in the case. Restraint of the patient is always to be avoided, if possible, except by having sufficient nurses. When delirium goes on long and there is the condition of incessant wakefulness, a hypodermic injection of morphia is required; trional sometimes acts well. Oxygen inhalations are often useful in toxic delirium, but must not be insisted on if the apparatus excite alarm.

Pyrexia is not now regarded as one of the chief dangers of typhoid fever, and too much importance is not to be attached to the mere temperature; a pyrexia of 103° or even 104° for two or three weeks in a healthy young adult lying in bed and taking food well will not itself produce serious consequences, and when an unusually high temperature occurs, the danger is usually due to the local complication which gives rise to it and not to the pyrexia. And on the other hand serious, even fatal, cases may occur without much or even any pyrexia. The temperature, if judged unduly high for the period of the disease, can usually be temporarily lowered by quinine or by drugs such as phenacetin, antipyrine, acetanilide, etc., but it is not advisable to use any of the latter for this purpose, as they have destructive influences on blood constituents and are depressing. Quinine in 5-grain doses is tonic in effect and is very effective, but it is slow in action. Much the most effective method of reducing pyrexia is the application of water at a low temperature. The simplest plan, and one which is sufficient in most cases, is tepid sponging. But the cold pack or cold bath has a more powerful effect in reducing the temperature. The cold-bath treatment consists in repeated immersions of the patient from the beginning of the disease in water at a temperature of from 65° to 70° or 80° F. Cases of typhoid vary very much in severity in different epidemics, in different countries, and at different ages of life; moreover, at the early stage of the fever at which the cold-bath treatment is commenced by some, the diagnosis is so uncertain that some of the cases said to be aborted by the bath treatment may not have been typhoid at all. Still, the statistics show the mortality under this method of treatment to be only 8 or 9 per cent, very much less than the mortality in England of about 17 per cent. The mortality in the Maidstone epidemic was only 8 per cent without cold baths.

In the routine treatment all patients must commence it from the earliest period of the disease. Out of every 100 cases of typhoid, 92 would have recovered without the treatment, and 8 who would have died under any treatment are subjected to it unnecessarily. And it is not that doubts are felt as to the benefits of the treatment, but because of the difficulties in private practice of carrying it out, that the cold-bath treatment is not more generally adopted.

It is impossible, it must be conceded, to foretell with certainty whether a given case of typhoid will turn out to be mild throughout its course; still in many cases there are indications that it will be so, and, as the cold-bath treatment stands a much better chance of being made use of if it be observed in what class of cases it is most requisite, we reiterate insistence on its necessity in every case.

Osler and Liebermeister only give the baths if the temperature rise over 102·5°, Singensen at over 104°.

Hare of Brisbane finds that the temperature of the bath need not be so low as sometimes recommended; he obtained equally good results with water at 75° or 80° F.

Dr. Barr recommends that the patient be kept in a tank of water, continually changing, for many days, the temperature of the water to be 90° to 98°.

Hyperpyrexia is mostly due to some local complication, and if discovered the treatment of it will be indicated; as a symptom nothing is so effective in its treatment as cold bathing, and quinine may be given also.

Asthenia and Toxæmia are commonly combined. The treatment consists in endeavouring to nourish the patient from the very beginning of the illness, giving as much food as he can assimilate, in preventing losses by hæmorrhages and diarrhœa, and by promoting sleep. In a more advanced stage of the disease, with signs of exhaustion and toxæmia, every endeavour must be made to promote the action of the eliminating organs and to stimulate the circulation by strychnine, alcohol, sumbul, musk, and other diffusible stimulants, etc. It is in the reduction of the deaths from asthenia that the routine cold-bath treatment lessens the mortality, and not from lessening the frequency of local "accidents." Many of the deaths appear to be contributed to by an actual want of blood, the vessels being empty, and a tendency arising for blood to accumulate in the *venous* circulation.

In these cases intravenous saline injections are sometimes useful, too often only temporarily. I have found they act better at a temperature of 100° than at the higher temperature at which they are sometimes given.

Iron and feeding to the utmost possible are advisable at this period, and I have given bone medulla internally with apparent advantage.

Typhomalaria. *See* MALARIA (*Complications*); TROPICS, UNCLASSED FEVERS OF (*Typho-Malarial Fever*); UNDULANT FEVER (*Synonyms*).

Typhomania. *See* DELIRIOUS MANIA (*Synonyms*).

Typhopneumonia.—Pneumonia occurring as a complication of typhoid fever. *See* TYPHOID FEVER (*Complications and Sequelæ*).

Typhotoxin. — A toxine ($C_7H_{17}NO_2$) obtained (by Brieger) from cultures of the typhoid bacillus; it causes death with lethargy and paralysis.

Typhus Fever.

DEFINITION 203
HISTORY 203
ETIOLOGY 203
DISSEMINATION 204
Incubation Period 204
Invasion Period 204
Period of Advance 204
Stage of Defervescence . . . 206
Convalescence 206
COMPLICATIONS AND SEQUELÆ . . . 207
VARIETIES 207
DIAGNOSIS 207
PROGNOSIS AND MORTALITY . . . 208
TREATMENT 209

See also BREATH (*Offensive Odour*); BRONCHI, BRONCHITIS (*Etiology*); EPIDEMIOLOGY (*History, Extinction of Typhus*); HYDROPATHY (*Cooling Pack in Typhus*); INFECTION (*Quarantine*); LUNGS, GANGRENE OF (*Etiology*); MILIARIA (*Symptoms*); PUERPERIUM, PATHOLOGY, SEPTICÆMIA (*Diagnosis*); PURPURA (*Symptomatic*); RELAPSING FEVER (*Diagnosis*).

SYN.—*French*, Typhus exanthématique; *German*, Fleckfieber.

DEFINITION.—An acute specific infectious fever, prevailing in epidemics, and usually associated with conditions of overcrowding and destitution. Characterised by a sudden onset, a petechial rash, marked nervous symptoms, with great mental confusion and physical prostration, and by a rapid defervescence on or about the fourteenth day.

HISTORY.—Typhus fever is a disease of great antiquity. Murchison has identified with it the pestilence at Athens, so graphically described by Thucydides. From that time onward we have abundant evidence of its frequent epidemic prevalence, and of the severe mortality caused by it. In the sixteenth century the descriptions of the fever stand out more clearly; Fracastorius, for instance, in 1528, describing it wonderfully accurately under the title of "Febris pestilens." Under this name it was common during the seventeenth century, and was particularly fatal during the Thirty Years' War, the camps of those days lending themselves especially to its propagation. It was apparently common in Great Britain,—the "black assizes," where judges, jurymen, and spectators all sickened, being undoubtedly due to its contagion. Later, the armies during the Napoleonic wars suffered severely from its ravages, and, so recently as in the Crimean campaign, French and English alike lost heavily by typhus.

During the nineteenth century there were several great epidemics in Great Britain and Ireland. They mainly appeared at periods when destitution and famine had lowered the resistance of the population, and they were not infrequently preceded by epidemics of relapsing fever. The disease in many instances appears to have been imported from Ireland to England. Scotland usually suffered markedly, and the fever seems to have persisted there longer than in England. In fact, at the present day, though cases occasionally occur in London, and there has been an outbreak recently in Liverpool, typhus is seldom seen in England, whereas Glasgow usually can show some dozens of cases every year, and the other Scottish large towns have not infrequently small outbreaks. Modern sanitation has taken away all chance of the fever again assuming epidemic proportions in Great Britain, and its interest is now very largely historical. There is no fever, however, which presents a more perfect picture of acute toxæmia; and it must be remembered that the "typhoid state," that poisoned condition so dreaded in acute disease, derives its name from its resemblance to the normal symptoms of typhus.

ETIOLOGY.—Murchison believed that under certain conditions this fever could originate *de novo*. Such a view is hardly tenable at the present day, and it is almost certain that typhus is caused by a micro-organism, which possibly may require the existence of certain favourable circumstances for its development. Unfortunately up to the present time, although different germs have been discovered by various observers, no proof is forthcoming that any one of them is the true cause of the fever. A diplococcus has been described by Dubief and Bruhl, and another by Balfour and Porter, but the organisms apparently differ in their cultural peculiarities, and inoculation experiments on animals have given negative results.

The most important of the predisposing causes of typhus is undoubtedly *overcrowding*. Whenever large numbers of persons live without sufficient air-space, whether in prisons, in ships, in camps, or in the slums of a city, the disease is likely to appear. The names given to it in past times—gaol fever, camp fever, and ship fever—give an idea of the conditions in which it is likely to arise. *Destitution* and *famine* are also given as predisposing causes. The first compels large numbers to crowd in one-roomed dwelling-houses, the second predisposes to the attack of any infection by lowering the resistance of persons exposed, and to this

extent their action may be admitted. But typhus often appears in times of comparative prosperity, and overcrowding by itself seems a quite sufficient cause. Again, if half-starved persons lived under favourable sanitary conditions there is no reason to suppose they would contract the disease. As regards age, it has been said that adults are most apt to take the fever, but this is merely because typhus in children is usually mild, and frequently unrecognised; sex exercises no influence. As to climate, the disease is most common in temperate regions. Season has considerable influence, the fever being most common in winter and early spring, the cold causing windows to be shut and thus diminishing the ventilation. Mental and physical fatigue and ill-health may also be regarded as secondary predisposing causes.

Dissemination.—Typhus fever is directly contagious from person to person, and in the vast majority of cases the disease is spread in that manner. Anyone who is in close contact with the sick, a nurse especially, is liable to take it. The poison is apparently carried by the exhalations from the patient, and is no doubt inhaled by the victim. If sufficient attention, however, is paid to the ventilation of the room the risk is reduced to a minimum. Indeed, the germ of typhus would seem to be particularly susceptible to fresh air. In any case the "striking distance" of the disease is an exceedingly short one, the fever in this respect differing markedly from such an infection as small-pox. Absolute contact, or at least entering a small room with no ventilation in which the patient is lying, seems to be necessary for infection. In the last outbreak in Edinburgh it was found that even in crowded and insanitary tenements, only those persons who were on intimate terms with the sufferers, and who were in the habit of entering their apartments, contracted the disease. Others, who were not on familiar terms with them, escaped even though the doors of their rooms opened on the same stuffy and unventilated landing. It has been said that prolonged exposure to the poison, even in well-ventilated wards, increases the risk of infection. This may well be the case, but is more probably due to increasing fatigue and failure of health on the part of the attendants exposed rather than, as has been suggested, to a gradual concentration of the poison in their systems.

That infection may be retained for long in fomites there is no possible doubt, and those who have to handle the clothes and bedding of the sick, such as laundry-women and public-health officials, often contract the fever. Infection seems also to persist long in old houses that have not been thoroughly disinfected or properly aired.

Incubation Period.—As a rule this would appear to be about twelve days. In two cases of the writer's, in which there was only one possible exposure, it was exactly thirteen. But shorter incubations are undoubtedly possible. Murchison regards the limits as from a few hours to twenty-one days. The longest incubation in the writer's experience was one of seventeen days at least.

Invasion Period.—This may be said to last from the first symptom of the patient until the temperature reaches the acme, which is usually on the third or fourth day of the fever, and until the appearance of the rash. The onset of typhus is usually abrupt, the patient being nearly always able to mention definitely the day, and sometimes even the hour at which he first felt ill. The most prominent symptoms are a very marked and persistent feeling of chilliness, causing the sufferer to sit wrapped up in front of the fire, severe headache, pains in the back and limbs, and vomiting. Occasionally there may be actual rigors, and in young children convulsions often occur. The temperature rises, in some cases rapidly, but as a rule slowly and steadily, till by the fourth day of the disease it has attained the acme, usually a point between 102° and 104°. The appetite fails, and the tongue is at first covered with a thin, close-set white fur, which gradually dries and turns yellowish or brown as the disease progresses. Insomnia is common, and even at this early stage the clouding of the intellect, which is such a marked feature of the fever, is frequently present. The patient can seldom keep on his feet beyond the third day of the disease, and takes early to his bed.

The Period of Advance.—On the fourth or fifth day of the fever the characteristic *rash* makes its appearance. It consists of small pink spots, a little smaller if anything than the spots of enteric fever. At first they are distinctly raised above the skin, but this is only in the first few hours of their existence. At first, like the enteric spots, they fade on pressure, but afterwards become stained into the skin and persist even if pressed. As the disease progresses they become darker and brownish in colour, and they do not as a rule show much sign of fading before the eleventh day. About that time the rash loses, in many cases, its distinctness; and sometimes all traces of it are gone by the time the crisis is finished. Occasionally a mottled yellowish staining persists. In addition to the spots on the skin there is often, indeed usually, an appearance also of similar spots seen dimly as it were through the skin, the so-called *subcuticular mottling*, seen as a rule best on the flanks. Occasionally this is the only appearance of eruption shown by the patient, and it is quite diagnostic. Lastly, in severe cases true hæmorrhages occur into the skin, appearing as purpuric spots usually small in size, and resembling the marks left by fleabites, except that they lack the central point.

This feature of the eruption is responsible for one of the synonyms of the disease, petechial fever. The dark-stained, purple to brown, colour of the rash has caused it often to be spoken of as the "mulberry rash of typhus." As a whole, it is fairly distributed over the body except the face. In cases where it is scanty it is best seen on the trunk, and it may be most marked on the back.

The *appearance* of the patient is worthy of attention. The face becomes bloated, the features seem to lose their distinctness, and the expression is that of a drunken man. The eyes are remarkable in that the conjunctivæ are deeply injected and the pupils small, and in severe cases exceedingly contracted. As regards the alimentary system the *tongue* is dry and brown. As the fever goes on it is apt to show transverse fissures and crusts of dried blood and sordes, which may also be seen on the lips and teeth. In severe cases the tongue seems to dry up into a shrivelled black ball, exactly resembling the tongue of a parrot. Vomiting at this stage is not common, but the digestive power is very poor. Constipation is the rule, but occasionally diarrhœa may be severe enough to assume the importance of a complication. The *temperature* remains at a high level, readings of 104° being quite common, and there is little or no morning remission, the chart at first in this stage showing an almost straight line. In cases, however, which are probably running to a favourable termination, there is often a gradual decline of the temperature, sometimes almost imperceptible, in the second week. About the eleventh day there is occasionally a fall of one or two degrees, or even more—a suggestion, as it were, of a false crisis. Hyperpyrexia is uncommon, except in fatal cases, when it occasionally immediately precedes death. The *pulse* is accelerated, as a rule, in proportion to the temperature. It usually ranges from 100 to 120, the latter figure being, of course, much exceeded in very bad cases. It has no special characteristic, and has not the marked dicrotism so common in enteric, but is apt to fail towards the end of the second week. The first sound of the heart as heard at the base soon gets faint, and in bad cases disappears altogether. The *respiration* is invariably rapid, most adult cases breathing 30 to the minute. In the second week of the fever, when there is nearly always more or less hypostatic congestion at the bases, respirations of 40 are quite common. The *urine* nearly always contains albumin, and has the usual characteristics of fever urine, being scanty and high-coloured. During the first week the amount of urea is much increased. The so-called diazo reaction of Ehrlich is given by practically every case of typhus at the height of the disease. The *spleen* is invariably much enlarged, often indeed palpable, and this condition is usually appreciable at a much earlier period than is the case in enteric fever.

But it is the *nervous symptoms* of typhus which are the most important and characteristic. The whole nervous system seems thoroughly poisoned. The clouded condition of the mental faculties has given the fever its name. Even the mildest cases suffer from deafness and mental confusion. *Delirium* is almost invariable and is of two kinds—the so-called "delirium ferox," which comes on about the seventh or eighth day, when the patient may require several strong men to control him, and has to be strapped to his bed, and more commonly the low, muttering delirium which few patients escape. *Insomnia* sufficiently marked to require treatment occurs in about half the number of adult patients. If the patient is allowed to be sleepless for more than one night the condition may become intractable and may turn the scale against recovery. *Coma* often precedes death, and is usually of the waking variety, the "coma vigil," which must always be regarded as of fatal prognosis. The grave condition of the nervous system is further shown by subsultus of the tendons, and in severe cases by carphology or picking at the bedclothes. Occasionally there is twitching also of the corner of the mouth or of the whole face. The *organic reflexes* are also interfered with. There may be incontinence of both urine and fæces, and frequently the incontinence of urine may be really due to retention, the overflow of a distended bladder escaping into the bed. Even with the most careful nursing bedsores sometimes occur, the trophic functions of the nervous system suffering with the rest. The headache which was so trying to the patient in the first few days of his illness does not usually trouble him much in the period of advance. It, as a rule, disappears when the delirium begins.

By about the twelfth day of his fever the patient may be regarded as entering the critical stage. He is at this period usually at the very worst. The acute toxæmia and the prolonged and severe pyrexia have thoroughly prostrated him. He lies absolutely helpless, passing his evacuations under him, and usually totally unconscious of his surroundings. The question now is will he recover or not? Fortunately, if he has been judiciously treated and carefully nursed, even when he is looking his very worst, he has still a very fair chance of recovery. But, on the other hand, should he have been over or under stimulated, should he have not been given sufficient sleep, above all, if he has been alcoholic, his chances are very poor. If death supervenes, as in such cases too often occurs, the temperature at this stage of his illness shows signs of rising rather than falling, he may suffer from cold, clammy perspirations, or he may fall into coma before the end comes. In other cases the enfeebled heart cannot stand, as it were, the shock of the crisis, and as the temperature falls the pulse-rate increases, and

death occurs with the temperature actually on the normal line.

Stage of Defervescence.—If, on the other hand, as is more often the case, the patient is to progress towards a favourable recovery, the temperature often shows a tendency to fall a little, if only half a degree to a degree, about the twelfth day. The *crisis* itself which follows this slight movement of the temperature is usually a comparatively slow one, but is nevertheless exceedingly well marked. It may commence on the thirteenth, fourteenth, or fifteenth days, or even later, but most commonly a considerable fall is noted on the evening of the thirteenth or the morning of the fourteenth day. The fall may last two or three days, but usually the evening of the fifteenth day sees the temperature normal. Thereafter it tends to remain subnormal for about a week. This gradual but steady abating of the pyrexia is accompanied by a proportionate decrease in the pulse-rate and by a general clearing of the mental confusion. The tongue becomes rapidly moist, the conjunctivæ clear up and lose their pink tinge, and the pupils return to their normal size. The skin is moderately moist, but sweating is unusual and is not a good sign. The urine is much increased in amount and much paler in colour, and the albumin disappears. A slight looseness of the bowels often precedes or accompanies this critical change. Occasionally hypostatic congestion of the lungs may be extensive or severe enough to keep the temperature slightly elevated for some days after the crisis has occurred.

Stage of Convalescence.—Except in a few cases the patient is practically out of danger after his temperature has reached normal. Some patients, however, take a little time to rally after the profound alteration which has occurred in their general condition. The vast majority, fortunately, give no trouble at all, and it is remarkable to see a man, who three days before may have looked absolutely hopeless, eating a liberal allowance of solid food with every sign of enjoyment. Moreover, in spite of considerable wasting having occurred during the fever, the short duration of the disease makes this wasting much less marked than it is in enteric, and convalescence is therefore short. The average patient is well able to be out of bed within a week from his crisis, but it must be, of course, remembered that he is infectious to others for probably a fortnight later. Rare instances of true relapse of typhus have been recorded by Curschmann.

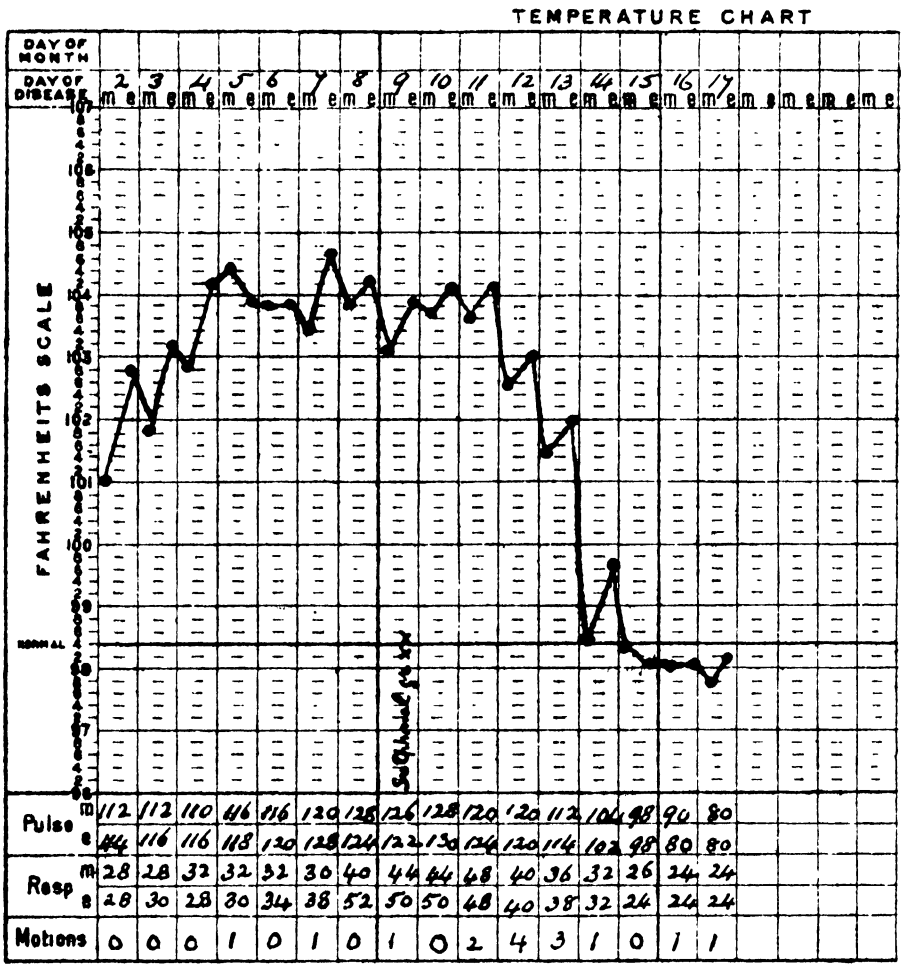


Chart 1.—Chart of a young woman of 23, suffering from severe typhus from second day; recovery. Showing rise of temperature to acme, improvement from 12th day, and final crisis on 14th.

Morbid Anatomy.—A certain number of cases will at a post-mortem examination show traces of the characteristic eruption. The stained spots, of course, are visible after death, and if there has been much true hæmorrhage into the skin the purpuric patches are also visible. Rigor mortis is slight. On opening the body there is nothing to be found which is pathognomonic of the disease. The internal organs are, as a rule, very much congested, and small hæmorrhages may appear on their surfaces, as also occasionally on the lining membrane of the trachea and bronchi. Meningeal hæmorrhages and hyperæmia of the surface of the brain are relatively common. The spleen is much enlarged and usually diffluent. The heart is often dilated, its muscle is flabby and friable, and the

blood is very dark and more fluid than normal. Curschmann has noted perichondritis of the arytenoid cartilages with suppuration as occurring comparatively frequently. There is always more or less congestion at the bases of the lungs.

Complications and Sequelæ. — Speaking broadly, typhus is a disease remarkably free from complications. As has been suggested above, the respiratory organs may be occasionally so severely affected that their condition assumes the importance of a complication. Thus the almost constant bronchial catarrh may be very much exaggerated, the laryngitis, not a very infrequent symptom, may be accompanied by severe and fatal ulceration, and occasionally a true croupous pneumonia may appear during or shortly after the fever. Gangrene of the lung has been also reported. Nephritis is occasionally met with both during the disease and in convalescence, and must be regarded as a serious complication, although by no means always fatal. Venous thrombosis or "white leg" has been frequently noted, as is the case in enteric fever, and also the more dangerous condition of arterial embolism. Internal hæmorrhages and passing of blood by the bowel are liable to occur in the more severe hæmorrhagic forms of the fever.

Of the other complications most frequently met with diarrhœa is perhaps the most important. Especially in alcoholic cases this condition, starting at the beginning of the second week, may cause great danger to the patient. Various inflammations are liable to occur in convalescence, the most common being enlargement and suppuration of the parotid gland, suppurative otitis media, and superficial skin abscesses.

Varieties of Typhus.—Numerous variations in the severity and course of the fever have been described. It will be sufficient here to briefly indicate the most important. Certain cases of great severity, the patient being early overwhelmed by the virulence of the poison, have been called "typhus siderans." Other types, where the nervous symptoms or the physical prostration have presented the main feature of the disease, are named "ataxic" and "adynamic" respectively. Cases in which hæmorrhages have occurred into the skin, and from the mucous membranes, are classed as "hæmorrhagic" or "scorbutic." There is also undoubtedly a type which may be termed "abortive," the fever lasting only a week or less. In children, it may be added, the disease usually runs a mild and brief course, and the eruption may be of a fleeting character or altogether absent.

Diagnosis.—In the presence of an epidemic diagnosis is not, as a rule, very difficult. The typically sudden onset of the disease, the early prostration, and the ultimate appearance of the characteristic eruption on or before the fifth day, should not present any difficulty. But it is well to remember that sporadic cases of typhus may occur, and it is very easy to confuse them with other conditions. Again, in children the rash may be very slight and scanty, and the general symptoms of a mild character, and it is sometimes very difficult to come to a satisfactory conclusion. In doing so the general appearance of the patient is of great assistance. Even the mildest cases have usually a flushed and somewhat bloated face, and the conjunctivæ may be said to be always suffused. If a patient with

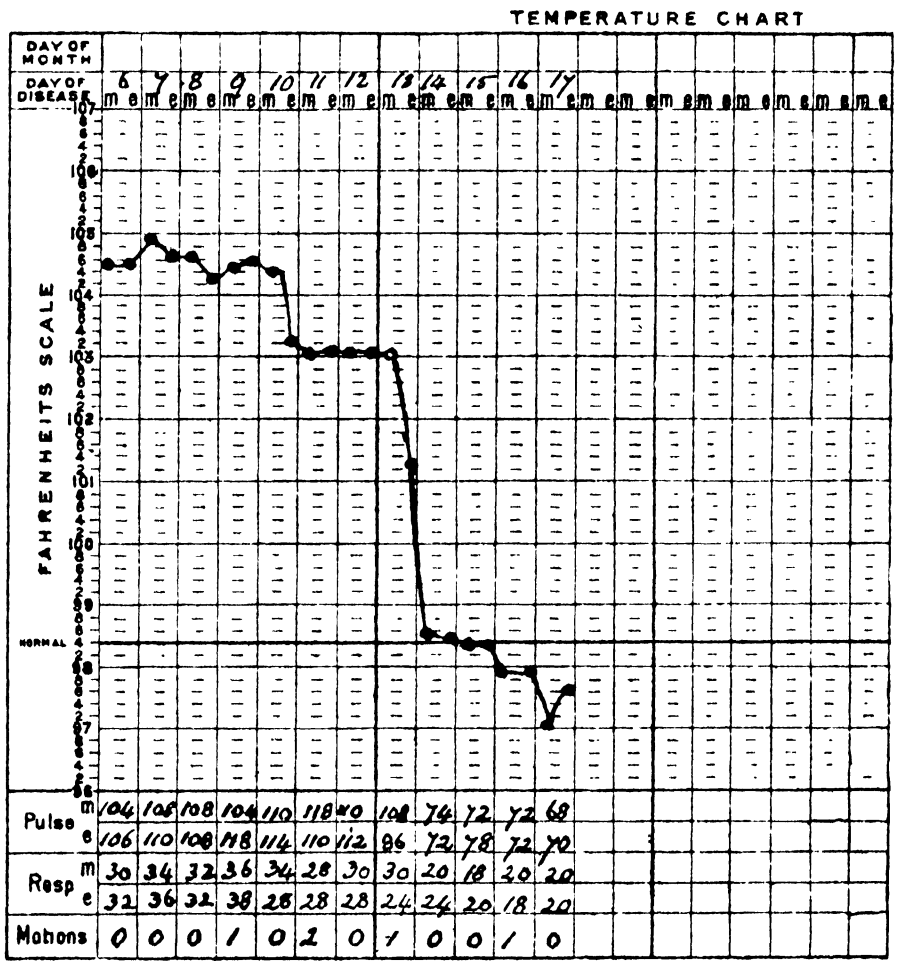


Chart 2.—Man of 26. A mild case of typhus from the sixth day. Showing temperature with slight remission, improvement from 11th day, and a well-marked crisis.

such an aspect has suffered from a sudden attack of chilliness accompanied by headache and vomiting, the presumption in favour of typhus is strong.

Those who have had experience of the fever often find assistance from the characteristic *odour* of the patient. This has been compared to that of rotten straw or of mice, but it really is indescribable. It is usually appreciated only on first lifting or disturbing the bedclothes to look at the rash, and appears to be quite independent of the dirty surroundings in which the typhus patient is usually found, for it has been noted in cases after they have been weeks in hospital. Its existence as a definite sign is, however, it is only fair to add, doubted by no less an authority than Curschmann.

It will be well to consider a few of the chief conditions liable to be mistaken for typhus. In its early stage it closely resembles small-pox in its onset, but this difficulty is so quickly cleared up that it is hardly worth noting. Much more important is its differentiation from the following diseases:—

Typhoid.—This distinction is not likely to cause much difficulty in a patient watched from the first day of his illness. But it is well to remember that all cases of typhoid do not start insidiously, and that in certain instances the onset may be quite abrupt. The difficulty is usually to distinguish a typhus case with a history of some ten days' illness from an enteric case in the third week. The table below gives the chief distinctions:—

	Typhus Fever.	Enteric Fever.
Face . . .	Congested and bloated.	Pale, with hectic flush.
Expression . .	Drunken.	Languid and dreamy.
Pupils . . .	Contracted.	Dilated, or in bad cases normal.
Conjunctivæ . .	Injected.	Clear.
Abdomen . .	Usually normal and not tender.	Tumid and tender.
Rash . . .	Fairly profuse. Not fading on pressure. Petechial.	Scattered rose-spots on trunk, disappearing on pressure.
Pulse . . .	Rapid.	Usually infrequent in comparison to the temperature, and dicrotic.
Termination . .	Crisis at about end of second week.	Lysis.

It must, however, be always borne in mind that a bad case of enteric, with delirium and a profuse eruption, may defy the skill of the most careful physician. The most reliable method of diagnosis is undoubtedly Widal's reaction, which, if done properly, is absolutely to be trusted. But here, again, the class of persons who contract typhus have often previously had enteric fever, and a reaction may be occasionally given, which is liable to confuse the diagnosis more than ever. Ehrlich's reaction is useless, as all cases of typhus give it.

Pneumonia.—The marked respiratory symptoms of typhus, the flushed face, and the sudden onset have often caused difficulty in distinguishing the two diseases. Moreover, cases of apical pneumonia with "typhoid" symptoms may closely simulate typhus, and the physical signs be not easy to make sure of. In cases where there is doubt in the early stage, before the rash has appeared, it is always well to examine the lungs, and especially the apics, most carefully; and in later stages, where the rash may be presumed to have faded, it is well to ascertain if the dulness at the bases of the lungs is limited to the lower lobes only. A true pneumonia usually affects the whole lobe. A hypostatic pneumonia, complicating typhus, usually affects both sides equally, and may not reach, or may overstep the limits of the lobe. Again, in the latter case, though there may be abundance of fine crepitations, it is unusual to find tubular breathing.

Meningitis.—Here again the absence of the rash is the chief distinction. The typhus patient is hardly so irritable as the meningitic one. If a child, he is not likely to utter the peculiar cerebral cry. He may, however, suffer from ptosis, strabismus, inequality of the pupils, and so on, although these symptoms are, of course, much more common in meningitis. In typhus the headache ceases when the delirium begins, whereas in the severer condition the headache usually persists. The "*tache cérébrale*" may be of some assistance in these cases.

Of the other conditions which may cause confusion we may instance, first, *measles*, the rash of which, when fading, may present a fair imitation of that of typhus. In measles, however, there is always a trace, or a history of the existence of the rash on the face, and usually a history of catarrh of eyes and nose. In typhus the rash never invades the face, and the eyes and nose do not suffer. *Purpura* is usually unaccompanied by fever, and its relatively large hæmorrhages are not seen in conjunction with a subjacent mottled rash. *Uræmia* also is seldom febrile; still, it is well to remember it has been occasionally confused with typhus. *Influenza* in its early stage has many features in common with typhus fever.

PROGNOSIS AND MORTALITY.—Questions of age and sex are of great importance in giving a prognosis. As regards the former, it may be broadly stated that the danger steadily increases as age advances. It has been stated by some

observers that the first five years of life show a relatively higher mortality than the subsequent ten, but that is not in accordance with my own experience. The death-rate in persons of under twenty years is almost a negligible quantity, seldom exceeding 3 or 4 per cent. After twenty years have been passed, however, the rate steadily increases, being at least 15 or 20 per cent between the ages of twenty and forty, and nearly 50 per cent when sixty years are passed. As regards sex men are admitted to have a much higher mortality than women. This is no doubt in part due to the greater likelihood that they are alcoholic; but persons of spare constitution, even if alcoholic, of both sexes have a much greater chance of recovery than powerful, muscular, and heavy subjects. The question of body-weight is, in my opinion, of considerable importance. A superabundance either of muscle or fat seems distinctly to add to the dangers of the disease.

Every one admits that alcoholism lessens the chance of recovery enormously. The class of persons who usually contract typhus is exceedingly liable to contain a high proportion of alcoholics, and this undoubtedly accounts for the high mortality common in the fever. Such cases suffer from an exaggeration of all the nervous symptoms that have been described above, and it may be said that in ordinary cases the severity is to be chiefly judged by the prominence of the nervous phenomena. Patients who have suffered from true delirium ferox practically never recover. Much muscular twitching, especially of the face, severe tremor, and intractable insomnia, are all symptoms of most evil omen. Coma must be regarded as a prelude to death. Hyperpyrexia, when it occurs, usually also means a fatal termination. Heart-failure from toxæmia is usually ushered in by a considerable increase in the rate of the pulse. A rate above 120 is to be regarded as a cause for anxiety; above 130, the patient's condition is most serious. Very rapid respirations, showing a large amount of congestion, or a possible complicating pneumonia, are also a bad sign. Diarrhœa is usually serious, except that looseness of the bowels which so often precedes or accompanies the crisis. Sweating is a particularly ugly symptom.

It may be said roughly that the severity of the disease may be judged by the amount and character of the eruption. Bad cases nearly always have very profuse, dark, and deeply stained eruptions, and much purpuric hæmorrhage. On the other hand, a scanty or transient eruption is usually found in cases of less gravity. A fall of temperature, very slight but comparatively steady from the commencement of the second week towards the time of the true crisis, is of favourable import.

TREATMENT.—In a disease which runs so definite a course it is hardly to be expected that we are likely to find any general treatment likely to check the course of the fever. We are therefore reduced to treating complications as they arise, while at the same time maintaining the strength of the patient to the best of our ability. All agree that typhus cases require a large amount of fresh air. While we are unaware if any experiment in actual open-air treatment has yet been attempted, there can be hardly any doubt that some such method will be eventually adopted. In the meantime it will be found useful to keep the temperature of the ward low, and all windows and doors open to the fullest extent. The patient should not be overburdened with bedclothes, a blanket and sheet being amply sufficient covering. Great comfort is given by the use of frequent tepid sponges, and some go so far as to use baths of cold water, as in typhoid fever, as a systematic treatment; but, as in the other fevers, there is little to be gained by direct attempts to lower the temperature. Still more undesirable is the use of antipyretic drugs with that object.

The diet should be light and nourishing. The digestion is practically as weak as it is in typhoid fever, and a milk and beef-tea regimen is to be preferred. To it may be added various meat juices, such as Bovinin, especially valuable when there is diarrhœa and when hot beef-tea is contra-indicated. Benger's food, or other similar preparations, plasmon, and an occasional egg-flip, will be found useful in maintaining the strength. It is absolutely necessary that cold water be supplied in abundance. It assists the elimination of toxines and helps to moisten the mouth and tongue. It should be forced upon the patients, who are often unable to ask for it. I consider five or six pints a desirable quantity in the twenty-four hours, and I have seen patients drink as much as ten pints with great benefit to themselves. As regards the question of stimulants, it is wise to allow all alcoholic cases a little from the first. Most cases require alcohol in their second week, and it may have to be very freely pushed at the time of the crisis.

As regards the use of drugs when the pulse is failing, the use of many of them will be found disappointing. Strophanthus is occasionally useful, but often fails to make any impression, and strychnine, which is on the whole more reliable, is too apt to increase the ataxic symptoms of the patient. It is, above all, important to secure sufficient sleep for the sufferer, and not more than one sleepless night should be allowed before recourse is made to hypnotics. I have had most success with sulphonal in the early stages of the disease, and later on paraldehyde, given freely. Before drugs are used, however, every device of nursing to induce

sleep, sponges, hot drinks, etc., should be tried.

Great attention must be paid to the condition of the mouth, which should be swabbed frequently with boroglyceride or some similar preparation. The skin of the back must be carefully watched, and bed-sores avoided by judicious nursing. The bladder is always a source of anxiety, and should be percussed daily to see that no accumulation of urine is allowed to occur. Constipation, a frequent condition in this fever, should be treated most cautiously. An injudicious dose of castor oil, if given too far on in the fever, may set up an intractable diarrhœa. An enema after the first week is over is much safer. As regards complications they must be treated on general lines.

Prophylaxis.—The general improvement in the sanitary conditions of our great cities has practically removed all chances of a recurrence of the great epidemics of former days. The regulation of common lodging-houses, the abolition of one-roomed dwellings, and the destruction of our worst slums, seem likely to abolish typhus altogether. In presence of an outbreak thorough inspection and disinfection of suspected localities, and a rigid quarantine of all persons exposed to the infection, will soon stamp out the disease. In hospitals the thorough ventilation of the wards, and a liberal allowance of cubic air-space per patient, say 2500 to 3000 cubic feet per bed, have been found to protect the attendants on the sick from contracting the fever. The frequent sponging of the patients with antiseptic solutions, such as Jeyes' fluid in water, is probably also of some prophylactic value. It is also important for the physicians and nurses to be in good health, and no one who is at all "run down" or ailing should be permitted to enter a typhus ward.

Tyremesis.—The vomiting of cheesy or caseous matters, as in the case of young infants (Gr. τυρός, cheese).

Tyro-.—In compound words tyro- (Gr. τυρός, cheese) means cheesy, curdy, or caseous, *e.g. tyroma*, a caseous mass; *tyrosis*, caseation; and *tyrotoxism*, poisoning with cheese.

Tyrosin.—An amido-acid ($C_9H_{11}NO_3$) produced by the decomposition of proteids under the influence of trypsin. *See* Physiology, Protoplasm (*Products of Decomposition*); Physiology, Food and Digestion (*Pancreatic Secretion*). *See also* Bronchi, Bronchitis (*Fœtid Variety*); Expectoration (*Crystals*); Urine, Pathological Changes in (*Sediments, Tyrosin*).

Tyrotoxicon. — A ptomaine obtained from poisonous cheese, ice-cream, milk, etc., and causing symptoms like those of summer diarrhœa (of which, indeed, it is regarded as the cause). *See* Toxicology (Animal Foods, *Cheese*).

Tyrrel's Fascia. — The recto-vesical fascia lying between the prostate gland and the rectal canal; Dénonvilliers' Fascia.

Tyson's Glands.—The sebaceous glands of the prepuce which secrete the smegma. *See* Penis, Surgical Affections of (*Balanitis, Causes*).

Uberty. — Fecundity or fertility (Lat. *uber*, an udder).

Ucambin or Ukambin.—An African arrow-poison, resembling strophanthus in its pharmacological actions.

Uchetu. *See* Leprosy (*History, Synonyms*).

Udransky's (Von) Test.—A name given to two tests, one for bile acids and the other for tyrosin; in both cases a solution of furforol ($C_5H_4O_2$) and sulphuric acid is used, and this in the presence of bile acids gives a red colour with a shade of blue and in presence of tyrosin a pink mixture.

Uffelmann's Test.—A method of distinguishing between lactic acid and hydrochloric acid in the stomach contents; a solution of carbolic acid, water, and a few drops of neutral ferric chloride which is blue in colour becomes yellow in the presence of lactic acid, while hydrochloric acid renders it colourless.

Uhlenhuth's Test.—A serum test for the recognition of human blood (in stains, etc.); it is a precipitin test, and enables one not only to differentiate human from animal blood, but also to discover what animal the blood has come from if it be not human. *See* Robertson, W. G. A., *Trans. Med.-Chir. Soc. Edinb.*, vol. xxv. p. 47, 1906.

Ulæmorrhagia. — Hæmorrhage from the gums (Gr. οὖλα, the gums), as in scurvy, mercurial poisoning, etc.

Ulcers and Ulceration.

Definitions 211
Conditions which prevent Healing . 211
The Healing Sore 211
A Septic Ulcer 212
Classification of Ulcers 212
A. *Arrangement of Ulcers according to Cause* . . . 212
1. *Ulcers due to Traumatism* . 212
2. *Ulcers due to Imperfect Circulation* . . . 212
3. *Ulcers due to Imperfect Nerve-Supply* . . . 213
4. *Ulcers due to Constitutional Causes* 213
5. *Ulcers due to Malignant Disease* 214

B. *Arrangement of Ulcers according to Condition* . . . 214
1. *Healing* 214
2. *Stationary* 214
3. *Spreading* 215
Treatment of Ulcers 215
Principles applicable to all Ulcers 215
Management of Healing Sore . 216
Treatment of Special Varieties . 216

See also BURNS AND SCALDS (*Treatment of Results*); DIABETES MELLITUS (*Symptoms and Complications, The Skin*); DYSENTERY (*Pathological Anatomy*); INTESTINES, DISEASES OF (*Ulcers*); MALINGERING (*Ulcers*); MAMMARY GLAND, DISEASES OF (*Nipple and Areola, Ulcers of*); MENSTRUATION AND ITS DISORDERS (*Vicarious Menstruation, from Ulcers*); OXYGEN (*External Uses*); PALATE (*Diseases, Syphilis, Secondary Lesions*); PERITONEUM, ACUTE PERITONITIS, GENERAL (*Etiology*); PUERPERIUM, PATHOLOGY (*Affections of Nipples*); SKIN, DISEASES OF THE TROPICS (*Tropical Phagedæna*); SKIN GRAFTING AND ALLIED PROCEDURES (*Cases Suitable*); SPRUE (*Pathological Anatomy*); STOMACH AND DUODENUM, DISEASES OF (*Ulceration*); STOMACH, SURGICAL (*Gastric Ulcer*); STOMATITIS (*Aphthous*); SUPPURATION (*Ulcers*); SYPHILIS; TABES DORSALIS (*Symptomatology, Perforating Ulcer of the Foot*); TONGUE, DISEASES OF; etc.

DEFINITIONS.—An *ulcer* may be defined as an open sore or wound in which there are present certain conditions tending to prevent it undergoing the natural process of repair.

The process by which an ulcer is formed, and by which it increases in size—*ulceration*—may be defined as the molecular or cellular death of tissue taking place on a free surface. It is, to all intents and purposes, of the same nature as the process of suppuration (*q.v.*), the only important difference being that while in suppuration the purulent discharge resulting from the death of the tissue elements collects in a closed cavity and forms an abscess, in ulceration it escapes at once on the surface.

CONDITIONS WHICH PREVENT HEALING.—Of the conditions which tend to interfere with the spontaneous healing of an ulcer the most potent is the presence of *pathogenic bacteria*. These organisms not only prevent healing, but by producing irritation and causing destruction of tissue they lead to an actual increase in the size of the sore.

Œdema and *venous congestion* impede healing by interfering with the nutrition of the affected part, and *induration* of the area surrounding an ulcer also prevents healing, as the tissues are unable to contract. *Interference with the nerve-supply* of the tissues, such as occurs, for example, in spinal disease, infantile paralysis, and certain nervous diseases, may also play an important part in delaying repair. In Bright's disease, diabetes, syphilis, and certain other *general constitutional conditions*, the vitiated state of the tissues is an impediment to repair. It goes without saying that unsuitable dressings, ill-fitting appliances, and other *mechanical causes*, may also interfere with the healing of an ulcer.

Clinical Examination of an Ulcer. — In examining an ulcer clinically attention should be directed to the condition of — (1) Its *base or floor*, noting the presence or absence of granulations, and, if present, their disposition, size, colour and vascularity, and whether they are depressed or elevated in relation to the surrounding parts. (2) The *discharge* from the sore should be examined as to its quantity, consistence, colour, composition and odour. (3) The condition of the *edges* is of great importance, and it should be observed whether or not the marginal epithelium is attempting to grow in over the surface; the shape, regularity and thickness of the edges should also be noted, and whether they are undermined or overlapping, everted or depressed. (4) The *surrounding parts* are examined as to whether they are congested, œdematous, inflamed, indurated, or otherwise. (5) The question of *pain* and tenderness in the raw surface or its surroundings should be inquired into. (6) Lastly, the *situation* of the ulcer should be noted, as certain varieties of ulcer tend to occur with greater frequency on some parts of the body than on others; for example, the varicose ulcer on the front of the leg and the perforating ulcer on the foot, and so on.

THE HEALING SORE.—So many varieties of ulcer are met with clinically that it is necessary for purposes of comparison to recognise a type, and this is conveniently found in the *healing sore*, which forms after a portion of skin has been excised aseptically, for example, in the removal of a small cutaneous tumour, or in the operation of skin grafting. After the removal of such a portion of skin the raw surface soon becomes covered with a layer of coagulated blood, fibrin, and lymph, which in the course of a few days is replaced by the growth of granulations. These granulations are of uniform size, pink in colour, and exude a small amount of serous fluid containing a few dead leucocytes. They grow until they reach the level of the surrounding skin, and so fill in the gap with a fine velvety mass of "granulation tissue." The marginal epithelium spreads in over this granulation tissue as a fine pellicle. Towards the inner edge the young epithelium is very thin, of a delicate bluish-white colour, and slightly striated; further out it is thicker and paler. The surrounding parts are healthy or they may show a slight degree of hyperæmia. There is no pain in the sore, and the patient is not feverish. Such a healing sore may be

taken as the ideal form of ulcer, and used as a type with which to compare the ulcer seen at the bed-side.

A Septic Ulcer. — We may contrast, for example, the characters of an aseptic healing sore with those of one in which septic infection has occurred. As a result of the action of the bacteria the effused blood and serum undergo septic changes, and are converted into pus. The base of the ulcer becomes red and inflamed, and is bathed in thin yellow or grumous pus, with perhaps here and there small sloughs of the superficial layers of tissue. No granulation tissue forms. The discharge is profuse, thin, acrid, and offensive, and consists of pus, broken-down blood-clot, and sloughs. The edges are inflamed, irregular, and ragged, and there is no sign of young epithelium growing in at the margins. On the contrary, the sore may be actually increasing in area by the breaking down of the tissues at the margins. The surrounding parts are also inflamed, hot, red, swollen, and œdematous, and the whole region is painful and tender. The patient's temperature is probably raised, and there are other signs of fever. Between the aseptic healing sore and the septic ulcer here described all transition forms are met with.

Classification of Ulcers.—The chief difficulty in arriving at a satisfactory and useful classification of ulcers is that of finding a basis of classification. Theoretically the best basis would be that of cause, and if this were adopted a classification which would at least be logical might be framed. It would, however, involve abandoning many of the terms now in current use, and the substitution of new names might lead to confusion. The nomenclature is already sufficiently involved. We speak, for example, of the "traumatic," the "septic," and the "varicose" ulcer in relation to the factor which produces or maintains the condition; of the "syphilitic," the "gouty," and the "diabetic" ulcer, according to the underlying constitutional element; and of the "weak," the "inflamed," the "callous," and the "healing" ulcer, according to the condition in which the sore happens to be when seen by the surgeon. It must be confessed that many of these terms are so convenient and descriptive, and are so well established in surgical nomenclature, that it is undesirable to attempt to abolish them. It would appear, therefore, that we must remain content with a clinical arrangement of ulcers,—it cannot be called a classification,—considering a given ulcer from two points of view—firstly, its *cause*, and, secondly, its *present condition*. Such a method of studying ulcers has the practical advantage that it furnishes us with the main indications for treatment. The cause must be removed, and the conditions so modified as to convert the ulcer into an aseptic healing sore.

A. *Arrangement of Ulcers according to their Cause.*—If we consider ulcers from the point of view of their cause we find that they fall into five main groups:—

1. Ulcers due to traumatism.
2. ,, ,, imperfect circulation.
3. ,, ,, interference with nerve-supply.
4. Ulcers due to constitutional causes.
5. ,, ,, malignant disease.

Each of these groups will be found to contain several subdivisions which will be described below. In all varieties the influence of pyogenic bacteria is a most important factor in their causation.

1. *Ulcers due to Traumatism.*—(*a*) An ulcer may be produced by a direct *crush* or *bruise* by which the skin of the part is completely destroyed, or so far devitalised that it forms a suitable soil for bacteria. This is very common on the leg, for example, where the skin has been grazed by a falling object or by a carriage wheel. In the course of a few days the damaged area becomes of a greyish white colour, large blebs form over it, and the skin dies. When the dead skin is thrown off a raw surface is left covered with feeble granulations, and having small, shreddy sloughs adhering to it.

(*b*) Similarly an ulcer may be the result of a *burn* or *scald* (vol. ii. p. 34), or of exposure to the Rontgen rays.

(*c*) The "*pressure sore*" is another example of an ulcer due to traumatism. It usually occurs over bony prominences such as the malleoli, the condyles of the humerus or femur, the head of the fibula or the base of the fifth metatarsal, as a result of ill-fitting splints or other appliances. While it is most frequently met with in patients who are paralysed, or who suffer from some nervous lesion, such as infantile paralysis or injury of the spinal cord or nerve-trunks, it may also occur in patients with a perfectly healthy nervous system. On removing the splint the skin of the part pressed upon is found to be of a red or pink colour, with a pale grey spot in the centre, which eventually gives way by sloughing, and leaves an ulcer. *Bed-sores* (vol. i. p. 365) are also to a certain extent the result of undue or prolonged pressure.

2. *Ulcers due to Imperfect Circulation.*—Any interference with the *venous return* from a part plays a most important rôle in producing ulceration. One of the most familiar examples of ulceration due to defective venous circulation is the common "leg ulcer," so frequently met with in the out-patient room in hospital practice. This form of ulcer occurs almost invariably on the front and inner aspects of the lower third of the leg, where, according to Hilton, the anastomosis between the superficial and deep veins is less free than elsewhere, with the result that extra stress is thrown upon the surface veins

and the nutrition of the skin is thereby interfered with. That the backward pressure of the column of venous blood and the retarded circulation through the veins are important factors in the causation of this variety of ulcer, is proved by the fact that healing only takes place if the patient is confined to bed and the limb elevated. So long as he is going about, all methods of treatment are equally futile, whereas improvement is evident at once if he remains recumbent. In a considerable number of cases, but by no means in all, this form of ulcer is associated with the presence of varicose veins, hence it is often spoken of as the *varicose ulcer*.

The actual ulceration is usually determined by some slight injury or irritation which permits of septic infection of the devitalised skin. As dermatitis and eczema are common along the course of varicose veins, the scratching or rubbing of the part is very liable to break the surface and afford a point of entrance for bacteria. In other cases the ulcer is due to the bursting of a small periphlebitic abscess.

When the ulcer is comparatively small and superficial the destructive process frequently spreads to the walls of the subjacent vessels, and leads to profuse and often dangerous bleeding. In large ulcers which have existed for a considerable time the adjacent veins usually become thrombosed, and may even be transformed into fibrous cords, so that hæmorrhage in such cases is not a common complication.

These "leg" or "varicose" ulcers are at first small and superficial, but from want of care, continued standing or walking, or injudicious treatment, they gradually become larger and deeper. They are not infrequently multiple, and this, together with their depth, may lead to their being mistaken for syphilitic ulcers. The base is covered with imperfectly formed, soft, œdematous granulations, which give off a thin, sero-purulent discharge. The edges show no evidence of healing, and are usually somewhat inflamed. The surrounding parts are pigmented and œdematous, and there is as a rule little pain. This variety of ulcer is particularly prone to pass into the condition known as "callous."

Defective arterial supply is less frequently a cause of ulceration than defective venous return. An atheromatous condition of the minute vessels of the skin, especially in peripheral parts of the body, is apt to favour ulceration by diminishing the supply of arterial blood.

In anæmic patients, especially young girls, ulcers are occasionally met with which have many of the characters of those associated with imperfect venous return. They are very slow to heal, and are liable to pass into the condition known as "weak."

3. *Ulcers due to Interference with Nerve-Supply.*—Any interference with the nerve-supply of the superficial tissues predisposes to ulceration. For example, *trophic ulcers* are liable to occur in injuries or diseases of the spinal cord, in cerebral paralysis, in infantile paralysis, in ascending or peripheral neuritis, or after injuries of nerve-trunks. As undue pressure is usually an associated cause these ulcers are sometimes spoken of as *pressure ulcers*. As examples may be cited the ordinary *pressure bed-sore* (vol. i. p. 365), which in many cases occurs in patients suffering from some nervous lesion, and the sore which results from the pressure of ill-adjusted splints.

The *acute bed-sore* is a very rapidly progressing form of ulceration which occurs in patients who have had the trophic nerve-supply to the skin interfered with.

The perforating ulcer of the foot is a peculiar type of sore which occurs in association with different forms of peripheral neuritis, and with various lesions of the brain and spinal cord, such as general paralysis, locomotor ataxia, or syringomyelia. It also occurs in patients suffering from glycosuria. Perforating ulcer is met with most frequently under the head of the metatarsal bone of the great toe of labouring men over forty years of age. A callosity forms, and suppuration occurs under it, the pus escaping through a small opening in the centre. The process slowly and gradually spreads deeper and deeper, till eventually the bone or joint is reached, and becomes implicated in the destructive process—hence the term "perforating ulcer." Sometimes the flexor tendons are destroyed, and the toe is dorsiflexed by the unopposed extensors. A few red, firm, wart-like granulations cover the base of the ulcer, and the cavity is filled with effete and decomposing epithelium thrown off from the ingrowing margins. The ulcer is often represented by a long, narrow sinus, and its true nature is liable to be overlooked. It is characteristic of perforating ulcer that the raw surface is absolutely insensitive, and the sinus can be probed and handled without giving the patient the least discomfort. The chronic and intractable character of these ulcers is due to the interference with the trophic nerve-supply of the part, and to the fact that the epithelium of the skin grows in and lines the tract leading down to the ulcer, and so prevents it closing. Perforating ulcers are sometimes met with on the sides or dorsum of the foot, or even on the hands and on other parts of the body where pressure is but seldom applied. It is to be borne in mind that other ulcers occur in the foot under callosities independently of any nerve lesion. Although very slow to heal they do not show the same tendency to burrow and persist as the perforating ulcer does.

4. *Ulcers due to Constitutional Causes.*—Various constitutional dyscrasiæ favour the production of ulcers by diminishing the power of

resistance in the tissues. In some cases the tissue weakness results from the action of a specific pathogenic virus, as in tuberculosis, syphilis, or leprosy; while in others, so far as we at present know, it depends on impurities or deficiencies of the blood, as for example in the ulcers met with in patients suffering from diabetes, Bright's disease, scurvy, and gout.

(*a*) The *tuberculous ulcer* so often seen in the neck, in the vicinity of joints, and over the ribs and sternum, usually results from the bursting of a tuberculous abscess through the skin. The base is soft, pale, and covered with feeble granulations and grey shreddy sloughs. The edges are of a dull blue or purple colour, and they gradually thin out towards their free margins. But the most characteristic feature of the edges of this variety of ulcer is the fact that they are undermined, so that a probe can be passed for some distance between the floor of the ulcer and the thinned-out edges. Devitalised tags of skin often stretch from side to side of the ulcer. The outline of the ulcer is irregular, and small perforations often occur through the skin. A thin, watery discharge containing grey shreds of tubercular débris escapes. Pain is seldom present.

In the disease known as *lupus* (vol. vi. p. 89) we have another form of tuberculous ulceration, resulting from the fusion of several broken-down tuberculous nodules. The resulting raw surface has a base similar in character to the last, but the edges, instead of being undermined, are studded over with small brown nodules which have not yet fused with the main area.

(*b*) The *syphilitic ulcer* is usually formed by the breaking down of a cutaneous or subcutaneous gumma. It is at first covered by a tough, greyish slough, aptly compared to "wash-leather," which slowly separates and leaves a more or less circular, deep, punched-out surface, on which are seen a few feeble, unhealthy granulations and small sloughs. The edges are raised and indurated; and the discharge is thick, glairy, and peculiarly offensive. The parts around the ulcer are congested and of a dark-brown colour. There are usually several such ulcers together, and as they tend to heal at one part while they spread at another, the affected area assumes a serpiginous outline. Syphilitic ulcers may be met with in any part of the body, but are most frequent in the upper part of the leg, especially around the knee-joint in women, and over the ribs and sternum. The cicatrix which results after healing is usually somewhat depressed.

(*c*) The *scorbutic ulcer* occurs in patients suffering from scurvy, and is characterised chiefly by its marked tendency to bleed.

(*d*) In *gouty* patients small ulcers which are exceedingly irritable and painful occur.

5. *Ulcers due to Malignant Disease.*—It is necessary to mention here certain ulcerative conditions which occur in association with malignant disease. In scirrhus of the breast, epithelioma of the tongue or lip, and in sarcoma, for example, the skin or mucous membrane is liable to be invaded and destroyed by the growth which "fungates" and forms an open vascular sore. These are sometimes spoken of as malignant ulcers, but as they are essentially different in their nature from all other forms of ulcers, and call for totally different treatment, it is best to consider them as complications of the particular tumour and tissue in which they are met. The so-called rodent ulcer and the epithelioma of the skin are described elsewhere.

B. *Arrangement of Ulcers according to their Condition.*—Having arrived at an opinion as to the cause of a given ulcer, and placed it in one or other of the above groups, the next question the surgeon has to ask himself is, "In what condition do I find this ulcer at the present moment?"

Any ulcer may be said to be in one of three states; it is either healing, stationary, or spreading. It is true that we frequently find healing going on at one part while the ulcer is spreading at another.

1. *The Healing Ulcer.*—The process of healing in an ulcer is brought about by the formation of granulation tissue, which becomes converted into fibrous tissue, and is covered over by epithelium growing in from the adjacent skin edges.

The characters of an aseptic healing sore have already been described (p. 211), and we have seen that these characters constitute the ideal state of an ulcer. Save as a result of treatment, however, we seldom meet with ulcers in the healing state clinically.

2. Those that are *stationary*—that is, neither healing nor spreading—may be in one of several conditions.

(*a*) *The Weak Condition.*—Any ulcer may get into a weak state from receiving a blood-supply which is defective either in quantity or in quality. The granulations on the base are small, smooth, and of a pale yellow or grey colour. The discharge is small in amount, and consists of thin serum and a few pus cells. This dries on the edges and forms a scab which prevents the growth of epithelium.

Should the part become œdematous, either from general causes, such as heart, kidney, or liver disease, or from local causes, such us varicose veins, the granulations share in the œdema, and a large quantity of thin serous discharge escapes, but no healing takes place.

The excessive use of moist dressings leads to a third variety of weak ulcer, namely, one in which the granulations become large, soft, pale, and flabby, projecting beyond the level of the skin, and overlapping the edges, which become pale and sodden and cease to grow. This con-

stitutes what is popularly known as "proud flesh." The same condition may arise when induration around prevents contraction of the sore.

(*b*) *The Callous Condition.*—This condition is usually met with in ulcers on the lower third of the leg, and is often associated with the presence of varicose veins. The want of healing is mainly due to the impeded venous return and to the œdematous and indurated condition of the parts around. This induration of the surrounding tissues results from coagulation and partial organisation of the inflammatory effusion, and prevents the contraction of the sore necessary to repair. The base of a callous ulcer lies at some distance below the level of the swollen, thickened, and white edges, and presents a glazed appearance. Such granulations as are present are unhealthy and irregular in size. Foul-smelling yellow pus or sloughs cover the surface, from which a quantity of thin serum exudes. As a rule the condition is painless. The sufferers from this variety of ulcer usually belong to the labouring classes who are underfed, overwrought, wanting in cleanliness, and often excessive in the use of alcohol.

Bone Changes.—The prolonged hyperæmia of the tissues in relation to a callous ulcer of the leg often leads to peculiar changes in the underlying bones (Reclus). The periosteum is abnormally thick and vascular, the superficial layers of the bone become injected and porous, and the bone as a whole is thickened. "The surface is covered with irregular stalactite-like processes of foliaceous masses, which to a certain extent follow the line of attachment of the interosseous membrane and of the intermuscular septa" (Cathcart). When the whole thickness of the soft tissues is destroyed by the ulcerative process the area of bone which comes to form the base of the ulcer projects as a flat, porous node. In course of time the ulcer eats its way into the bone and produces depressions or erosions on its surface. These changes have often been mistaken for primary bone diseases.

3. *Ulcers which are spreading* may be met with in one of several conditions.

(*a*) *The Inflamed Condition.*—Any ulcer may become acutely inflamed as a result of fresh infection with active bacteria. Mechanical irritation, such as is produced, for example, by direct injury, by the pressure or friction of ill-fitting splints or bandages, or even by excessive use of the part, tends to aggravate the inflammation, as does also chemical irritation by strong or unsuitable antiseptic applications. The most typical example of an inflamed ulcer is the venereal soft sore. The base of the ulcer becomes red and angry-looking, the granulations disappear, and a copious discharge of thin yellow pus mixed with blood escapes. Sloughs of granulation tissue or of connective tissue may form. The edges become red, ragged, and everted, and the ulcer increases in size by spreading into the inflamed and œdematous surrounding tissue. Such ulcers are frequently multiple. Pain is a constant symptom and is often severe, and there is usually some constitutional disturbance.

(*b*) *The irritable condition* is met with in ulcers which occur, as a rule, just above the external malleolus in women of neurotic temperament. They are small in size and have prominent granulations, and by the aid of a probe, points of excessive tenderness may be discovered scattered over the surface of the ulcer. These, Hilton believed, correspond to exposed nerve filaments. The small ulcers met with in gouty subjects are also liable to pass into this irritable condition.

(*c*) *The phagedænic condition* is the result of an ulcer being infected with specially virulent bacteria. It often occurs in syphilitic ulcers, and leads to a very rapid and destructive infiltration of the surrounding tissues, with the formation of sloughs and much pus. All the signs of infiltration, both local and constitutional, are present to a marked degree, and the absorption of toxines may lead to a fatal result.

(*d*) *The hæmorrhagic condition* is seen in its most typical form in scorbutic ulcers. The sore is swollen and vascular, bleeds very readily, and is often covered with a thick coagulum. Ulcers which have been brought into a weak condition by too much poulticing frequently bleed freely, as do also those associated with varicose veins should a vein become eroded.

Treatment of Ulcers.—The prompt and patient treatment of ulcers is a matter of great practical importance. An ulcer is not only an immediate cause of suffering to the patient, crippling and incapacitating him for his work, but is a distinct and constant menace to his health. The prolonged discharge reduces his strength; the open sore is a possible source of infection by the organisms of suppuration, erysipelas, or other specific diseases. Phlebitis, with formation of septic emboli, leading to pyæmia, is an ever-present risk; and in old persons it is not uncommon for ulcers of long standing to become the seat of epithelioma. In addition, the offensive odour of the ulcer renders the patient in many cases a source of annoyance and discomfort to his friends; and the expense incurred for dressings is a constant drain on his limited resources.

Principles of Treatment applicable to all Ulcers.—The primary object of treatment in any ulcer is to bring it into the condition of a healing sore.

When this has been effected nature will do the rest, provided extraneous sources of irritation be excluded.

Rest is an essential condition to the repair of all ulcers, and is best secured by putting the patient to bed and applying some form of splint.

Elevation of the part to facilitate the venous return is also most important. The limb should be placed on pillows, or the foot of the bed raised upon blocks, so that the ulcer lies at a higher level than the heart. These measures will at the same time encourage the absorption of inflammatory exudations around the ulcer, and this may be aided by *massage*.

The use of *elastic pressure* or of strapping also accelerates the removal of exudation, and so facilitates the contraction of the sore. When the induration is very dense nothing is so good as *blistering*. A cantharides plaster, or a few coats of blistering fluid, may be applied all over the indurated area, avoiding the open surface.

The *removal of all sources of irritation*, such as unsuitable dressings, ill-fitting splints, irritating applications, and, above all, septic organisms and their products, is imperative.

A septic ulcer must be thoroughly disinfected by washing not only the open surface, but also the skin for some distance around it, with lysol (1 in 100) to remove grease, and then with Lister's strong solution, or carbolic lotion (1 in 20). Hair-covered parts should be shaved. The raw surface should then be swabbed over with undiluted carbolic acid. If the granulations are œdematous and unhealthy they may be removed with a sharp spoon.

An antiseptic dressing of gauze or boracic lint is then applied and changed daily till the wound assumes a healthy appearance.

As in all conditions in which the patient's health is below par, good nourishing food, tonics, and general hygienic treatment are clearly indicated.

By paying attention to these general rules, and by employing certain special measures suited according to the particular varieties of ulcers in hand, the conditions of a healing sore will soon be obtained.

Management of a Healing Sore.—Perhaps the best dressing to apply to a healing sore is a layer of Lister's perforated oiled-silk protective, which is made to cover the raw surface and about a quarter of an inch beyond its margins. Over this three or four thicknesses of boracic lint, or of sterilised gauze wrung out of boracic lotion, or sterilised (boiled) water, are applied, and covered by a moderate quantity of antiseptic absorbent wool. A splint is applied to insure rest, and the part is elevated to avoid congestion.

Once in two or three days is often enough to renew the dressing, and care must be taken to avoid any rough handling of the sore. Any discharge which lies on the surface should be removed by a gentle stream of lotion rather than by rubbing. The area around the sore should be carefully cleansed before the fresh dressing is applied.

In some cases healing goes on more rapidly under a dressing of weak boracic ointment (one-quarter the strength of the pharmacopœial preparation). We have found the daily application of bovinine and similar preparations useful in promoting the growth of epithelium and hastening healing. In other cases any local application seems to irritate the sore and hinder healing. Under these conditions it has been recommended that a wire cage covered with moist antiseptic gauze be placed over the sore and removed frequently to allow of the discharge being bathed from the surface of the sore.

In the treatment of aseptic healing sores, dusting powders and oily or moist dressings should, as a general rule, be avoided.

Treatment of Special Varieties of Ulcers.—Before beginning to treat a given ulcer two questions have to be answered: first, what is the cause underlying this ulcer? and, second, in what condition do I find it? or, in other words, in what particulars does it differ from a healing sore?

If the cause be a local one, it must be removed; if a constitutional one, means must be taken to counteract it. This done, the condition of the ulcer must be so modified as to bring it into the state of a healthy healing sore, after which it will be managed on the lines already laid down.

A. *Treatment in relation to the Cause of the Ulcer.*—I. *Traumatic Group.*—The *prophylaxis* of these ulcers consists in rigidly excluding bacteria, by cleansing crushed or bruised parts with antiseptic lotions, and applying mild, non-irritating dressings, such as boracic lint and corrosive wool, and properly adjusted splints. These measures will often prevent a grossly injured portion of skin from sloughing, and will insure its asepticity should it do so. In the event of the skin giving way the same form of dressing should be continued till the slough has separated and a healthy granulating surface is formed. The protective dressing appropriate to a healing sore is then substituted.

The treatment of *burns and scalds* is described in vol. ii. p. 36.

Pressure sores should be prevented by carefully padding, with rings of absorbent wool, any bony points liable to be unduly pressed upon by splints. The use of a dry dusting powder materially helps the avoidance of pressure sores. When they occur they are treated by protective dressings.

II. In the case of *ulcers due to interference with venous return*, for example those associated with varicose veins, the primary indication is to elevate the limb to facilitate the free flow of blood in the veins, and so insure that a supply of pure, healthy blood shall reach the sore. Should the varicose veins be suitable for operative treatment, this often greatly expedites the healing of an ulcer, although it is to be borne in mind that the operation wound is more

liable to become infected in the presence of an ulcer than after this has healed.

The quality of the blood may be improved by the administration of iron, strychnine, quinine, and similar tonics.

III. When an *imperfect nerve-supply* is the main factor underlying ulcer formation, prophylaxis is the chief consideration. In patients suffering from spinal injuries or diseases, cerebral paralysis, or affections of the peripheral nerves, all sources of irritation, such as ill-fitting splints or tight bandages, moist applications, and hot bottles, should be avoided. Any part liable to pressure, from the position of the patient or otherwise, must be carefully protected by pads of wool, air-cushions, or water-bags, and must be kept absolutely dry. The skin should be hardened by daily applications of methylated spirit.

Should an ulcer form in spite of these precautions, the mildest and least irritant antiseptics must be employed for bathing and dressing it, and as far as possible all dressing should be dry. Balsam of Peru is a suitable application in these ulcers until they assume the characters of the aseptic healing sore.

The *perforating ulcer* of the foot calls for special treatment. To avoid pressure on the sole of the foot the patient must be confined to bed. As the main local obstacle to healing is the down-growth of epithelium along the sides of the ulcer, this must be bodily removed by the knife or sharp spoon. The base also should be excised and any bone which may have become involved should be gouged away, so as to leave a healthy and vascular surface. The cavity thus formed is stuffed with iodoform gauze and encouraged to heal from the bottom. After the ulcer is healed the patient should wear in his boot a thick felt sole with a hole cut out opposite the cicatrix. When a joint has been opened into, the difficulty of thoroughly getting rid of all unhealthy and infected granulations is so great that amputation is the speediest and most satisfactory treatment. It is to be borne in mind, however, that ulcers sometimes recur on the stump after amputation.

Stretching of the plantar nerves has been successful in hastening the healing of perforating ulcers (Chipault).

The treatment for any nervous disease or glycosuria which may coexist is, of course, clearly indicated.

The ulcer which forms under callosities on the sole of the foot is treated by paring away all the thickened skin, after softening it with soda fomentations, removing the unhealthy granulations, and applying stimulating dressings.

IV. *Treatment of Ulcers due to Constitutional Dyscrasiæ.*—When ulcers are associated with such constitutional diseases as diabetes, Bright's disease, scurvy, or gout, these must be treated on general principles.

Tuberculous and syphilitic patients must also receive appropriate medicinal and dietetic treatment for their general conditions.

The *tuberculous ulcer*, however, calls for special local treatment, which, in the first place, consists in the thorough removal of all tissue invaded by the bacillus or its products. By means of knife, scissors, or sharp spoon, all foci of tuberculous disease, whether in the skin or in deeper parts, must be freely removed, so as to leave a healthy surface from which granulations may spring up. Should the raw surface left be extensive and likely to result in an unsightly scar, much benefit may result from skin-grafting.

In some cases the area involved is so extensive that the surgeon may hesitate to excise it bodily. The alternative is to clip or scrape away all grossly diseased tissue and granulations, to slit up all undermined sinuses, and then to purify everything with undiluted carbolic acid, after which an ordinary boracic dressing is applied. By many iodoform is held in high esteem as an agent which counteracts the tubercle bacillus and its products.

The healing of such sores is often slow and tedious, and experience shows that frequent changes in the dressings employed are often beneficial.

The Syphilitic Ulcer.—No ulcerative process responds so readily to medicinal treatment as the syphilitic ulcer does to full doses of potassium iodide and mercury and the local application of black wash. When the ulceration has lasted for a long time, however, and is widespread and deep, the duration of treatment is materially shortened by a thorough scraping with a sharp spoon.

B. *Treatment in relation to the Condition of the Ulcer.*—1. *Ulcers in a Weak Condition.*—If the cause of the weak state of the ulcer be general anæmia, or kidney disease accompanied by œdema, these affections must first be treated on general medical lines. Locally the imperfect granulations should be scraped away, and stimulating agents, such as red lotion or sulphate of copper (two grains to the ounce), applied to the raw surface till healthy granulations form.

Frequently this state of ulcers is due to an indurated condition of the surrounding tissues, or to the base of the ulcer having become adherent to bone or dense cicatricial tissue. In such cases incisions into the parts with a view to facilitating contractions are most useful.

When the ulcer has got into a weak condition from the prolonged use of moist dressings, these must be stopped, the redundant granulations clipped away with scissors, the surface rubbed with a silver nitrate stick or a piece of sulphate of copper (bluestone), and dry dressings applied.

When the ulcer has assumed the characters

of an aseptic healing sore a few Thiersch grafts should be applied to hasten cicatrisation.

2. *Ulcers in a callous condition* call for treatment in three directions: (1) The venous return must be facilitated by elevation of the limb, the removal of all constricting appliances, and the employment of massage. (2) The induration of the surrounding parts must be got rid of before contraction of the sore is possible. For this purpose the free application of blisters, as first recommended by Syme, leaves little to be desired. Liquor epispasticus painted over the part, or a large fly-blister (emplastrum cantharides) applied all round the ulcer, speedily disperses the inflammatory products which cause the induration. Pressure applied by imbricated strips of adhesive plaster or by elastic bandages is more troublesome and less satisfactory than Syme's method. (3) The septic element in the ulcer must be eliminated. When the ulcer is very foul, relays of charcoal poultices (three parts of linseed meal to one of charcoal), maintained for thirty-six or forty-eight hours, are useful as a preliminary step. The base of the ulcer and the thickened edges should then be freely scraped with a sharp spoon, and the resulting raw surface sponged over with undiluted carbolic acid, after which an antiseptic dressing is applied and changed daily till healthy granulations appear. When these are slow to form, the sore may be stimulated by a dressing of boracic lint wrung out of red lotion and covered with a layer of gutta-percha tissue. A more radical and very successful method of treatment consists in excising the whole ulcer and grafting the raw surface left.

So soon as the ulcer assumes the characters of an aseptic healing sore, it should be covered with Thiersch skin-grafts, which form a much better cicatrix than is left when the ulcer is allowed to heal without such aid.

Ambulatory Treatment.—When the circumstances of the patient forbid his lying up in bed the healing of the ulcer will be much delayed. He should be instructed to take every possible opportunity of placing the limb in an elevated position, and must constantly wear a firm bandage of *elastic webbing*. This webbing is porous, and admits of evaporation of the skin and wound secretions, an advantage it has over Martin's rubber bandage. The bandage should extend from the toes to well above the knee, and should always be applied while the patient is in the recumbent position with the leg elevated, preferably before getting out of bed in the morning. Additional support is given to the veins if the bandage is applied after the method suggested by Heron Watson.

Unna's Bandage.—A bandage stiffened with gelatine is useful, but somewhat inconvenient for the class of patients who usually suffer from this condition.

3. An *inflamed condition* of an ulcer is usually the result of a fresh infection by active bacteria, and of external forms of irritation. Rest, elevation, and the application of moist antiseptic and soothing dressings are the indications for treatment. Ichthyol ointment spread on lint and applied to the surrounding parts is very soothing. Much relief is often given by making a free incision into the ulcer and the surrounding inflamed parts. A preliminary painting with undiluted carbolic acid will help to check the spread of the bacteria.

The *phagedænic* ulcer is simply an aggravated degree of the inflamed variety produced by some virulent organism or poison, and calls for more energetic treatment to arrest its spread, which is often rapid and destructive. The whole of the affected surface may be touched with the actual cautery at a white heat, and the seared surface sponged over with pure carbolic acid. A simple boracic dressing or iodoform gauze is then applied.

4. The *irritable* state of an ulcer may depend upon constitutional neurotic tendencies, uterine irregularities, or upon a gouty diathesis, and in treatment these factors must be counteracted. Locally the most satisfactory treatment is complete excision and subsequent skin-grafting. Failing this, the use of the sharp spoon, silver nitrate, or pure carbolic acid may be tried. Hilton found that small transverse incisions on the proximal side of the tender spots gave relief from the pain, probably by dividing the exposed nerve filaments.

5. A *hæmorrhagic condition* is more or less accidental, and must be treated according to whether the bleeding results from blood causes as in scurvy, or from excessive friability of granulations as in some cases of weak ulcers.

Ulcerated.—Affected with ulcers, *e.g.* an ulcerated sore throat. *See* PHARYNX, ACUTE PHARYNGITIS (*Septic*).

Ulceration. *See* ULCERS; BLADDER, INJURIES AND DISEASES OF (*Ulceration*); COLOTOMY (*Indications, Ulceration*); GLANDERS (*Pathology*); SCLERODERMIA (*Circumscribed, Morphœa, Clinical Features*).

Ulcerative.—Characterised by the presence of ulcers, *e.g.* ulcerative colitis (*see* TYPHOID FEVER, *Diagnosis*); ulcerative endocarditis (*see* HEART, MYOCARDIUM AND ENDOCARDIUM, Malignant Endocarditis; MALARIA, *Diagnosis*; *and* TYPHOID FEVER, *Diagnosis*); and ulcerative stomatitis (*see* STOMATITIS, *Ulcerative*; MEASLES, *Complications*).

Ulcus.—An ulcer; *e.g.* the *ulcus erodens* (lupus), *ulcus exedens* (rodent ulcer), *ulcus grave* (Madura Foot), *ulcus molle* (chancroid), and *ulcus venereum durum* (chancre).

Ulē.—A cicatrix or scar (Gr. οὐλή, a scar),

hence *ulemorrhagia* (hæmorrhage from a cicatrix), *ulerythema* (erythema with the formation of cicatrices, such as *ulerythema centrifugum* or lupus erythematosus).

Ulexine.—An alkaloid ($C_{11}H_{14}N_2O$) got from *ulex europæus*, which has been used as a diuretic and local anæsthetic.

Ulitis.—Inflammation of the gums (Gr. οὖλα, the gums).

Ulna.—The inner one of the two bones of the forearm; *ulnar* or *ulnaris* is the adjective signifying belonging to the ulna or to a structure (nerve or artery) which is related to that bone. *See* Fractures (*Radius and Ulna*); Arteries, Ligature of (*Ulnar*); Nerves, Peripheral (*Diseases and Injuries of Special Nerves*); Tabes Dorsalis (*Symptomatology, Ulnar Symptom of Biernacki*).

Ulo-.—In compound works *ulo-* (Gr. οὖλον, the gum, οὐλή, a scar; or οὖλος, fleecy or woolly) means usually connected with the gums, but in some words it means scar-like and in others fleecy, e.g. *uloncus* (swelling of the gums), *ulorrhagia* (bleeding from the gums), *ulodermitis* (inflammation of the skin with cicatrices), and *ulotrichous* (having woolly hair).

Ultra-.—In compound words ultra- (Lat. *ultra*, beyond) means excessive, beyond measure; e.g. *ultrabrachycephaly* (an extreme degree of brachycephaly), *ultramicroscope* (a microscope for seeing objects measuring only the millionth part of a millimetre in diameter), *ultraquinine* (homoquinine), etc.

Ultra-Violet Rays. *See* X-Rays (*Nature and Production*).

Ultzmann's Test.—A test for bile pigments, which in solution give an emerald green colour when treated with caustic potash and excess of hydrochloric acid.

Ululation.—Howling or hooting like an animal, *e.g.* in hysteria (Lat. *ululare*, to howl).

Umbilical.—Related to the umbilicus or to the vessels and cord which are associated with it. *See* Umbilicus.

Umbilicus, Diseases of.

Hæmorrhage 219
Sepsis and Infective Processes . . 219
Eczema 220
Syphilis. 220
Fistulæ. 220
Persistent Omphalo-Mesenteric Duct . 220
Tumours 220
Benign 221
Malignant 221
Hernia and Exomphalos . . . 221

See also Abdominal Tumours, Diagnosis of (*Umbilical Region*); Cretinism (*Description, Umbilical Hernia*); Embryology; Fœtus and Ovum, Development of (*Umbilical Cord*); Gall-Bladder and Bile Ducts, Diseases of (*Congenital Obliteration of Bile Ducts, Umbilical Hæmorrhage*); Hæmophilia; Heart, Physiology of (*Embryology*); Hernia (*Varieties, Umbilical*); Labour, Management of (*Tying Cord*, etc.); Labour, Precipitate (*Rupture of Cord*); Labour, Prolonged (*Pelvic Deformities, Prolapse of Cord*); Labour, Faults in the Passenger (*Cord*); Labour, Accidental Complications Affecting the Child (*Abnormalities of the Cord*); Labour, Injuries to Generative Organs (*Inversion, Causes*); New-Born Infant (*Cord, Management, Umbilical Infections*, etc.); Pregnancy, Physiology (*Local Changes, Abdominal Wall*); Pregnancy, Diagnosis (*Funic Souffle*); Pregnancy, Diseases of Placenta and Cord; Urachus (*Tumours*).

Hæmorrhage. — This may occur immediately after birth from slipping of the cord ligature. A more important kind is spontaneous hæmorrhage occurring usually about the fifth day, when the cord separates; it may occur, however, as early as the second or as late as the fifteenth day. Some of these cases have a hæmophilic ancestry; in a few congenital syphilis is present; by some observers imperfect closure of the umbilical vein has been thought to be the cause (hæmorrhagic fistula). Male children are affected in 75 per cent of the cases. The result is usually fatal. In about half the cases jaundice is present with subcutaneous hæmorrhages or purpuric spots in different parts of the body. In some cases there has been loss of blood from the rectum, bladder, or gums. The treatment is unsatisfactory, the hæmorrhage being usually stopped, but after a time recommencing Pressure and various styptics have been tried: cauterisation is inadvisable. Underpinning, or some method of ligature, seems to have been most successful. Possibly the administration of calcium chloride and the local use of suprarenal liquid might be useful.

Sepsis and Infective Processes.—It cannot be too strongly insisted that the ligature and subsequent treatment of the cord stump should be conducted on ordinary antiseptic principles. Inflammation at the umbilicus (omphalitis) in the new-born may be slight, or may go on to ulceration, cellulitis, gangrene, general septicæmia, or pyæmia, this last condition depending probably upon the passage of septic thrombi by the umbilical vein to the liver, lungs, or elsewhere. Erysipelas or general peritonitis may occur, and are almost universally fatal. Severe icterus neonatorum is due to umbilical septicæmia. Diphtheria has affected the umbilicus alone with a fatal result. In tetanus neonatorum the umbilicus is probably the point of

infection, and the bacilli can be detected in the pus at the navel.

In adults, especially fat people, the accumulation of dirt and sebaceous matter in and around the umbilicus may give rise to inflammation or ulceration; erysipelas, cellulitis, or abscess may follow. Actual concretions, made up principally of inspissated sebaceous material, have also been found.

Treatment must be conducted on ordinary lines. In omphalitis mercurial ointment and lotion are of value. The umbilicus may be removed if necessary.

Eczema.—This occurs both in children and stout adults, being in some cases associated with want of cleanliness, in others with the presence of tumours.

Syphilis. — Ulcerative processes, jaundice, and hæmorrhage are more common in congenital syphilis. Hunterian chancres and condylomata have been seen at the umbilicus.

Fistulæ.—(*a*) *Fœcal.*—These, when appearing at the fall of the cord, are due either to the persistence of the omphalo-mesenteric duct (Meckel's diverticulum), in such cases the bowel below the diverticulum being normal or more or less closed, or to inclusion of a piece of bowel in the cord ligature. The fistulous opening may be quite minute, being perhaps situated at the apex of a little tumour consisting of prolapsed mucous membrane, or if the opening is wide, large portions of intestine may be everted through it. Rarely the small gut has been found to open at the umbilicus just above the normal position of the cæcum, all the intestines below this point being absent.

The treatment of these fistulæ depends upon the condition of the bowel below: a minute opening may close without treatment, in other cases a plastic operation may be necessary: finally, a wide diverticulum may be detached from the umbilicus and removed. If the bowel below is closed the umbilical opening must be kept patent.

Fistulæ of later date, in children, are usually due to tuberculous peritonitis. In adults they may arise from peritonitis, tuberculous or from other causes, especially appendix trouble, or from rupture of a strangulated hernia, cellulitis of the abdominal wall that has involved the bowel, or from injury. In rare cases worms have passed through the umbilicus after the adherence of a circumscribed ulcer of the gut. Umbilico-gastric fistulæ, from simple or carcinomatous ulcer of the stomach, may occur.

The treatment of these cases is difficult, often can be merely palliative, and must be conducted on ordinary lines.

(*b*) *Urinary.*— These are always due to a patent urachus; *see* under "Urachus," p. 240.

(*c*) *Biliary.*—These are very rare. They may be congenital, in connection with some abnormality of the gall-bladder or ducts; or more commonly acquired, when, if due to injury or certain inflammatory processes, they may close spontaneously or after operation; but if due to a malignant growth or stricture of the common bile-duct they are best left alone. Gall-stones may pass through these abnormal openings.

(*d*) *Purulent.*—These are due to the opening of an abscess associated with peritonitis from tubercle, appendicitis, or some other cause, intestinal contents and pus being both present in the discharge; from suppuration in an ovarian or hydatid cyst, bile being also present in the latter case; from a urachal abscess, when urine is mixed with the pus; from an abscess in the abdominal wall, or from similar causes.

Persistent Omphalo-Mesenteric Duct. — Hence arise fæcal fistula (*see* above), adenoma in the new-born (*see* below), cysts containing a modified succus entericus, or sacs discharging a similar fluid by a fistulous opening at the umbilicus. Such conditions may be readily understood by remembering that the duct may undergo complete involution in one part, another may remain as a solid fibrous cord, another may undergo development into a structure identical with that of the small intestine.

Tumours.—Of innocent tumours the most important are the polypi; of these the so-called *granulomata* are due to overgrowth of granulation tissue after the fall of the cord. They are little, pedunculated growths, strawberry-coloured, varying in size from a pea to a hazel nut, bleeding easily, and secreting an abundance of sero-purulent fluid. Possibly some originate from allantoic remnants. Microscopically the little tumours show round and fusiform cells and many fine vessels. They may drop off spontaneously or on the application of powdered nitrate of lead. If not, a ligature round their base soon causes desiccation and separation.

The other class of polypi, appearing at the same period, are the *adenomata*. Here a portion of the omphalo-mesenteric duct persists, and the mucous membrane, prolapsing and proliferating, gives rise to a little tumour having the structure of small intestine. Such tumours, the writer has found, have a solid cord passing back from their base to the apex of a Meckel's diverticulum. Clinically we find a red vascular tumour much resembling a granuloma, but having in the middle a little canal ending blindly. Their appearance much resembles rectal prolapse in an infant or a rectal polypus. They grow slowly and may remain stationary for months, seldom becoming larger than a cherry. There is an abundant secretion of fluid which soaks the child's clothes. The writer measured this in one case, and found it averaged 10 to 15 c.c. daily. The fluid was viscid, clear, and distinctly alkaline. It contained albumin, it had no digestive action on proteids or starch. Microscopically these tumours are covered with a layer of glands identical with Lieberkuhn's follicles, some of

which are proliferating. Beneath these are connective tissue and unstriped muscular fibres. The tumours in every way resemble those which surmount a minute fæcal fistula, and are evidently identical with them except that the part connecting them with the Meckel's diverticulum is impervious. They can be removed by ligature or by the knife.

Three cases of umbilical polypus of similar naked-eye appearance showed microscopically the structure of the pyloric end of the stomach; in one (Tillmanns) the secretion was abundant, acid, contained pepsin, and digested the skin.

Other innocent tumours are rare. Angeiomata occur, and sometimes surmount a congenital hernia; dermoid and sebaceous cysts have been seen and sometimes suppurate. Empty hernial sacs which have been tied off may form serous cysts at the navel. Lymphangeiomata have been described, sometimes in association with ovarian cysts. Papillomata, fibromata, and myxomata also occur, but with the above must be regarded as pathological curiosities. Subperitoneal lipomata are common near the umbilicus, making their way to the subcutaneous tissue along the track of one of the perforating arteries.

Malignant tumours are more frequent than benign: scars are not infrequently the seat of malignant change, especially when subjected to the irritation resulting from accumulation of dirt and sebaceous material. Of primary growths epithelioma is the commonest, not differing in appearance from similar growths elsewhere, but glandular enlargement is uncommon, while the tumour has a tendency to grow deeply, invading the peritoneum and intra-abdominal organs. In some cases the abdominal wall around is hardened by the growth, forming a kind of cancer *en cuirasse*.

Secondary carcinomatous growths at the umbilicus are not uncommon, and sometimes invade a hernial sac, the growth spreading from the contained omentum to the sac wall. The primary growth may be in the stomach, intestine, colon, ovary, uterus, peritoneum, or liver. A liver growth may travel to the umbilicus along the lymphatics of the round ligament. Sometimes the intra-abdominal growth is latent.

Sarcomata occur at the umbilicus, but are very rare. Rodent ulcer has been described.

With regard to treatment, primary growths are removed by an elliptical incision, and the same mode of treatment is applicable to secondary growths, if advanced and troublesome, the operation giving an opportunity for exploration of the abdominal cavity.

HERNIA AND EXOMPHALOS.—*See* the article "HERNIA" in vol. iv. p. 201.

Umbo.—A projection or rounded boss, *e.g.* the central part of the tympanic membrane to which the handle of the malleus is attached, drawing it inwards (*umbo membranæ tympanæ*). *See* EAR, EXAMINATION OF (*Tympanic Membrane*).

Unavoidable Hæmorrhage.—Placenta prævia. *See* PREGNANCY, HÆMORRHAGE (*Antepartum, Unavoidable*).

Unborn Infant. *See* FŒTUS AND OVUM, DEVELOPMENT OF; etc.

Unciform.—Shaped like a hook (Lat. *uncus*, a hook), *e.g.* one of the carpal bones which has a hook-like process.

Uncinaria Duodenalis. — The *Ankylostoma duodenale* or *Dochmius duodenalis*, a parasite belonging to the group of the Strongylidæ. *See* PARASITES (*Nematodes, Strongylidæ*).

Uncinariasis. — Disease due to the nematode worms known as uncinaria; ankylostomiasis, miner's anæmia, or hook-worm disease. *See* PARASITES (*Nematodes*).

Uncinate.—Having a hook (Lat. *uncus*, a hook), *e.g.* the uncinate convolution of the brain (the anterior end of the hippocampal gyrus).

Unconscious Cerebration.—Mental and intellectual activity of the brain of which the subject is at the time unconscious, but whose results show themselves later, *e.g.* the recurrence to memory of a name which could not be recalled at first and to recall which all conscious attempt had ceased; mental latency.

Unconsciousness.

INTRODUCTION 221
PHYSICAL BASIS OF CONSCIOUSNESS . . 222
UNCONSCIOUSNESS—
Partial 225
Double Consciousness . . 225
Complete 227

See also BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Hæmorrhage, Symptoms*); COMA and *Cross References*; etc.

THE primary and obvious problem of psychology is the explanation of psychical phenomena. By "explanation" is meant the reduction of the phenomena of our inner world, which are given merely in time, to those actual events which lie at their foundations, and the establishment of the laws according to which the former follow from the latter. For Volkmann the fundamental principles of psychology lie in the actual psychic events, and his genetic method of psychology traces and explains the development of mental life by a combination of empirical and speculative or metaphysical principles.

For the present purpose it would be a work of supererogation to review the polemical and critical discussions on the science of the phenomena of consciousness, for this would involve the production of a treatise on psychology both descriptive and explanatory. It will be expedient to adopt merely some sort of attitude towards those implications of the known phenomena of consciousness, without reference to the various metaphysical hypotheses which have been introduced solely in the attempt to explain the facts.

In our study of the concept of mind it is important to recognise the definite standpoint that all consciousness, and every phenomenon of consciousness, ought to be considered as a form of functioning, and not as a mere differentiation of content. And, just as the task of scientific psychology is truly a description and explanation of phenomena of consciousness, considered as forms of active functioning, so our present task of considering the morbid phenomena or disruptions, so to speak, of consciousness must perforce be descriptive and explanatory without falling back upon the speculative or metaphysical doctrines as to the ground and essence of all mental life.

Active discriminating consciousness and consciousness of *passive* contents must be differentiated and recognised as being in their totality the sum of all the so-called elements comprising consciousness, even though they are only two sides of one and the same consciousness. Inasmuch as consciousness involves the sum total of mental life with its successions and laws of sequence, so "unconsciousness" ought to involve a full consideration of the disruptions of successions of psychoses which derive their characteristics from the nature of their sequence, and of the laws which are shown by the states of consciousness in this sequence.

For simplicity the various cognitive states of consciousness will herein be termed "knowledge," arrived at through the process of development and involving all forms of intellectual activity. Unconsciousness, therefore, implies interference with knowledge, and this interference may be partial or complete.

THE PHYSICAL BASIS OF CONSCIOUSNESS is a subject which has engaged the attention of nearly all writers in physiology, psychology, and philosophy. Here we grant that the mind holds intercourse with the external world through the medium of the nervous mechanism, and any disorder or disease of that mechanism may impair or suspend the intercourse. It is only when we have gained an adequate knowledge of the physiological data, and are able to place them in juxtaposition to concomitant psychological data, that we shall be able to cope with this most difficult of all questions of mental physiology.

Hitherto the tendency has been to assign causes where causes cannot be shown to exist, and deduce extempore doctrines from a very partial view of the influence of cerebral disease upon the phenomena of the mind, and these partial deductions have served to form the basis of an irrational materialism. It will be readily understood that some hallucinations and mental changes cannot be explained by physiological perversion; similarly in discussing the phenomena of unconsciousness we must simply assume that disease of the cerebrum impairs or suspends the intercourse of the substantially unknowable mind with the data derived from the external world.

The scheme of the hierarchy of nervous centres enunciated by Hughlings Jackson is based on an anatomico-physiological basis, and a reference to this scheme serves to explain upon a rational hypothesis many of the phenomena of unconsciousness.

Briefly stated it is as follows:—

"1. The lowest motor centres are the anterior horns of the spinal cord, and also the homologous nuclei for motor cranial nerves higher up. They extend from the lowest spinal anterior horns up to the nuclei for the ocular muscles. They are at once lowest cerebral and lowest cerebellar centres; hence lesion of them cuts off the parts they represent from the whole central nervous system.

"The lowest centres are the most simple and most organised centres; each represents some limited region of the body indirectly, but yet most nearly directly they are representative. The middle motor centres are the convolutions making up Ferrier's motor region and the ganglia of the striatum. These are more complex and less organised, and represent wider regions of the body doubly indirectly; they are representative. The highest motor centres are convolutions in front of the so-called motor region. I say so-called, as I believe, and have urged for many years, that the whole anterior part of the brain is motor, or chiefly motor.[1] The highest motor centres are the most complex and least organised centres; and represent widest regions (movements of all parts of the body), triply indirectly; they are re-re-representative. That the middle motor centres represent over again what all the lowest motor centres have represented, will be disputed by few. I go further, and say that the highest motor centres (frontal lobes) represent over again, in more complex combinations, what the middle motor centres represent. In recapitulation there is increasing complexity, or greater intricacy of representation, so that ultimately the highest motor centres represent, or, in other words, co-ordinate, movements of all parts of the body in the most special and complex combinations."

In regard to the scheme of the sensory centres

[1] *Brit. Med. Journ.*, March 6, 1869.

his conclusions are:—(1) That the highest (chiefly) motor centres, parts in front of the so-called motor region, make up the physical basis of consciousness; and (2) that just as consciousness represents, or is, the whole person psychical, so its anatomical basis (highest centres) represents the whole person psychical, represents impressions and movements of all parts of his body; in old-fashioned language, the highest centres are potentially the whole organism. States of consciousness attend survivals of the fittest states of centres representing the whole organism.

Meynert endeavoured to prove a logical sequence in the evolution of association, and Waller[1] tried to demonstrate the essential similarity of neural processes concomitant with the whole range of subjective phenomena, from the simplest sensation to the most complex judgment. Hitherto the logical mode of symbolising neural processes has merely resolved itself into a disquisition upon the ordinary rules of the syllogism with, in addition, the conception that the physical organism in some way or other subserves the phenomena of sensation.

It must suffice here to state that the various presentations and representations in consciousness are correlative states to physiological activities. Every presentation and representation is an objective content of consciousness. The sum total of the complex physiological activities become manifested as objective contents of consciousness; the *ego* does not view the activities themselves, it merely becomes cognisant of their mental correlative as objective states. The writer has elsewhere said:[2]—"The study of the nervous mechanism has disclosed to us that just as our bodies are associated with the activities of the cosmical system, so our organic nervous structures are associated with the objective phenomena of consciousness. Further, the details of physiology and anatomy would appear to point to the fact that some ultimate and intra-bodily activity is essential to, and possibly conditions every diversification of the sensory and other kinds of experience. Beyond this we cannot go. The mere experience of sensory consciousness in itself affords no scope for ratiocination. In other words, our cerebro-spinal system is capable of receiving and of propagating specific agitations, and also of exerting, in some way or other, a determinate modification of activity in its substance, but we do not in the least degree understand the *modus operandi* whereby we experience the current vicissitudes of consciousness.

The question as to whether individual parts of mental operations are associated with definite parts of the cortex has, in the case of aphasia and allied conditions, assumed fairly definite proportions; but at the best we must remember that the clinical manifestations in these cases may furnish us simply and solely with evidences that the physical paths of conduction, or the organic substrata, are not performing their physical functions satisfactorily. When we attempt to deal with complex mental activities, we cannot imagine any specific functions in the nervous elements which would serve as physical counterparts, and the presence of a chaotic mass of tracts, cells, and other nervous elements, viewed either singly or in combination, do not suggest any explanation of the very simplest psychical process.

The processes of consciousness are manifestly dependent upon the physical organisation of the nervous system, and the cerebral cortex appears to have a more direct and intimate relation with "knowledge" than is manifested by other regions of the cerebro-spinal system.

Professor James has asked the question: "Is consciousness which accompanies the activity of the cortex the only consciousness that man has? or are his lower centres conscious as well?" He conceives that the lower centres themselves may possibly have a "split-off" consciousness of their own similarly ejective to the cortex consciousness. This question may be answered by saying that the cortex cerebri is the sole organ of consciousness in man, and if there be any consciousness pertaining to the lower centres it is a consciousness of which the self knows nothing. But the possibility of consciousness pertaining to any of the lower centres is not to be estimated by the complexity or fitness of the reactive manifestations as viewed objectively, otherwise we are free to assume the existence of consciousness in still lower centres of the cerebro-spinal system; for, as is known to be the case, such lower centres are capable of effective reaction apart from the cerebral influence. The facts, as presented to us by a study of the evolution of the central nervous system, would lead us to the conception that our physical organism is eminently adapted to react to stimuli derived from environment; and of our physical organism the central nervous system is the more immediate means of effecting such reaction; it is through this nervous system that the outside world is perceived subsequently by the mind. Beyond this we cannot go. A satisfactory reply, as to whether the activities of the lower or subcortical structures are accompanied by subjective expressions in consciousness, can only be given when we have eliminated the cortex in its entirety from its substructures.

At this point we may ask, Does consciousness exist only when the cortex cerebri is functionally active as a whole? or may consciousness exist in conjunction with activities occurring in one or several regions, while other regions are inactive as concomitants of mental phenomena? the answer is readily conceivable, and it is fair to assume that full knowledge is dependent

[1] Waller, *Brain*, 1892, p. 355.
[2] Hyslop, *Mental Physiology*, p. 138.

upon complete seriality of presentation and representation derived from activities within the *whole* of the cortex, and possibly incomplete knowledge or partial consciousness may be derived from a disruption of that seriality or sequence through inactivity of *part* of the cortex. The phenomena of double consciousness and the various epileptiform states would appear to warrant this assumption, and by its aid some of the greatest difficulties of our subject may be unravelled.

Before discussing this question more fully, however, it is advisable that we should again refer to Hughlings Jackson's scheme of evolution. We have in this scheme (1) a level for representations, (2) a level for re-representations, and (3) another for re-re-representations. The difficulty of determining what areas of the cortex are concerned with the manifestations of subjectivity, while unsurmountable on the assumption that any areas are concerned more directly than others, disappears with the hypothesis that all areas are concerned directly and indirectly, and that subjectivity is merely in its *completeness* dependent upon a complete psychical seriality and sequence, and that this completeness of seriality is dependent upon a complete functioning of the whole cortex.

Were the præfrontal lobes the physical substrata for the reception of re-re-representations, these lobes would be essential for the proper correlation of modes of matter to modes of mind. We have no proof, however, that any part of the human cortex is indispensable to the manifestations of subjectivity. The contents of consciousness—previously acquired, and serving as the mental data upon which the ego preserves its continuity—may be rendered temporarily inert by interference with the modes of matter concerned with the local memories (*e.g.* word-deafness, etc.), but this is all. No amount of philosophy or argument will explain the evolution of a subject from an object. Neither will it prove the nature of that subject by the demonstration of the sources from which it receives its objective supplies, or data in consciousness.

We have, therefore, ample experimental and pathological evidences that the mind can see, feel, and will, in spite of the physical and structural discontinuities of the brain cortex. Destruction of any brain-areas is, so far as our knowledge goes at present, not necessarily attended by alteration in the subjectivity of the mind. We may destroy, extirpate, or sever the connection of any of the cortical areas, and we can thereby cut off present supplies, or even render the results of former activities inert (memory); beyond this, however, we cannot go.

There is no way of evading the difficulty of adapting the subjectivity of the mind to anatomico-physiological data. When we advance in our knowledge of the data of consciousness (which are much the same now as they were) and the relations of the various structures of the brain, we may be able to formulate some more definite doctrines as to cerebral localisation. In the meantime, however, we can offer no ultimate solution of the one great difficulty. If we localise determinate activities within restricted areas, we become responsible for an account of some supreme site, where the mental correlates of these activities are viewed by the subject. Or if, on the other hand, we are indifferentists, we find ourselves confronted with the problem of having to account for the mode by which the subject obtains its view of the objects correlated with physiological activities in widely apart localities. It is needless to say, that whichever view we take, our difficulty will be reaching the truth so far as the subjectivity is concerned.

The answer of cerebral anatomy and physiology to the question of localisation of consciousness has, therefore, been of little value hitherto, and we are as yet unable to account for the phenomena of mind by the study of material structures. The mere fact that the formerly experienced contents of consciousness are rendered inert or incapable of being revived (as in cases of word-blindness and word-deafness, etc.) proves nothing, inasmuch as we are unable to prove whether the contents themselves are destroyed or only rendered inert through lesions of the tracts through which their causal or effectual activities have been or should be transmitted.

The weighty and philosophical arguments of Hughlings Jackson, that, from an evolutionary standpoint, each and every part of the body must be represented in any one unit of the cerebral cortex, would appear to bear out the contention that consciousness or knowledge is not dependent upon one special area, but upon the integrity and active co-operation of all.

Amœboidism of Nerve Cells.—Without entering upon a discussion as to the value of all the ingenious and suggestive views concerning the amœboid movements of nerve-cells, it will simplify the subject if these views are here briefly referred to.

The *neuron theory* holds that every nerve-cell with its protoplasmic proceeds, axis-cylinder process, and collaterals, is an anatomical unit, structurally independent of other nerve-cells. The researches of Cajal, Kolliker, Duval, etc., lead one to adopt the view of the contiguity of the ramifications of the neurons, as against the view of the continuity entertained by Gerlach, but it is natural to inquire whether these protoplasmic ramifications are susceptible of approaching to, or receding from one another, by virtue of their contractile properties. In 1890 Rückhardt suggested that these movements might account for the differences in the functional

states of nervous areas, and in 1894 Lepine suggested that possibly sleep (*vide* article "SLEEP, NORMAL AND MORBID) might be due to the retraction of cellular prolongations leading to their isolation from one another.

In 1895 Duval argued that as imaginations, memory, and association of ideas become more active under the influence of certain agents (tea, coffee), whose functions would be to excite amœboid movements in the contiguous extremities of nerve-cells, it is therefore conceivable that the ramifications do approach each other and thereby facilitate the passage of impulses. The numerous experimental researches on fatigued animals appear to show that fatigue of the nervous elements brings about the isolation by retraction of the cellular prolongations.

The earliest anatomical researches upon the amœboid movements of cells were carried on upon retinal and olfactory cells, and by analogy the theory of nervous amœboidism holds good. The theory of *nervi-nervorum*, advanced to explain how the arborisations can be incited to approach or separate from one another, finds favour with many writers. The theory assumes that centrifugal nerve-fibres preside over the protoplasmic movements of the ramifications of the nerve-cells in an analogous way to vaso-dilatation and vaso-constriction. For the evidences afforded as to the existence of these centrifugal nerve-fibres the reader must refer to the numerous works upon the subject, and he will find an able summary of the subject in Ford Robertson's text-book of *Pathology*, in which the matter is summarised in the statement that the present position of the controversy justifies the conclusion that no discovery that has yet been made really weakens the case for the neuron theory.

UNCONSCIOUSNESS may be *partial* or *complete*. By "partial" is meant a suspension of psychical or physical elements which are necessary to a full or complete consciousness. By "complete" is meant the seriality of thought and discrimination (or "*knowledge*") which is psychically available by reason of a concomitant structural contiguity of function within the cerebral cortex, whereby the translation of physical events, both external and internal, is fully operative. By fully understanding the meaning of these terms the subsequent attempts to deal with the complicated subject of double consciousness will be more comprehensive.

Partial loss of consciousness is exemplified in those individuals who by reason of disorder or disease suffer from partial amnesia. It is commonly assumed that every recollection has its seat in a definite and determinate portion of the cerebral hemispheres. Each portion has its special function to perform, but is in intimate relation with its fellows. Ribot compares each particular form of memory with a contingent of clerks charged with a special and exclusive service: any one of these departments might be abolished without serious detriment to the rest of the work, and that is what happens in these partial disorders of memory. It may be objected that in these partial amnesias there is not immediate loss of consciousness to present facts, but the answer to such an objection is manifest. Any present data which are incapable of being incorporated with the general stream of consciousness, seriality of thought, or knowledge, by reason of discontiguity or discontinuity of function of the neurons, are outside the limit of "knowledge," and the reflex, if there be one, is the result of activities within the undamaged cerebral remainder, and therefore only incomplete, and oftentimes merely automatic. The writer has elsewhere divided the forms of partial amnesia into two groups, viz. (*Mental Physiology*, p. 361):—

(1) Those forms in which there is a loss of memory of a series of mental states, without any obvious interference with the activities of the mind as a whole; (2) those forms in which the loss is due to disease or injury of certain nerve-elements. Among the insane one meets with nearly every form of partial amnesia, and in general paralysis more especially do we find partial loss of consciousness of a class of events derived through the medium of the special senses. In idiots or imbeciles congenital defects of consciousness are common. The failure to register or interpret impressions through one or several of the special senses results in a permanent defect of consciousness and consequent failure in intellectual development. Before discussing the various states of complete loss of consciousness or coma reference must be made to the various grades of so-called mental automatism, or double consciousness.

Double Consciousness. — This condition has been compared to dream, somnambulistic, epileptic, amnesic, and insane states, and the writer has elsewhere cited instances of the various gradations between them and health ("Double Consciousness," *Brit. Med. Journ.*, Sept. 23, 1898). In childhood night terrors or somnambulism may precede a condition in which during the daytime there are moral or volitional perversions, of which in the fully awake state there remains no memory or power to recall. These children usually belong to the group of unstable neurotics who may become criminal or insane according to surroundings. The writer finds much to be said in favour of the hypothesis that dual consciousness is only complete somnambulism. The successive awakening of the senses constitutes a gradation from ordinary sleep to complete somnambulism, which give to the individual the appearance of leading a dual life. Questions of criminal responsibility arise in those cases in which criminal acts are committed in the hypnagogic or in the somnambulistic state.

We may assume that the sleep, though apparently profound, has really only suspended some of the highest level functions, leaving uncontrolled play to the lower levels. During the fully awake state the highest level has little or no knowledge of what happened when only the lower levels were at work.

Sometimes, in cases of exhaustion, deep sleep is followed by a partial awakening attended by confusion and pranks of the senses, and later in the day complete awakening. In other types abnormal or automatic states are preceded by profound sleep, the normal state only being reached again after prolonged sleep. After any injury or shock the power of recall of mnemonic images may be inhibited, and recent or remote events may, for the time being, be quite obliterated.

This inhibitory process may extend both backward and forward, and there may, or there may not, be recovery from the loss. It is assumed that either the registration of anterior states is interfered with or effaced; or if persisting, their power of revivification by association with the present is destroyed. In organic brain disease the former is probably the case, whereas in most of the known cases of double consciousness the latter is the probable explanation, and the coupling apparatus between the past and present is rendered inert by some defect in its organisation.

Numerous cases are recorded in which—through injury or shock—a blow on the head, a fall, a fever, or an acute illness may produce effacement from the memory of events of either recent or remote occurrence. Owing to traumatism extensive periods of time may be obliterated. External violence, causing concussion or injury to the brain structures with loss of consciousness, will require great care in the formation of a diagnosis. The fact of the external injury is usually obvious, and the immediate unconsciousness may be due to shock or actual damage to the skull, membranes, or brain substance. The writer has seen several cases in which not only has unconsciousness immediately followed the injury, but at a subsequent period, months or even years later, curious anomalies of unconsciousness have occurred. These abnormal states may be likened to double consciousness, inasmuch as the patient may for weeks or even months respond to external impressions in a dazed, confused manner, and be apparently in a degree conscious. On awakening, however, to full consciousness there is more or less complete failure to recall the details of the partially conscious period. The failure to recall events may be complete for several days, but it is not common in such cases to have complete failure in regaining their knowledge.

When dealing with loss of consciousness due to injury, it is important to note that trivial accidents may cause lacerations of the brain substance or even extensive meningeal hæmorrhages. On the other hand, the injury may have occurred in consequence of some form of cerebral seizure. In an epileptic attack the period of unconsciousness is usually of short duration; and the presence of signs of paralysis, unilateral rigidities or clonic spasms, inequalities of the pupils, or conjugate deviation of the head and eyes, will help to indicate the existence of some organic mischief. The three forms of epilepsy—viz., *grand mal*, *petit mal*, and *epileptic vertigo*—are regarded as but different degrees of the same morbid state, and the types are so common and so well known that it is not necessary to do more than merely refer to them. The coma following epilepsy has in itself nothing distinctive.

Note, however, may be made that many of the hallucinatory auræ preceding the actual attack, also the automatic states either preceding or following the fit, are evidences of partial unconsciousness as compared with the more profound coma.

The next type of unconsciousness is seen in the insane, and as the writer has pointed out ("Double Consciousness," *Brit. Med. Journ.* Sept. 23, 1899), there are two main groups according as the condition is complete or incomplete. In complete alternation the personality of the individual is entirely different in the two states: there is no continuity of thought, and the memory of one state is absent during the occurrence of the other. In the "partial" types the variations may depend upon sensory experiences which are different to those habitual to the normal self, and there may be new bodily sensations, or loss of old ones, and there may be illusions of identity of part or of the whole of the individual.

The *anæsthetic hysterical types* are those in which the symptoms are mainly hysterical with anæsthesia and temporary amnesia. In these cases there are certain "inhibitions" (Janet) or "negative lesions" (Hughlings Jackson) which prevent the recall or revival of the former sensations. Thus, the occurrence of anæsthesia or akinæsthesia may be attended and followed by an amnesia which forms the basis of a change in personality. Another type in which the individual assumes the possession of a mediumship and acts as if animated by others, living or dead, is fairly common. Automatic writing, inspirational speaking, trance utterances, etc., fall into this category.

Psychologically considered it appears justifiable to conclude with regard to all these conditions of partial consciousness that in the abnormal states the permanent group or mass of individual feelings is only in part or slightly subject to the power of volition. There are wanting those conscious accompaniments which go to raise consciousness into self-consciousness, and the disjoining of these bodily feelings and

images which go to make up the *ego* removes the background of the awareness of self. The condition, therefore, is one of "sciousness" (James) pure and simple, inasmuch as it does not include those elements or adjustments which go to make up the complete self and which constitute *con*-sciousness.

Physiologically considered it appears fair to assume that consciousness has a diffuse substratum, and the physical equivalents of the elements of consciousness are everywhere throughout the brain substratum. Each unit of the highest evolved parts of the sensorium has its own physical equivalent to consciousness, which exhibits itself under certain physical conditions occurring within that unit. The succession of physical elements constituting seriality of thought, conception, knowledge, is correlative to continuity of action between the units of the cortical substance, and the destruction or inertness of any of the physical correlatives of this seriality appears to be the explanation of these freaks of dual and even multiple personalities.

It now remains to consider those conditions of unconsciousness in some instances termed "coma," and in which the loss of *consciousness is complete.*

The following tabular statement will serve as a guide:—

- Unconsciousness due to
  - Convulsions.
  - Epilepsy.
  - Traumatism (due to injury, sunstroke, etc.).
  - Organic brain disease
    - Meningitis.
    - Hæmorrhage.
    - Embolism.
    - Thrombosis.
    - Tumours, abscesses, etc.
    - General paralysis.
    - Other paralyses.
  - Toxins
    - Drugs.
    - Autotoxines.
  - Artificial
    - Hypnotism.
    - Anæsthesia (chloroform, etc.).

Convulsions occurring in infancy may be caused by fear, anger, improper diet, hyperpyrexia, dentition, entozoa, rickets, cerebral or bodily irritation. Sometimes the seizures are difficult to distinguish from epilepsy, and not infrequently epilepsy may supervene. The convulsions may pass off without doing injury to the cerebral structures, or cerebral congestion or asphyxia may be brought about with serious sequelæ. The attack usually begins with tonic contraction and palmar flexion of the thumbs, succeeded by tonic spasms of particular muscles or group of muscles of the face, arms, legs, or of one side of the body, and sometimes leaving motor impairment or even paralysis.

Consciousness is usually lost, respiration irregular, and face livid. The tonic spasms may simulate the clonic contractions of epilepsy. Treatment resolves itself into removal of the cause of reflex irritation, suitable diet, and removal from unhygienic surroundings.

In adults the convulsions may be *tonic*, affecting any muscles or set of muscles, *static* or convulsive auræ, *rotatory* with general action of the extensor muscles, *unilateral* affecting one side of the body, *epileptiform* simulating epilepsy, *apoplectiform* simulating apoplexy, and lastly *epileptic* or *apoplectic.*

Where certain muscles or sets of muscles are alone affected, consciousness may be retained or be slightly affected. In the other types, however, it is usually completely suspended.

Epileptic coma usually accompanies sensori-motor discharging lesions of the nervous system (*vide* epilepsy) and the symptoms will vary according to the site affected—cerebrum, cerebellum, or spinal cord—and according as the lesion is discharging or destructive in nature. There is no sudden line of demarcation between the minor forms of epilepsy, in which the mental or motor functions are but partially affected, and the graver forms attended by total loss of consciousness and general clonic convulsions.

Briefly enumerated, the various relations of epilepsy to loss of consciousness are:—*Abortive*, with loss of consciousness; *feigned*, in which there is no biting of the tongue, no relaxation of the sphincters, no pupillar anomalies, and none of the post-epileptic phenomena; *gastric*, *intestinal*, *laryngeal*, *renal*, *ovarian*, and *auditory*, due to reflex irritation; *hysterical* (*vide* hystero-epilepsy); *partial*, limited to one muscle or set of muscles; *cerebral*, *spinal*, according to the region mainly affected; *Jacksonian*, in which the spasm is in a definite direction: *idiopathic*, without observable lesion; *vaso-motor*, with loss of consciousness, but without muscular spasm; *thalamic*, supposed to be due to a lesion of the optic thalamus with a special sense auræ, and loss of consciousness without muscular spasm; *psychic* or *masked*, with partial or complete loss of consciousness without muscular spasm; *trau-*

matic, *syphilitic*, *malarial*, *toxic*, etc., due to these factors respectively.

For simplicity these types may be tabulated as follows:—

- 1. Without loss of consciousness, but with spasm
  - Abortive.
  - Feigned.
  - Hysterical.
  - Partial.
- 2. With loss of consciousness, but no spasm
  - Psychic, affecting one or several senses.
  - Masked, or mental automatism.
  - Vaso-motor.
  - Thalamic.
- 3. Loss of consciousness and motor spasm
  - Idiopathic, without known lesion.
  - Jacksonian, with local lesion.
  - Traumatic, organic lesion usually.
  - Reflex
    - Gastro-intestinal.
    - Renal.
    - Ovarian.
    - Laryngeal and auditory.
  - Hystero-epilepsy.
  - Organic disease
    - Cerebral.
    - Cerebellar.
    - Spinal.
  - Toxic
    - Syphilis.
    - Malaria.
    - Fevers, etc.
    - Drugs.

Concussion and Compression of the brain require but brief notice here, as the subject is so fully dealt with in works on Surgery. Concussion includes all those instances in which there is unconsciousness after injury (to a greater or less extent), unaccompanied by paralysis; compression includes all those in which there is paralysis. In concussion the unconsciousness is attended by pale face and cold skin; the pulse is weak, often irregular or imperceptible; the pupils vary, but usually respond to light; breathing is shallow; urine and fæces pass involuntarily, but there is no paralysis of the sphincters nor any other symptoms of paralysis; the temperature is usually lowered. After the stage of collapse the patient can usually be roused. There is, however, usually some headache and drowsiness which may last some time.

Compression is attended by definite evidence of paralysis—stertorous, oppressed and slow breathing; dilation of pupils, with occasionally insensibility to light; slow pulse, relaxation of the sphincters, coma, and paralysis.

Other cases, however, occur in which the unconsciousness is by no means complete, and there may be delirium with or without spasms or convulsions.

Where the insensibility and paralysis increase in severity and extend to the functions of deglutition and respiration, a fatal result is apt to occur.

Sunstroke, when due either to the direct rays of the sun (*coup de soleil*), or indirectly to heat or other influences (*coup de chaleur*), is attended by loss of consciousness. Usually the fatal character of the heat is derived from aerial moisture. The bodily causes arising in connection with faulty habits or diseases of the individual are of special importance, and among these may be mentioned fatigue, over-exertion, overcrowding, bad ventilation, unsuitable dress, retained excretions, defective secretions, in fact, anything tending to debilitate or contaminate the system. Injuries to the head, malaria, scorbutic taint, venereal disease, former attacks of insolation, and alcoholism, all predispose to its occurrence.

Malaria, syphilis, and alcoholism not only play an important part in the etiology of insolation, but they also tend to modify and influence the subsequent history of the individual, and give rise to symptoms of great variety and complexity. The writer found that out of fifty-five cases of affections of consciousness following sunstrokes eight had malaria, five syphilis, and seven had been alcoholic ("Sunstroke," *Dict. Psych. Med.*). In addition to the actual loss of consciousness consequent upon the sunstroke, there are apt to be symptoms of varying degrees of severity due to impaired functional energy of the cerebro-spinal system, and this impairment shows itself either in motor paralysis, sensory paralysis, either of common or special sensation, hyper- and dysæsthesiæ of the nerves of common and special sense, in debility and undue excitability of emotional centres, and in similar states of the cerebral hemispheres and spinal cord. The mental sequelæ are of extreme interest, and the unconsciousness is often attended with meningitis or cerebral damage which may destroy the life or intellect sooner or later, or

permanently compromise the whole health or that of some important function.

In many cases the sequelæ may be attributed to the injury the brain suffers during the primary attack, probably from loss of nutrition in syncopal, and from congestion in the apoplectic forms. Of the syncopal, asphyxial, and hyperpyrexial forms of insolation, the two latter appear to be the most important and dangerous, and are more liable to be attended with sequelæ of a degenerative type. In children idiocy and imbecility may result from sunstroke. It must be remembered, however, that parents are ever ready to attribute the idiocy of their children to an accidental fall or blow, so that in any case of idiocy or imbecility attributed to the effects of sunstroke, the existence of an hereditary neurosis, the occurrence of fits, or other diseases, as well as the nature, extent, and immediate consequences of the attack, ought in great measure to be considered before making a diagnosis.

Sometimes mental symptoms, such as delirium or excitement with hallucinations, are found intercurrent with the stupor and coma following the shock, and the condition may gradually become very similar to that of primary dementia. As a general rule, however, although there may be some trace left of the primary injury to the brain, the prognosis is more favourable in such cases than when the psychosis develops some months, or even years after the injury.

The possession of an unstable nervous system is attended by a peculiar liability to easily succumb to the effects of heat. The writer has seen several cases of syncopal attacks, and even of an asphyxial character, occurring in imbeciles after exposure to a hot sun for a few hours, and their exceeding proneness to be affected in this manner renders it necessary to exercise care that they are not exposed unduly during the hot months. Epilepsy is one of the most common of the sequelæ of insolation, and occurs in various degrees of severity, from slight epileptiform convulsions to the severest forms of the disease. Sometimes the epileptiform or apoplectiform seizures of general paralysis of the insane have been mistaken for sunstroke. The frequent occurrence of epilepsy is suggestive, and, as in the cases of periodical psychoses already mentioned, the disorder appears to be a manifestation of an unstable vaso-motor state.

Both idiocy and imbecility may be dependent upon early epilepsy, but the absence of spastic symptoms, contractures, and other deformities, together with the absence of progressive deterioration associated with the convulsions, are characteristic of an acquired rather than a hereditary state; and in epilepsy following upon sunstroke, the mental defects and convulsions are collateral phenomena, both depending upon the same cause, whilst the positive signs of alienism, such as anomalies of character and moral perversions, with defective or one-sided development of special faculties, all point to an acquired psychosis, in a great measure differing from the progressive deterioration of ordinary idiopathic or hereditary epilepsy.

In adults episodical attacks somewhat analogous to epilepsy occur; thus there may be a periodical attack of depression, maniacal delirium, or even conditions closely resembling the maniacal, epileptiform seizures of paretic dementia.[1]

Meningitis is sometimes ushered in by vertigo, or convulsions, or epileptic seizures. In the later stages the occurrence of coma is very frequent. The symptoms are so well known that they need be but briefly referred to here. In the early stages hyperæsthesia, tremors, photophobia, illusions, and in children the characteristic hydrocephalic cry, are generally present. Later these symptoms are followed by drowsiness and apathy, or there may be delirium or paralysis of various forms.

In the third stage there is anæsthesia, and coma may supervene. Paralysis becomes more pronounced, and convulsions increase in frequency and severity as the fatal end approaches. Sometimes the patient is able to rouse himself from the coma for a few days, but almost invariably a relapse occurs. Drowsiness, coma, and collapse may be the only indications of what proves to be extensive meningitis. The symptoms are sometimes very similar to delirium tremens. The diagnosis of a tubercular origin must depend upon careful attention to the history of the patient. Profound coma is more liable to occur when the lateral ventricles are distended with fluid, and their parietes softened and compressed.

Apoplexy, with effusion of blood between the cranium and dura mater, or from the subarachnoid tissue, or into the substance of the brain, may be due to lacerations from violence or rupture of diseased vessels. The attack usually comes on suddenly and unexpectedly, and there may be premonitory symptoms of local disturbances of the cerebral circulation. Of these symptoms the most important are numbness or tingling, loss of power, diplopia, and signs of paralysis. These symptoms, however, may be purely functional, or indicative of the presence of a tumour or other circumscribed lesion. When actual effusion of blood has taken place the patient becomes aware of pain in the head and sudden giddiness followed by paralysis, or there may be syncope passing into coma and paralysis when the hæmorrhage begins in the external capsule (Broadbent). Sometimes the attack is ushered in by convulsions followed by coma. The symptoms vary in severity very considerably according to the extent, nature, and locality of the lesion. When coma supervenes the patient is sometimes capable of being

[1] "Lectures on the Diagnosis of Insanity," by Theo. B. Hyslop, *Med. Press*, February 1896.

roused, but soon he becomes utterly unconscious. The pupils may be natural, irregular, and insensitive to light, contracted when the hæmorrhage is into the pons, or dilated especially towards the fatal end.

The occurrence of coma implies either a large effusion of blood or effusion into some vital part. Effusion into the pons is usually attended by convulsions and profound coma and paralysis, especially if the hæmorrhage is great in extent. Hæmorrhage into the cerebellum is attended by severe occipital pain, vomiting, etc., and giddiness rather than actual loss of consciousness.

Thrombosis and embolism, causing obstruction of one of the cerebral arteries, is attended by symptoms closely resembling apoplexy, and, as in apoplexy, the characters of the symptoms vary according to the size and distribution of the vessel affected. In embolism the obstruction almost invariably occurs in the middle cerebral artery, or one of its branches, and is attended by more or less complete hemiplegia, etc. The prognosis is usually unfavourable, and seldom are the effects of thrombotic or embolic softening completely recovered from. When consciousness is interfered with the intellect is apt to fail until dementia supervenes. Coma is more strikingly characteristic of hæmorrhage than plugging.

Tumours in the brain are usually attended by vertigo, headache, vomiting, paralysis, convulsions, and spasms, and various intellectual and emotional disorders. The convulsions may be epileptiform, but, unlike true epilepsy, unattended with loss of consciousness, or later unconsciousness may come on. Sometimes the earliest indications of cerebral tumour may be momentary attacks of incoherence, aphasia, or loss of consciousness, or attacks stimulating hysterical fits or apoplectic seizures. Failure of memory and mental hebetude go on to fatuity and dementia. The diagnosis of the site of a tumour must depend upon anatomical and physiological considerations, and the pathological nature of the growth upon other considerations, such as arise in connection with tubercle, syphilis, aneurysms, malignant growths, and hydatids.

Functional disturbances of the brain may be evidenced by drowsiness, epileptiform or apoplectiform seizures, or coma. The modes of onset differ somewhat from those due to effusion of blood, in that the paralysis is more general and attended with flaccidity, acceleration of the pulse, and rise in temperature.

These comatose attacks occur in association with lateral and disseminated sclerosis, occasionally in children, and simulating meningitis, and especially in general paralysis of the insane.

The congestive seizures occurring in general paralysis of the insane may be epileptiform or apoplectiform in character. Epileptiform seizures vary in severity from *petit mal* to *epilepsia gravior*. They may occur at any stage of the disease, but more commonly they occur when the disease is advanced. A succession of seizures results in the *status epilepticus* well known to all asylum physicians. Sometimes with even severe epileptiform seizures consciousness is retained. The number of seizures may amount to several hundreds daily.

Quasi-syncopal attacks are also not uncommon. Apoplectiform seizures may occur with or without convulsions; they are less frequent than the epileptiform, and may or may not be attended by temporary paralysis, of which the most common is a partial or incomplete hemiplegia. Various simple paralytic seizures also occur, but almost invariably the paralysis clears up and vanishes in a few days.[1]

Auto-intoxications[2] may occur from suppression or disturbance of the functions of an organ. Affections of the various glands, with or without pathological lesions, may give rise to symptoms which are attended by or culminate in loss of consciousness. Myxœdema, cachexia strumipriva, diabetes, acute yellow atrophy of the liver, and Addison's disease, may be productive of attacks varying from partial loss of consciousness to complete coma. Gout and oxaluria, etc., depend upon absorption of faulty products of metabolism, and may be attended by affections of consciousness. In carbonic acid poisoning, uræmia, and eclampsia gravidarum, the products of metabolism are apt to be retained in the different organs. Huppert arrived at the conclusion that albumin appears in the urine after every well-marked fit of epilepsy, and the more severe the fit the more abundant the albumin. Mere cases of epileptic vertigo may be quite unattended by this phenomenon unless the attacks follow one another rapidly. Albuminuria is more common in paralytic insanity than in any other cerebral disease. De Will found albuminuria after the convulsions of general paralysis, while Konig, Richter, and Mendel, on the other hand, usually found it absent. The writer[3] has elsewhere stated that renal disease is associated with affections of consciousness in two ways: (1) acute transient delirious mania, an acute toxæmia, or uræmic insanity; and (2) a progressive cerebral degeneration, with chronic renal disease as the primary cause. In this latter type the mental symptoms during the earlier stages vary from a mild dementia to mania or delirium. In due course, however, complete dementia, not unlike that of general paralysis, results. The writer has seen many cases of mania with excitement and even delirium, in which the pupils were contracted, reflexes diminished, muscular tremors, and convulsive attacks with loss of consciousness, due

[1] See "General Paralysis."
[2] Van Giesen, *States Hospital Bulletin*, vol. i.
[3] "On the Mental Conditions associated with Bright's Disease Uræmia," *Practitioner*, November 1901.

to an over-production of toxic substances in the body and eliminated in the urine. The dyspnœic and gastro-intestinal forms of uræmia are sometimes seen in the insane, but it is with the comatose and convulsive types that asylum physicians have mostly to do. Other auto-intoxications, due to the over-production of physiological and pathological products of the organism, are (Van Giesen) hydrothionæmia, ammoniæmia, acetonuria, diaceturia, cystinuria, etc., also diabetic coma, coma carcinomatosum, etc., and Basedow's disease.[1] Malaria is sometimes attended with or followed by extreme collapse, coma, or delirium, epileptiform or tetanoid convulsions, or various mental symptoms. Simple uncomplicated attacks of malaria are rarely followed by such affections; but when the nervous system has been weakened by syphilis, alcohol, and various excesses, not only is some neurosis likely to supervene, but it is likely to be of a serious, intractable nature. Simple cases, where no other factor than malaria has been present, generally recover.[2] (For an account of the various alterations of consciousness occurring in association with or following various fevers and bodily diseases, the reader is referred to the article on "Post-Febrile Insanity" by the writer, in Tuke's *Dictionary of Psychological Medicine.*)

Drugs are productive of loss of consciousness and severe or fatal coma. Of these drugs the following must be referred to briefly:—

Alcohol may cause convulsions, epileptiform or apoplectiform seizures with coma. In mania due to alcohol, convulsions may be so severe as to lead to a fatal result from syncope or coma. The occurrence of these seizures, together with inequalities of the pupils and various indications of paralysis, have frequently led to a faulty diagnosis of general paralysis. The apoplectiform, or more particularly the epileptiform attacks, differ in certain respects from those occurring in general paralysis, and the pseudo-general paralysis due to alcohol generally ends in recovery.

Lead poisoning is also sometimes attended by not only a progressive physical and mental decay, but also by acute delirium or convulsions which may prove fatal. The various epileptic or eclamptic disorders due to lead are apt to be followed by dementia and symptoms which have been mistaken for those of general paralysis, and to these cases the term saturnine pseudo-general paralysis has been applied. When the cases progress unfavourably the end is usually due to convulsions and coma. Other intoxications due to morphia, cocaine, absinthe, ether, chloral, haschisch, tea, nicotine, oxide of carbon, are commonly met with, and may be characterised by vertigo, syncope, delirium, epileptiform or apoplectiform seizures, or other derangement of consciousness.

Lastly, unconsciousness may be induced artificially by hypnotism (*see* article "HYPNOTISM"), or by anæsthetics.

[1] See also article "Urine," Tuke's *Dictionary of Psychological Medicine*, by Blyth and Hyslop.

[2] Hyslop, "Malaria," *Dict. Psych. Med.*, p. 756.

Uncontrollable.—Not to be restrained, *e.g.* the uncontrollable impulses towards suicide, drinking, and lust seen in cases of insanity; also, the grave forms of disease, such as the uncontrollable vomiting of pregnancy or hyperemesis. *See* PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Digestive System, Uncontrollable vomiting*).

Uncus.—A hook or hook-like process. *See* UNCIFORM; UNCINATE.

Underhung Jaw.—A lower jaw which projects beyond the upper jaw.

Underpinning. *See* UMBILICUS, DISEASES OF (*Hæmorrhage, Treatment by Underpinning*).

Undue Influence. *See* LUNACY (*Will-making*).

Undulant Fever.

HISTORY AND GEOGRAPHICAL DISTRIBUTION	232
ETIOLOGY	232
PATHOLOGY	233
PROGNOSIS	234
DIAGNOSIS	234
SYMPTOMS	234
TREATMENT	237

See also MALARIA (*Diagnosis*); TROPICS, UNCLASSED FEVERS OF (*Non-Malarial "Remittent" Fever*).

SYNONYMS.—Mediterranean, Malta, rock, Cyprus, Neapolitan, Cretan, etc., fever, from its geographical distribution; intermittent typhoid, recurrent typhoid, typho-malarial, etc., from its resemblance to typhoid fever; gastric remittent, bilious remittent, or simple remittent fever, from its remittent character; simple continued fever from its lengthened course and want of definite symptoms.

Definition.—A pyrexial disease, endemic in certain localities, having a long and indefinite duration, with a tendency to undulatory waves of temperature.

HISTORY.—There is very little doubt that Hippocrates in his *Epidemics*, about B.C. 450, described this disease. The next accounts of this fever that are in any way reliable we get from French military surgeons during the French occupation of Malta (1798-1800).

During the earlier part of the nineteenth century we get numerous accounts of this fever by naval and military surgeons serving in the Mediterranean, which are chiefly of interest in showing the extreme difficulties they experienced in classifying the fevers they met with. In the

writings of Sir Wm. Burnet (1810), Henner (1818), Davy (1842), Marston (1860), and Notter (1876), this fever is undoubtedly recognised and described, but it is not until 1879 that we get any stress laid on the fact of this being a separate disease, *sui generis*, when Veale published a paper in the *Army Medical Report* of that year. From 1879 to 1889 nothing of importance was published about this disease, but in the latter year Bruce published an account of this fever in the *British Medical Journal*, which at once brought this disease out of the general confusion surrounding febrile conditions occurring in the Mediterranean, and established it as a separate specific disease.

Geographical Distribution.—The chief objection to the names Mediterranean or Malta fever for this disease (the terms by which it is generally known) is that it undoubtedly occurs in many other places besides Malta and the Mediterranean ports. It is certainly endemic in Malta, Gibraltar, Crete, and Cyprus; probably also in Italy (southern portion), Greece, Turkey, and Sicily. There is now very good evidence that it occurs also in India, North and South Africa, South America, Straits Settlements, and China. The author has no doubt that when this disease is more generally understood it will prove to have a very wide distribution, as he has been shown cases of so-called typho-malarial fever in the Straits Settlements, Borneo, and China which, with his present knowledge of the disease, he would have no hesitation in calling undulant fever.

Etiology.—The discovery by Bruce in 1886 of the micrococcus Melitensis in the spleen of cases suffering from this disease finally established its casual agent. How this organism enters and leaves the human body, or what its habitat outside the human body may be, are up to the present unsettled points.

Theories with Regard to Causation.—(1) The popular theory of the causation of undulant fever in Malta where, it may be mentioned, practically all the research work on this subject has been done, is that it is caused by the insanitary condition of the tideless harbours of that island.

(2) Hughes, in his work on this fever, inclines very strongly to the theory that the disease is caused by an aerial fæcal poison.

(3) The latest and at the present time most popular theory, at any rate in Malta, is that it is caused by the sting of some infected insect, such as the mosquito or sandfly, in exactly the same way as malaria is.

From the evidence the writer has been able to collect during the past three years from naval, military, and civilian sources in Malta, there are insuperable difficulties to acceptation of any of these theories.

The matter is still unsettled, but from the work on this subject that has been done up to the present the writer has come to the following conclusions:—

(*a*) That the disease is air-borne. This is arrived at by a process of exclusion of the other possible sources of infection, such as water, food, bites of insects, etc. (*see* Bacteriology).

(*b*) That it is a very widely diffused organism in the localities where the fever is endemic, *e.g.* Malta. This conclusion is drawn from the fact that it occurs practically all over the island, attacking people living under totally different sanitary and social conditions.

(*c*) That it has to be taken into the healthy individual in a large dose to produce its characteristic effects.

By this is meant that in all probability the dwellers in an area where this fever is endemic inspire in the air they breathe a certain number of the organisms producing the disease, and that the natural bactericidal power of their blood is sufficient to destroy the organisms thus taken into the system, but that granted (1st) a lowered bactericidal power from any cause, such as excessive fatigue, chill, the effects of some other illness, noticeably influenza and pneumonia, etc., or (2nd) that the organisms are taken into the system in sufficient quantities and for a sufficient length of time to overcome the natural bactericidal powers of the blood, then the organism gets the upper hand and finds its way to the spleen, and there is as much difficulty in dislodging it as it has found in arriving at its suitable nidus.

There are many facts that point to the probability of this theory being correct. The fact that the Maltese population (still taking Malta as an example) suffers in much smaller proportion than the European, points to their having a much stronger bactericidal power towards the organism, partly hereditary and partly acquired, the last being probably due to the fact that from earliest childhood they have been taking small doses of the organism into their system, and so increasing their bactericidal power towards it.

It is also a fact that individuals recently arrived into an endemic area are more liable to contract the disease than people who have resided there for some time, showing that a partial immunity is acquired by length of residence in the infected area.

Influence of Age.—Children under four years of age and old people are seldom attacked, but people of all other ages seem equally prone to attack.

Influence of Season.—The fever is usually most prevalent in the months of May, June, July, and August, the period of lowest rainfall, and what the writer would lay stress on, the period of greatest variability in the direction of wind with frequent dust storms.

Incubation Period.—The exact incubation period of a disease usually starting with very

indefinite symptoms is difficult to settle, but the evidence collected on this subject points to the fact that from ten to sixteen days is the usual incubation period. In the cases of accidental inoculation with the mic. Melitensis, of which several have now occurred, the incubation period has varied from five to fifteen days.

Not infectious.—In spite of the popularity of the recent mosquito theory of its origin, the writer is convinced that it is not directly communicable from the sick to the healthy.

Immunity.—One attack usually confers immunity, just as is the case with most specific diseases. There has been much controversy on this subject, but the evidence obtainable since more accurate diagnosis has been established practically puts this fact beyond any doubt.

Prophylaxis.—Avoidance of the infective areas during the months of the year when this disease is most prevalent.

Attendance to the ordinary hygiene of every-day life would prevent many attacks if the writer's theory with regard to the distribution of this organism is correct. The histories of such a large proportion of these cases give some hygienic indiscretion prior to the attack, the commonest being (*a*) getting a chill after taking violent exercise; (*b*) sleeping in cotton garments, which become saturated with perspiration; (*c*) being out in a hot sun all day, and returning in the cool of the evening without additional wraps; (*d*) over-fatigue, followed by sitting about in cool draughts; (*e*) drill or other hard work on an empty stomach; and many other indiscretions of a like nature.

Better sanitation of most of these localities is urgently required, as tending to improve the general health, but people living under what are apparently perfect sanitary conditions are still attacked by this disease.

Pathology.—*Morbid Anatomy.*—The opportunities for working out the pathology of this disease, in spite of its frequency in the endemic areas, are very few. This is owing partly to the fact of its very low mortality.

In the cases that die during an acute attack of fever the only morbid changes noticed were confined to the following organs:—

Heart.—Very pale and flabby. The walls thinned, and showing marked degeneration of the muscle fibre; no endocarditis or pericarditis; in some cases the heart walls become so thinned as to be almost transparent when held up to the light.

Lungs.—Usually a little passive congestion of the bases, secondary to a failing heart.

Liver.—Congested and increased in weight. Average weight 58 oz.

Spleen.—Much enlarged, very soft, and in some cases the spleen pulp quite diffluent, running out like semi-fluid blood when the capsule is cut into. The capsule of the spleen is thickened and tough. Average weight 19 oz.

Brain.—Usually congested, with slight increase of fluid in ventricles, but often quite normal in appearance.

Kidneys.—Sometimes slightly congested, but usually normal in appearance and weight.

Alimentary Canal.—Has always been found quite normal from mouth to anus in the cases examined by the writer.

In cases which die late in the disease, usually from heart-failure or intercurrent diseases, there are the signs of long-continued toxic irritation of the tissues. The liver and spleen, but more especially the latter, are both enlarged, but firm to the touch and with excess of fibrous tissue in them. The heart is usually very pale, with thin walls and often dilatation of the cavities.

Microscopic Changes.—The only changes that have been observed microscopically, apart from those produced by long-continued toxic irritation of the tissues, have been in the spleen, liver, and kidneys. Sections of the spleen show an increase in the lymphoid tissue, with enormous numbers of single micrococci scattered through the section. The liver cells are found to be in a condition of cloudy swelling, and show granular degeneration of their protoplasm. The kidneys show the same changes of granular degeneration of the glomeruli.

Bacteriology.—The micrococcus Melitensis is a minute, oval-shaped coccus, occurring generally singly, sometimes in pairs, but never forming chains. It measures about 33 mm. in diameter (Bruce), and when examined in the hanging drop shows rapid movement. Gordon states that it is provided with from one to four flagellæ, and the character of its circular movements in the hanging drop certainly suggests the possession of flagellæ. It stains well with the more powerful of the aniline dyes, but very rapidly loses its stain. It does not stain with Gram's method.

Growth Characteristics.—It is a very slowly growing organism, usually taking four days to make its appearance on suitable media. The degree of alkalinity of the media employed is a matter of great importance to this organism. It grows best on gelatine, agar, or in bouillon of an alkalinity of -10 (Eyre's method); it can be educated to grow on media of a greater alkalinity, on neutral media, and even on faintly acid media, but the growths are feeble and take many days to appear. Many disappointments will be met with in working with this organism, if this extreme susceptibility to the reaction of media is not constantly borne in mind. The shortest time in which the writer has been able to obtain a growth was forty-six hours, on agar of a very suitable alkalinity inoculated direct from the spleen. In liquid media slight turbidity shows about the fourth day, the medium never becoming more than slightly opaque, as the growth settles to the bottom of the tube in a thin flaky deposit. No pellicle is formed.

On solid media the colonies appear as minute, circular, clear growths, like little drops of sweat coming out on the surface of the medium; in four to five days these drops assume a pale amber colour, and later on become somewhat opaque, though even in quite old growths there is still a considerable amount of translucency. No liquefaction of media or formation of gas occurs.

The optimum temperature for this organism is 37° C. to 38° C., but it will grow well at any temperature between 20° and 40° C. This micrococcus will retain its vitality after a considerable amount of drying.

Prognosis.—The prognosis, as regards life, in undulant fever is extremely good, as the mortality from this disease is under 2 per cent, but the prognosis as to length of attack is very uncertain. It may be taken as a general rule that, in cases commencing with one or two high waves as fever, and generally acute symptoms, with a serum possessing a high agglutinating reaction, the attack will be of much shorter duration than in cases with either the remittent or low type of fever. It must, however, be remembered that a case may suddenly or gradually change its type of fever, and it is quite common to have temperature charts showing all three types of fever at different periods of the same attack.

The tongue is a very good guide as to prognosis, because as long as it remains lightly furred, flabby, and slightly enlarged, the patient is always liable to relapse, even if he has been for two or three weeks with normal temperature; but once the tongue really clears up and assumes its healthy aspect, then it is pretty safe to say that the patient has really got rid of his fever.

Much disappointment is met with in practice from want of attention to this guiding principle, as patients are frequently sent back to their duties, after that their temperature has remained normal for a week or two, as cured, only to return to hospital with relapses in any time from a few days up to three months. In the author's opinion these cases have not got rid of the organism from the spleen, where it remains quiescent until such time as the bactericidal power of the blood is again lowered sufficiently in some way, *e.g.* excessive fatigue, etc., to allow the organism to once more get the upper hand, when the unfortunate patient again has to go through many weeks or months of fever just as if it were a fresh attack.

Diagnosis.—The diagnosis of undulant fever in its early stages, where Widal's test is not available, is an extremely difficult matter. This is owing to the fact that it commences in so many different ways; in some cases continuous high fever (averaging 104°) with intense headache, diarrhœa, distended abdomen, enlarged spleen, and in fact simulating enteric fever in all its signs and symptoms; in others a continuously low temperature, varying from 99° to 100°, often never going above 101°, with practically no other symptoms; in others, again, a high temperature for two or three days, 103° to 105°, with congested tonsils and fauces, furred tongue, and headache, the temperature dropping to 99° or 100° at the end of three or four days and continuing indefinitely at that range.

In its early stages this fever may be mistaken for almost any disease that commences with raised temperature and general malaise; those with which it is most usually confounded being enteric fever, rheumatism, acute tonsillitis, acute tuberculosis, and what, for the want of a more accurate term, is known as simple continued fever, or simple febricula. It is a matter of frequent occurrence to get cases sent into hospital as undulant fever which turn out to be tubercle, subacute rheumatism, deep-seated abscess, malaria, or secondary syphilis.

Latterly the serum test for this fever has been much more widely used in the endemic areas, with the result that there is some accuracy about the statistical returns of the various fevers, whereas before Widal's test came into popular use for this disease the statistical return of fevers was totally unreliable.

The sedimentation or agglutination test with the serum of cases of undulant fever is extremely reliable and satisfactory in every way. The reaction is nearly always obtainable by the fourth day of raised temperature, sometimes earlier and seldom later than the sixth day. It is also obtainable in much bigger dilutions than is the case with B. typhosus.

The writer, after having had very large numbers of these cases under his care, has found that a dilution of 1 in 50 with a time limit of half an hour for the agglutination test is a reliable, easy, and rapid method of applying the test for this disease. In doubtful cases this test should be supplemented with varying dilutions and the sedimentation test.

The serum test is also very useful from a prognostic point of view. Cases with high agglutinating powers usually make a more rapid recovery, especially if the agglutinating reaction gradually increases as the disease progresses; on the other hand, if the agglutinating power is low or gradually diminishes during the course of the disease, it usually means a very long illness, though not necessarily of a severe type. The writer has also observed that with a rising wave of temperature the agglutinating power also rises, and that it generally goes on rising for a day or two after the temperature wave has reached its extreme height and has already begun to come down.

Symptoms.—Undulant fever is a disease remarkable for the fewness of its symptoms, a large proportion of the cases having practically no symptoms beyond the raised temperature,

so that the question of pyrexia in this fever will be dealt with first.

Pyrexia.—The most noticeable points about the temperature charts of these cases are their extreme length, the variety of types they assume, and the tendency underlying the various types to produce undulatory waves of fever.

The duration of the pyrexia, and the various types the chart may assume during the course of the attack, are both quite indefinite, and appear to follow no rule that can be made applicable to even a majority of these cases.

The remittent type of chart is by far the commonest, but there may be a continuously high run of fever, or a long-continued low pyrexia, there may be remissions or there may be none, and the type of pyrexia may vary during the course of the attack, so that all three of these types may be produced in different parts of the same chart.

A glance at the accompanying charts, which are selected from many hundreds of cases, will show much more clearly than any written description what is meant by the different types of pyrexia met with in this disease.

The chart A is typical of the remittent type of fever. This type of the disease usually lasts a very long time, is accompanied by the more severe forms of neuritis, the sweating during the night is excessive, but patients feel comparatively well all day and take food well.

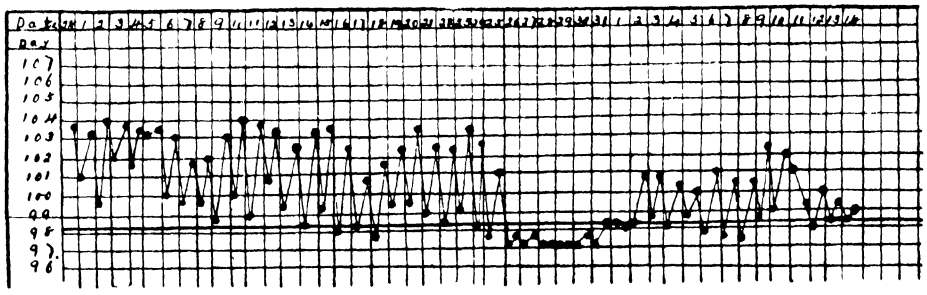


CHART A.

The chart B is typical of the continuously low form of pyrexia. These cases seem quite unaffected by treatment, do not as a rule suffer from neuritis, keep up their weight if generously fed, but seldom get rid of their disease without change of climate.

The chart C illustrates a case, starting with continuously high form of pyrexia, lasting for nearly three weeks, and then taking on the remittent type.

It is in this type of the disease that it is almost impossible, at any rate at the commencement of the illness, to make a diagnosis between undulant and enteric fever, without examination of the serum for Widal's reaction. These are the cases which have diarrhœa, distended abdomen, a large liver and spleen, with foul tongue and delirium. The enteric spots are of no value as a diagnostic help, as many cases of enteric, in tropical and subtropical climates, have no spots, and many cases of undulant fever have spots which may easily be mistaken for enteric spots, both by their appearance and the fact of fresh ones appearing day after day.

The chart D illustrates in a very marked manner the waves of pyrexia from which this disease derives its name. It is not very common to have such definitely marked waves, but this undulant character will be found in every type of the pyrexia of this disease.

Alimentary System.—The fauces and tonsils always show signs of more or less congestion, or even inflammation, in the first few days of the illness, with occasional enlargement and tenderness of cervical glands.

The tongue is furred; in acute cases a thick, dirty fur covering the whole tongue, in mild cases a light fur on the posterior half of the tongue, with the edges and anterior portion moist and clean. The tongue may be denuded of its surface epithelium in the acuter cases, and assume the glazed, raw ham appearance. In the later stages of all types of cases, provided they are under appropriate treatment, the tongue gets an appearance which is very typical of this disease—the whole tongue slightly enlarged and flabby, with tip, edges, and anterior portions moist and clean, but posterior portion lightly furred and moist, with transverse striation. These transverse striæ sometimes go quite deep into the surface epithelium, and look like cracks. There is often slight epigastric tenderness during the first few days of fever, but very rarely any vomiting.

Constipation is the general rule in this fever, though during high waves of temperature it is not uncommon to get diarrhœa with loose yellow motions.

The appetite is poor for the first few days until the intestinal tract has been cleansed with purgatives and intestinal antiseptics, after that it is remarkably good, the patient suffering from this disease being able to digest solid food extremely well, even when his temperature is running fairly high.

The liver and spleen are both always enlarged, especially the spleen. During the height of a wave of fever the spleen is tender, enlarged, and very soft, hardening again as the temperature comes down.

Epistaxis is by no means uncommon in this disease, occurring in 6 per cent of the writer's cases. Nearly always it comes on at the height of a wave of fever, is severe as a rule, and the temperature usually goes steadily down after it.

Tegumentary System.—Sweating is usually

profuse, especially in cases with the remittent type of chart; at the height of a wave the skin remains hot and dry, but when the temperature is falling the patient is bathed in perspiration every night.

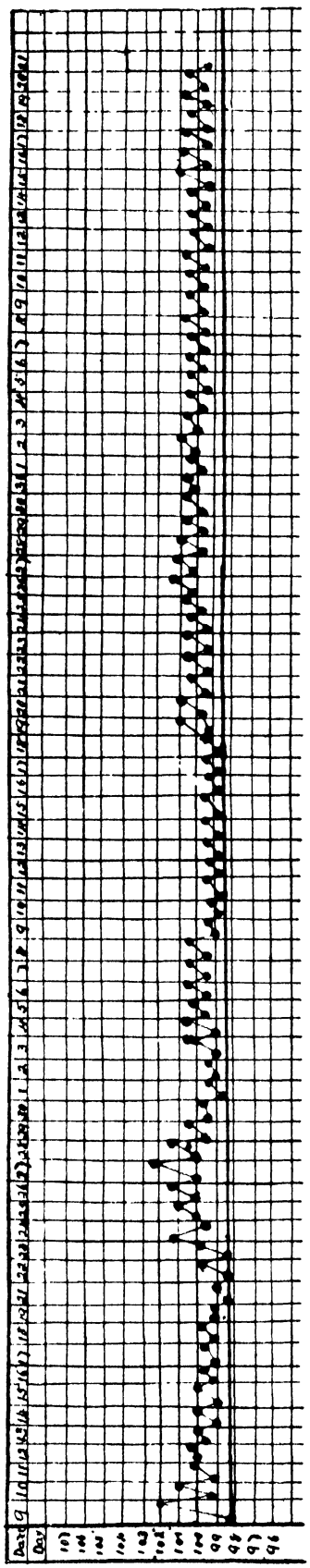


CHART B.

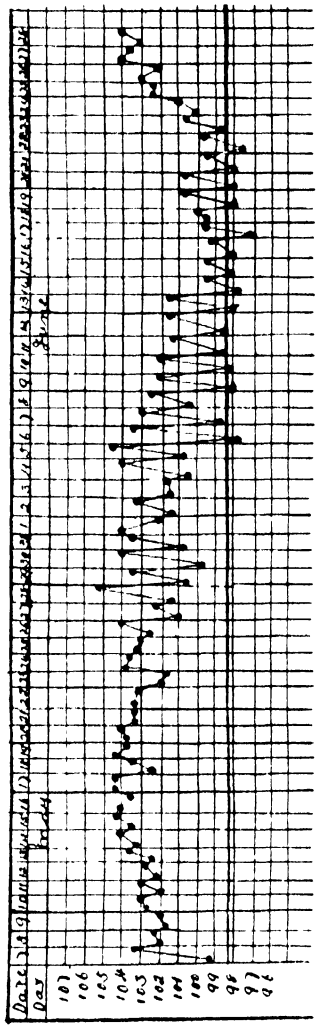


CHART C.

The hair gets dry and brittle, and frequently falls out to a great extent. In cases of long illness the nails also get brittle and break easily.

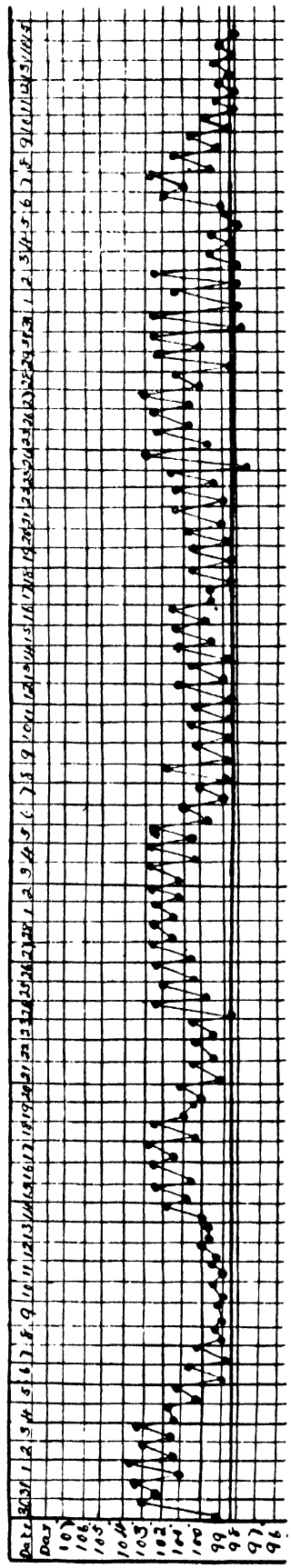


CHART D.

The profound anæmia of the long cases of

undulant fever, though always described, is very rarely seen in patients that have been kept throughout on a generous diet. In the same way subcutaneous hæmorrhage or purpura, though apparently extremely common two or three years ago, are now scarcely ever seen.

Nervous System.—Next to the pyrexia the writer would place affections of the nervous system as the most common symptom of this disease; a neuritis, more or less severe, occurring in quite half the cases. The nerves especially affected are the sciatics, circumflex, and peroneals, named in the order of frequency, although almost any nerve may be attacked. This neuritis comes on quite suddenly, is usually acutely painful, preventing any voluntary movement of the affected limb, and pressure along the course of the affected nerve being almost unbearable.

The acuteness of the symptoms passes in twenty-four to forty-eight hours, but a subacute neuritis of the nerve is usually left, which may continue, varying in severity from day to day, but often lasting, with remissions, throughout the course of the fever, and sometimes for many months after the fever has quite subsided. There is no doubt that the products of this micrococcus seem to have a special and selective action on the central and especially the peripheral nerve tissues, and also probably on the fibrous sheaths of nerves. Their localised and metastatic nature, the fact that they often persist for months after the subsidence of the fever, and the severity of the pains at the back of the neck, in the lumbar region, at the back of the eyeballs, the mental irritability and the wakefulness, all point to this virus having a special action on the cerebro-spinal system.

In severe cases with high temperature an active form of delirium is usually present, and at the end of a long attack of fever there is considerable nervous prostration with impairment of mental capacity. Irritability of the nervous system is also shown by an increase in the reflexes and cutaneous hyperæsthesia over certain spots supplied by specially affected nerves.

The importance and frequency of this neuritis has not been sufficiently noted in the literature of this disease, whereas, on the other hand, much is written about the frequency of arthritic symptoms, Hughes stating that effusion into joints occurs in 40 per cent of the cases.

Arthritic Symptoms.—In the writer's experience effusion into joints is extremely rare. It is common to hear that a patient with this fever is "crippled with rheumatism," but on examination of the case it almost invariably turns out that the patient is crippled with neuritis, there being no effusion into the suspected joint or joints.

Effusion into joints, however, does occur; in the writer's cases in under 1 per cent of the total cases. It usually takes the form of a chronic or subacute effusion which takes a considerable time in getting absorbed, and is quite unaffected by the administration of salicylates. The knee is the joint most commonly affected.

A passive effusion also occasionally occurs into tendon sheaths, the extensor tendons at the wrist and the tendon Achillis being the ones the writer has seen affected.

Circulatory System.—The only special point about the circulatory system in this fever is that the nerves of the heart are affected in the general nerve disturbance, causing an increased rate of heart's action, not at all in proportion to the degree of fever; also palpitation from the most trivial causes is common. A patient who by his temperature chart appears to be having a not at all severe attack of fever, may really be in much greater danger from heart-failure than a case with severe symptoms and high fever. The implication of the nervous vaso-motor system in this disease must always be kept in mind in the treatment of cases. The few deaths the writer has seen from this disease have all been caused directly by heart-failure, and at the post-mortem examination the muscle fibre of the heart has been found thin, pale, greasy, and acutely degenerated.

Urinary and Sexual Systems.—The urine is usually normal, except during periods of high fever, when a trace of albumen may be present. The fact of retention of urine being likely to occur in acute cases must always be kept in mind.

Epididymitis and orchitis are fairly common complications in this disease, not usually of a very acute or painful type, and soon subside with rest and warmth.

Complications. — Hyperpyrexia may occur; it is usually easily controlled with ice-packing, but cases sometimes are very stubborn to any form of external cold application or antipyretic drugs. The best guard against getting hyperpyrexia is to have a routine rule for external application of cold whenever the temperature goes above 103·6 degrees.

Cardiac failure is the great complication to be guarded against in the writer's opinion. It must be remembered we are dealing with a fever of very long duration, and not only that, but a fever in which the nervous apparatus of the heart is often seriously involved, and our treatment must be directed towards keeping the heart muscle in as good condition as possible for the long-continued extra work that is thrown on it.

Pulmonary congestion often occurs, but is secondary to a failing heart. Pleuritic and pericardial effusions are said to occur, but the writer has not met with them.

TREATMENT.—It may be at once stated that no drug has any specific action on the course of this fever, although almost every drug in the

Pharmacopœia, and a good many not in it, have from time to time been vaunted as specifics.

In the treatment of cases of undulant fever there are three main points to be kept in mind, viz. (1) that the patient is suffering from a febrile disease, which is probably going to last for many weeks or even months; (2) that it is of the utmost importance that the patient's temperature should be regulated for him; and, lastly (3) that the heart should be assisted in its work by every possible means, as not only does the poison of this coccus affect the muscle fibre of the heart itself, but it seems to have a specially deleterious influence on the cardiac nerve-supply.

The statement is often made that there is no treatment for undulant fever, and yet there are few diseases in which more can be done to ameliorate the patient's condition, by a careful attention to detail, and by basing the treatment on the three above-mentioned guiding principles.

At the commencement of the attack the patient should be kept in bed, clothed in flannel because of the profuse sweating, and given a purgative. It is usual to prescribe some form of febrifuge mixture or an intestinal antiseptic; the writer has found Burney Yeo's chlorinated quinine mixture the most suitable.

After the first few days it will be seen what sort of a course the temperature is likely to run, and the treatment will vary somewhat accordingly. The medicinal treatment of this disease consists essentially in keeping the intestinal tract clean and in a fit condition to digest and absorb food. Purgatives are required in the majority of cases, and a dose of ordinary sulphate of magnesia mixture taken the first thing every morning is a good routine treatment.

Treatment of the Pyrexia.—This is a point to which special attention should be given all through the disease, and there is no doubt that the most satisfactory results are obtained by lowering temperature by the external application of cold, either sponging with ice-cold water, ice-packing, or ice suspended in buckets on a cradle over the patient's body. The administration of the usual antipyretic drugs—such as phenacetin, antifebrine, antipyrine—should be discouraged as much as possible in this disease, because of their depressant action on the circulation, as, where a patient's temperature is probably going to run up to unsafe limits very many times during perhaps very many weeks, it means very constant dosage with depressants of the circulation.

The external application of cold should be made use of systematically and not spasmodically, as when the patient's temperature has run up to 105° or 106°. The best system is to apply cold whenever a temperature goes above 103·6°, to bring the temperature down to about 101°, and then cease application of cold. The principle underlying this treatment is to prevent as much as possible the temperature getting to the heights where it throws excessive work on the circulatory system. If this system is adhered to throughout an attack of such a prolonged disease, the amount of work thrown upon the various organs of the body, and the amount of cellular wastage and general tissue irritation, must in the aggregate be enormously diminished.

In some cases of high fever the external application of cold will not bring the temperature down, even if continued till the patient's skin is blue and cold. In these cases, before having application to antipyretic drugs, the writer has often found that the administration of a copious enema, a pint and a half to two pints of ice-cold water, will have the desired effect of bringing down the temperature. Failing these measures recourse must be had to drugs, and of these phenacetin in 5-grain doses is by far the most reliable. The writer made many experiments with the various antipyretic drugs in this disease, having the temperatures taken hourly for ten hours after the administration of a dose, and found that phenacetin acted most regularly and constantly, its effect on the temperature usually being perceptible for much longer (averaging seven hours), and its depressant effects on the circulation being less than in the case of other drugs.

The treatment of the neuritis in this disease is on the whole extremely disappointing. When acute, the application of warmth gives the most relief; when subacute or chronic, the administration of salicylate and iodide of potash, small blisters along the course of the affected nerve, inunction with iodide of potash ointment, and a lot of other remedies, appear in some cases to do good, in others to have no effect at all.

For the cardiac weakness and irritability strychnine gives the best results, but should be administered early, and not left till the heart is beginning to show signs of failure. The writer has found that it gives much more satisfactory results if given hypodermically, either once or twice daily for a few days, then omitted for a few days, and so on, according to the condition of the heart, than if taken by the mouth in the usual mixture.

The real treatment of undulant fever consists in keeping the patient in such a condition that he will avoid many of the symptoms of the disease, especially the anæmia, the cardiac weakness, and the neuritis, and this can be done by feeding the patients properly, and so keeping up their strength, weight, and spirits.

Two or three years ago the ordinary, and, in fact, universal dietary for these cases, was a fluid diet consisting of beef-tea and milk. It was supposed that solid food, even in the smallest quantity such as a few grains of rice in the patient's soup, at once made the tem-

perature rise. The only reason why this idea could have lasted so long, was the ever-present fear in the mind of the practitioner that he might be dealing with a case of enteric, as until the Widal test came into general use there was often every excuse for the most accomplished diagnostician being quite unable to decide whether he was dealing with a case of undulant or enteric fever.

Now the effect of keeping cases, with pyrexia, lasting often continuously for months, on a diet of beef-tea and milk only, and constantly in bed, can hardly be imagined by any one who has not happened to see them. The patients are anæmic, emaciated, and listless, taking no interest in whether they get better or not, often with œdema of legs and feet, purpura, and even bed-sores.

The sight of wards full of these miserable objects started the writer on a series of dietary experiments, with the result that the "generous diet" treatment is becoming very generally adopted in Malta, at any rate, by the naval and military medical officers on the station.

It is remarkable how well cases of undulant fever will digest solid food, even with their temperature running fairly high. This is, in fact, one of the diseases where we may trust a good deal to the patient's own feelings in the matter of his dietary, and give him an ample diet when he declares himself equal to taking it. The writer's routine is to keep patients in bed for the first two or three days on fluid diet. Then as soon as the diagnosis is verified, and the tongue has cleaned after the administration of saline purgatives and chlorinated quinine mixture, if the patient's temperature is not going above 103° at night, he is placed on solid food in the shape of eggs, bread and butter, and rice pudding, as well as getting his two or three pints of milk; and again, a few days later, if this diet appears to be agreeing with him, he is given fish in addition, or chicken, or butcher's meat, according to his own taste in the matter. Of course, if the case is a severe one, with a temperature keeping continuously high and a dirty tongue, with probably diarrhœa, he is kept on fluid nourishment till his fever has altered its character and the temperature come down. Stimulants are not usually required in this disease, except when there is a failing heart, when they should be given freely. In the cases, however, which are running an ordinary course, with the temperature even going high, a pint of beer or stout is often useful, given with the solid food, as tending to increase the appetite and general well-being of the patient.

Another important point in the treatment of undulant fever is to decide when a patient should be kept in bed and when allowed up. No general rule can be laid down about this, but the tendency is to keep them confined to bed much too long; in the writer's opinion, patients whose temperatures keep below 102° at night, benefit greatly by being allowed up for the greater part of the day. The great thing a patient must not do is to overtire himself in any way, as this generally means a rise in temperature the same evening; but if that is avoided, the fact of being allowed up and sitting about in the garden seems to have a great effect in cheering the patient up, increasing his appetite, strength, and weight. Each patient's case, however, must be considered separately, both with regard to his getting up and amount of solid food, as some cases with temperatures not running higher than 102° seem to be profoundly affected by the toxins of the virus, whereas others, with temperatures running much higher, seem to suffer very little inconvenience from their disease.

If the treatment described be followed, it will be found that patients seldom become anæmic, the percentage of cases with neuritis is diminished, the body-weight keeps up, and the patient generally is put into a much better condition to withstand all the changes brought about by a long-continued pyrexial disease.

Ungual.—Relating to a nail (Lat. *unguis*, a nail) or having the shape of a nail; *e.g.* the *ungual phalanx* (the terminal phalanx of a digit), and the *ungual bone* (lacrimal bone).

Unguentum.—A semi-solid pharmaceutical preparation made up of a fatty material and a drug, and intended to be applied to or rubbed into the skin; there are many unguenta in the B. P. *See* PRESCRIBING (*By the Cutaneous Surface, Ointments*).

Ungulates. *See* LYMPHATIC SYSTEM, *Physiology and Pathology* (*Distribution of Lymphatic Glands in Animals*).

Uni-.—In compound words *uni-* (Lat. *unus*, one or single or sole) means one or solitary; e.g. *uniaxial* (having one axis), *unicellular* (composed of one cell), *unilocular* (having one cavity), *unipara* (a woman who has borne one child), and *unipolar* (having one pole or process).

Uniceptor.—In immunity this term is given to a receptor which has a single uniting bond for combining with the toxine. *See* IMMUNITY, RECENT VIEWS.

Unicornis.—Having a single horn, *e.g.* the *uterus unicornis*. *See* UTERUS, MALFORMATIONS OF.

Unilateral.—Affecting one side only; *e.g.* unilateral paresis or paralysis, unilateral hypertrophy, unilateral hyperæmia, etc.

Uniovular.—Developed from a single ovum, *e.g.* uniovular or monochorionic twins. *See* PREGNANCY, MULTIPLE.

Unit.—A single thing or a quantity used as a means of estimating or comparing other quantities, *e.g.* an immunising unit or a serum unit.

Unna's Bandage.—A bandage stiffened with gelatine, used in the treatment of callous ulcers. *See* Ulcers and Ulceration (*Treatment*).

Unna's Dermatosis. — Seborrhœa. *See* Acne (*Unna's Theory of Causation*); Skin, Diseases of Sweat and Sebaceous Glands (*Seborrhœa*).

Unna's Paste.—A paste consisting of two parts each of mucilage of acacia and glycerine and of one part of oxide of zinc; it is used in eczema. *See* Eczema (*Treatment*).

Unsound Food.—Food (meat, poultry, game, fish, fruit, vegetables, bread, milk, etc.) which is unfit for consumption or unwholesome in any way; *e.g.* the meat from animals suffering from foot-and-mouth disease, anthrax, actinomycosis, etc., eggs which are decomposed or too far advanced in development, fish containing parasites or ptomaines, etc.; a medical officer of health should have knowledge of the meat inspection, even although he has not himself to carry it out; there are various Acts of Parliament regulating the detection of unsound food and the penalties attached to the selling of it, *e.g.* the Public Health Act of 1875 for England and Wales, the Public Health (Scotland) Act of 1897, etc.

Unsound Mind. *See* Lunacy.

Unwell.—In a special sense "unwell" means menstruating, and the monthly recurrence of menstruation is called the "unwell time." *See* Menstruation (*Synonyms*).

Unzen. *See* Balneology (*Japan*).

Urachus.

Introductory	240
Malformations	240
Diseases	240
Tumours	240
Urachal Fistula	241
Acquired Fistula	241
Urachal Cyst	242

See also Ascites (*Distinction from other Conditions*); Embryology, Human (*Bauchstiel*); Ovaries, Diseases of the (*Tumours, Diagnosis*).

The segment of the allantois which is included in the body of the fœtus appears in a human embryo of the second month as a hollow fusiform body (Hertwig). In its middle part is a dilatation which becomes the bladder; the lower end is the sinus urogenitalis, whilst its upper part, a tube lined with epithelium, becomes the *urachus*. Retrograde metamorphosis is already in progress in the fœtus at term. After birth atrophy proceeds in an irregular manner. The surrounding connective tissue presses at different points against the urachus so as to cause septa to develop in its canal (Jahn). Hence the bead-like dilatations and the bilocular cysts which sometimes develop in the urachus. The urachal canal is lined with stratified epithelium, triple below, double higher up. In any unobliterated portion of the canal this epithelium steadily grows up to adult life. A valvular fold of mucous membrane covers the vesical orifice of the urachal canal and prevents escape of urine upwards from the bladder (Wutz). This *Wutz's valve* is liable to be forced open when there is any obstruction to free micturition, and sometimes yields after a urachal cyst has been excised. Traces of urachal canal remain in every individual (Suchannek). Wutz found cystic dilatations, chiefly near the lower end, in twenty-four out of seventy-four bodies.

The sheath of the canal is composed chiefly of white fibre towards the umbilicus, and of plain muscle fibres lower down. This sheath is the "superior true ligament" of the bladder, and runs from the apex of that organ to the umbilicus. It lies between the transversalis fascia and the peritoneum, surrounded by loose connective tissue. Occasionally the urachus pushes the parietal peritoneum inwards so as to form for itself a mesentery.

Malformations.—The urachus is more or less absent in ectopia vesicæ. Garrigues, in a post-mortem on a virgin aged forty-five, who died after hysterectomy, found that the urachus was practically absent, the bladder being triangular and extending nearly to the umbilicus, to which it was connected by a very short tube Thorn, in an operation for the removal of a pair of broad ligament cysts, noted that the bladder had a double fundus, with a double urachus joining in a cystic dilatation behind the umbilicus.

Diseases.—In two out of the twenty-four cases where Wutz discovered cystic dilatations, suppuration, due to septic inflammation elsewhere, was found in these minute cysts. Thus *inflammation* of the urachus is not unknown. It may account for some abscesses of the abdominal wall near the middle line, independent of suppuration around an old urachal fistula.

Tumours.—Bland-Sutton and Aveling report one case of myosarcoma of the sheath of the urachus, weighing nearly five pounds. Hoffmann records one case of epithelioma of the mucous membrane of the urachus in a man aged twenty-eight. The patient was born with a urachal fistula; within three years of his death it suppurated, and pus, with urine, escaped through the umbilicus.

The most important diseases of the urachus are two closely allied conditions, *fistula* and *cyst*. Possibly some *umbilical calculi* may have de-

veloped by slow leakage of morbid secretion from an unobliterated canal. An umbilical calculus may attain the size of a pigeon's egg (Hahn). Spencer Wells, in an ovariotomy, found the urachus, though closed at both ends, open for the whole length of the abdominal incision, and filled with small urinary concretions.

Urachal Fistula; Unobliterated Urachal Canal.—The canal is often open when local malformations exist. Freund observed this condition in a woman with uterus unicornis and masculine characteristics. Pearson of San Francisco describes the case of a male child two years of age, robust and masculine, yet with no external organs; the urachus served as a urethra. Extreme distension of the fœtal bladder does not necessarily force open the urachus; thus in Couvelaire's case the urachus was found intact.

Urachal fistula, the result of patency of the canal, may be *congenital* or *acquired.*

Congenital urachal fistula is seen at birth, or develops early in childhood; under the latter circumstances it merges into the acquired variety. It may be associated with phimosis, with congenital stricture of the male urethra, and with an abnormal mucous fold closing the meatus in the female (Stierlin). But in at least fourteen cases, out of which twelve were males, no cause for the patency of the urachus could be found (Stadsfeldt). In some there were adjacent malformations, such as unusually high insertion of the ureters (Mikulicz).

Symptoms and Diagnosis.—Escape of urine from the umbilicus betrays a congenital urachal fistula. Sometimes there is a deep, funnel-shaped orifice at the umbilicus; more often the umbilical canal opens out the apex of a protuberance on the navel made up of granulations. Inversion of the bladder through the umbilicus has been recorded (Gusserow). Simultaneous catheterism through the umbilicus and the urethra will settle the diagnosis. But the urachal canal may be too narrow to admit even a probe; in such a case milk, or a solution of starch, injected into the bladder through the urethra, will most probably escape out of the umbilicus (Freer). The only other embryonic canal opening into the umbilicus is the omphalomesenteric duct, but a patent Meckel's diverticulum is very rare. Lexer, however, dissected out what he thought was a patent urachus in a boy aged one year and a half. On microscopical examination it proved to be a Meckel's diverticulum.

Treatment.—In every case careful search must be made for the cause of the continued patency of the urachus, then that cause, stricture of the urethra, phimosis, etc., must be treated. Spontaneous closure of the urachus often follows an operation which insures normal micturition. When no cause can be discovered, yet some complication like cystitis exists, it must first be treated, then operative measures may be advisable. In infants firm pressure from strapping has often caused permanent cure, and thus the disadvantages of an operation-scar may be avoided. Caustics, closure by hare-lip pins, or even resection of the canal alone, are known to be unsatisfactory. The umbilicus must be dissected off by a circular incision, then the urachus is set free, and a small piece of the bladder around its inferior attachment is excised by a transverse incision. The wound is at once closed by suture. During the operation a catheter must be held in the bladder, and any wound of the peritoneum closed at once with fine silk. It is best to drain the lower angle of the abdominal wound with a strip of gauze. Unless the small piece of bladder which includes Wutz's valve is removed that valve may be forced, and a fistula will develop in the stump of the urachus. A ligature is apt to cut through the stump without obliterating the canal. This operation has given very good results (Delagenière, Lexer, Mikulicz). The abdominal wound must be carefully closed with sutures.

Acquired urachal fistula signifies the forcing open of an imperfectly closed urachus in adult life or late in childhood. Wutz's valve is forced by pressure of urine, which enters the urachal canal and opens up its umbilical orifice. Among the causes of this form of fistula are stricture of the urethra, enlarged prostate, chronic cystitis, pressure of an ovarian cyst on the bladder (Cinti), and retention of urine from retroversion of a gravid uterus (Unterberger, Neugebauer, Drummond Robinson, and probably Chopart's case). In Levêque's case, where a man aged eighty had passed urine through the umbilicus ever since he was fifty-five, there was neither stricture nor any other apparent cause for the fistula. Vesicle calculi may be associated with the fistula (Goldschmidt). In Paget of Leicester's case the calculus was ring-shaped, and had developed on a hair. It was extracted through the umbilicus.

Acquired urachal fistula is a serious condition, for it nearly always implies disease of the lower urinary tract, and not rarely "surgical kidneys" as well. There is often much low inflammation around the fistula, which then forms a sausage-shaped swelling (von Bramann).

Diagnosis of acquired fistula is easy when simultaneous catheterism through the urethra and the umbilicus is practicable, or when a probe can be passed into the bladder from the umbilical orifice. But catheterism is not always possible. Freer's starch solution (*see* above, under "Congenital fistula") should be tried. Gall-stones and echinococci have been known to escape through the umbilicus, and discharge of pus and serum often precedes their exit. Umbilical calculi made up of cotton and woollen fibres felted together with sebaceous matter may also set up free discharge. In all these

conditions the discharge must not be taken for urine mixed with pus. But gall-stones have been found in the urachal canal (Bramann), and in Faber-Köstlin's case, where fifteen gall-stones had been discharged through the urethra in three years, it was discovered after death that the urinary and gall-bladders communicated through an open urachus. It is doubtful whether a diverticulum of the bladder can push its way to the umbilicus. Alleged cases may have been instances of urachal fistula or of bladder replacing the urachus (*see* Garrigues' case, above). Some authorities believe that extravasated urine may leak along the connective tissue of the urachus and find its way out of the umbilicus. A patent Meckel's diverticulum is to be distinguished by examination of a piece of its mucous membrane (*see* Lexer's case, above). Instead of the stratified epithelium of the urachus most of the structures of intestinal mucosa will be detected.

Treatment.—The fact already noted, that acquired urachal fistula is often associated with disease of the urinary tract, must never be overlooked. Neglect of this principle means the certain failure, if not worse, of any radical operation. If, on the other hand, the state of the bladder, etc., is considered and treated, spontaneous closure of the fistula may follow. This occurred in Fraser's case, treated by free washing out of the bladder and urachus, and in Goldschmidt's (*see* above) after suprapubic lithotomy, but not in Paget's case, for urine continued to escape through the umbilicus six years after the stone had been extracted from the fistula. Exposure of the urachal canal and scraping its mucosa are dangerous and caustics unsatisfactory. When the urinary tract and the tissues around the urachus are healthy, complete resection of the fistula, as described above under the treatment of congenital fistula, has proved permanently successful.

Urachal cyst includes two varieties: cystic fistula and true cyst. The first is *cystic dilatation* of the urachal canal which opens into the bladder, but is closed at the umbilicus. G. Morgan's (Brighton) case was in a boy, aged five. Newman's patient was a man aged thirty-nine, and subject to a hypogastric swelling. Pain and retention of urine set in suddenly without apparent cause. Twenty ounces of bloody urine were drawn off from the bladder; a cyst was then detected above it. A fortnight later an operation was performed; the cyst was entirely in front of the peritoneum. It was drained, urine escaped from its cavity for about a month, then it closed completely.

The second variety is *true cyst of the urachus*, about which much has been written. But the term "urachal cyst" has been applied by Hoffmann and Lawson Tait to cases where there was little or no evidence that the tumour had developed from the urachus. Wutz has noted that in Hoffmann's often-quoted case, where fifty litres of fluid were found in the cyst, the urachus was quite distinct. Lawson Tait failed to prove that any of his big so-called "allantoic cysts" were urachal. Some were clearly intra-peritoneal. Tait's theory that the allantois may form a kind of serous membrane for the pelvic viscera has never been proved by dissection.

An unobliterated portion of the urachal canal is often detected at an abdominal operation. This condition represents the origin of true cysts of the urachus of which several authentic cases have been reported. They are not associated with malformation of the rest of the genito-urinary tract, excepting one of Bryant's cases, where there was hypospadias. In any case it is possible that Wutz's valve may be forced after the cyst has developed so that it is not always easy to distinguish true cyst from cystic fistula.

In the author's case the cyst was bilocular. The larger and upper cavity contained over a pint, the smaller about half a pint of a greenish brown fluid. In one case under Bryant ovarian cyst was suspected. The tumour, preperitoneal as in all urachal cysts, was closely associated with the fundus of the bladder. In a second case under that surgeon a painful swelling developed below the umbilicus in a man aged thirty-five, with slight hypospadias. During drainage after incision some urine came away. Ill, of Newark, New Jersey, reports a bad case of urinary fistula after draining a closed or true cyst. Ferguson, of Troy, N.Y., operated on a man aged forty-seven. The cyst contained two quarts of a watery fluid, and bore a distinct and separable lining membrane which sent digital prolongations between the layers of the abdominal wall near the navel, as often seen in umbilical hernia. Douglas of Nashville removed a urachal cyst containing twenty-five pints of fluid. Lawson Tait enucleated a hypogastric preperitoneal cyst which dipped into the pelvis; the patient was a woman aged twenty-eight. In Rotter's case the cyst-walls were continuous with the coats of the bladder, and at the point of junction was a villous cancer of the vesical mucous membrane already detected by the cystoscope. The cancer and the bladder-walls around it were dissected away together with the urachal cyst. Ill incised and drained a hypogastric cyst of ten years' duration during the third month of pregnancy; over eight pints of fluid came away. In Morestin's case two distinct cysts, developed from the bead-like dilatations mentioned at the beginning of this article, were detected during an operation for pyosalpinx.

Diagnosis.—Urachal cyst forms a fluctuating swelling between the umbilicus and pubes. When it varies in size in the course of a few months its nature will be more clear, as it probably communicates with the bladder. The true cyst is entirely separate from the bladder, it tends to increase slowly, and does not become

smaller when the bladder is emptied by the catheter. Its anterior surface is often flat, as it meets resistance from the sheaths of the recti, and so bulges backwards towards the peritoneum rather than forwards. In position it is not always symmetrical, sometimes it lies mainly on one side of the middle line. As a rule, it is only by an exploratory incision that the nature of this kind of cyst can be made evident. Then it will be found that the parietal peritoneum lies entirely behind it. In the female a preperitoneal cyst may arise from the broad ligament, burrowing forwards and upwards (Wutz or Wolff's case; author), but the base of such a tumour lies in the iliac fossa, not over or behind the bladder. The walls of a urachal cyst may show plain muscular fibre under the microscope (author's case); the urachal epithelium is usually destroyed. Encysted dropsy has been taken for a urachal cyst.

Treatment.—When the cyst, owing to chronic inflammatory changes, cannot be enucleated, it must be drained. This treatment answers well in bad cases, but urinary fistula may develop. In Bryant's second case, in Ill's, and in the author's, drainage was followed by closure of the cyst. When the cyst walls are healthy enucleation should be attempted. Tait, Bryant (case 1), and Rotter successfully enucleated the cyst, and had no trouble with the parietal peritoneum. Douglas lost his case of enucleation through sloughing of the large peritoneal flap detached from the back of the cyst. In Ferguson's case the inner wall of the cyst was stripped off almost entirely. Much of the outer wall with the underlying peritoneum was excised, and puckering of the remainder of the wall remedied by suitable application of sutures. The danger of urinary fistula through forcing of Wutz's valve must be remembered, as in operations for urachal fistula. Ill found a short tract of patulous urachus between a urachal cyst and the bladder. He treated this tract as though it were a diseased vermiform appendix, cutting it very short, forcing in the cut edges, and closing them in with a Lembert suture. Excision of a small piece of the walls of the bladder around the inferior attachment of the cyst, as in the operation for fistula, might be still safer surgery in such a case. It should always be practised, if possible, in operations on cysts of the first variety, mentioned above, where the bladder communicates with the cyst cavity from the first.

Uræmia.

DEFINITION 243
CLASSIFICATION 243
1. ACUTE 243
Premonitory Symptoms . . 244
Convulsive Attacks . . . 244
Temperature 244
Pupils 244
Special Senses 244
2. CHRONIC 244
Nervous Symptoms . . . 245
Respiratory 245
Digestive 245
3. LATENT 246
THEORIES, EXPLANATORY 246
DIAGNOSIS 248
PROGNOSIS 249
TREATMENT 249

See also ALCOHOLISM (*Acute, Diagnosis*); AMBLYOPIA (*Toxic, Uræmia*); ASTHMA (*Paroxysmal Uræmic Dyspnœa, Diagnosis from*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Hœmorrhage, Diagnosis*); BRAIN, SURGERY OF (*Compression, Diagnosis*); BREATH (*Odour*); CIRCUMCISION (*Phymosis, Complications*); DELIRIOUS MANIA, (*Diagnosis*); DRUG ERUPTIONS (*Diagnosis from Uræmic Rashes*); HEADACHE (*Diagnosis of Cause*); HICCOUGH (*Etiology*); HYDROPATHY (*Indications*); INSANITY, ETIOLOGY of (*Uræmia*); KIDNEY, SURGICAL AFFECTIONS OF (*Suppurative Pyelonephritis, Diagnosis*); LARDACEOUS DEGENERATION (*Effects*); LIVER, DISEASES OF (*Cirrhosis, Symptoms*); LUNG, TUBERCULOSIS OF (*Complications*); MORPHINOMANIA (*Effects*); MUMPS (*Complications*); NEPHRITIS; PREGNANCY, DEATH DURING (*Causes*); PRURITUS (*Causation of Pruritic State*); PUERPERIUM, PATHOLOGY (*Insanities, Causes*); RESPIRATION (*Cheyne-Stokes Respiration*); TYPHUS FEVER (*Diagnosis*); UTERUS, MALIGNANT TUMOURS OF (*Cancer of Cervix, Symptoms*).

URÆMIA.—Uræmia is a convenient comprehensive term for numerous toxic symptoms met with in the course of several forms of renal disease or in connection with interference with the ordinary eliminative work of the kidneys. The symptoms are, in the main, due to toxic functional disorders of the nervous system, and accordingly are generally of a nervous type, but occasionally they simulate diseases of the respiratory and digestive systems.

Uræmic symptoms may be divided into three broad clinical types:—

1. Acute or fulminating.
2. Chronic, subacute, gastro-intestinal, or respiratory.
3. Latent.

1. ACUTE OR FULMINATING URÆMIA.—This type occurs mainly with acute nephritis, especially when resulting from scarlet fever; it may arise in chronic nephritis, when the urine is reduced in amount, in consequence of a subacute extension of the disease. It often accompanies non-obstructive suppression of urine due to catheterism, abdominal injuries or diseases, or operations upon the kidneys. Severe renal congestion, the result of disease, or of poisons or toxins developed in the course of disease, is also frequently followed by acute uræmia.

The onset may occur without any warning, or it may be preceded by various premonitory symptoms.

Premonitory Symptoms.—The most marked premonitory symptoms are constant headache, mental apathy, drowsiness, and vertigo; nausea, or even vomiting, may sometimes occur, while in some few cases there may be severe dyspnœa. Occasionally there is marked diminution in the quantity of urine. Attention has been directed to a strange, fixed expression of the face, to dragging pains in the extremities, or to a transient rigidity of the face, of the lower jaw, or of one of the limbs. Tremors, similar to those of paralysis agitans, have also been described. Abnormal hardness and high tension of the pulse commonly precede the more obvious symptoms of uræmia.

Symptoms.—(i.) *Convulsive Attacks.*—The symptoms fall into two classes: Those due to paralytic affection of the brain and of some of the sensory centres, and those referable to irritation of the motor tract, leading to tonic and clonic convulsions. Frequently both types are present in varying degree. The immediate onset is often marked by sudden paroxysms of convulsions of an epileptiform type, which may occur with or without premonitory symptoms. The patient becomes unconscious and convulsed; sensibility and reflex actions are lessened and frequently abolished. In slight cases the spasms may be confined to passing contractions of the muscles of the face or of the extremities, but in serious cases they may be more violent and general, and the whole body may be shaken with violent convulsions, while respiration falters and becomes stertorous. In such severe cases the patients foam at the mouth and grind their teeth, while fæces and urine may be passed involuntarily.

The clonic spasms ordinarily only last a few minutes. When but one fit occurs the convulsions cease gradually, and the patient passes into a drowsy or comatose condition, from which he may be partially roused for a short time, and again appears to fall asleep. After an interval, varying from a quarter of an hour to several hours, he awakes as from profound sleep. Generally, after a few hours or days, the attacks return with renewed vigour, the intervening stupor becomes more profound, and at last passes on to permanent coma. Death may occur during the comatose state, or at the height of one of the convulsive seizures.

The clonic convulsions are sometimes unilateral, and may be preceded or replaced by tonic spasms, constituting "tetanic uræmia," which may be of long duration.

Occasionally there are no spasms, the acute uræmia being marked by vomiting and an apathetic condition, which gradually passes on to coma or delirium. In a fatal case without convulsions recently observed aphasia and marked contraction of the pupils were the chief signs preceding coma.

A convulsive attack is sometimes accompanied or followed by some form of motor paralysis, especially hemiplegia; this is frequently transient and incomplete, but reflexes are abolished or diminished, and the condition may be followed by contracture. When the face is affected ptosis is exceptional, but conjugate deviation of the eyes has been observed.

(ii.) *Temperature.*—During the convulsive attacks the temperature is generally low, or even subnormal, but in exceptional cases great rises of temperature have been noted. Low temperatures ranging from 86·1° Fahr. to 94·4° Fahr. have been noted in uræmia consecutive to diseases of the urinary passages; it has been suggested that low temperatures are more frequent when uræmia affects persons advanced in years, or when it occurs in conjunction with exhausting conditions, such as vomiting, diarrhœa, or hæmorrhages, or in connection with cancerous cachexia.

In the rare cases in which the temperature has been found to rise as high as 105·8°, or even 107·4°, this sudden elevation has always been followed by a rapid fall.

(iii.) *The Pupils.*—During the convulsion and subsequent coma the pupils usually retain their sensitiveness to light, but authorities are not agreed about their size. Fagge says they are more often contracted or normal, while Wagner states that they are as a rule dilated, seldom small; in my experience I have found no definite change with sufficient frequency to warrant a general statement.

(iv.) *Special Senses, Sight and Hearing.*—Convulsions may be followed by dimness of vision or even by actual blindness, which may be transient or permanent. The condition has been attributed to transient œdema of the retina, or to œdema of the brain. Ophthalmoscopic examination during uræmic amaurosis generally gives negative results, and the pupillary reflex is intact. Vision may be regained after twenty-four or thirty-six hours, and recovery has been known to occur even after seventeen days of total blindness.

The sense of hearing is comparatively rarely affected, but occasionally it is impaired as the result of uræmia. Singing in the ears, difficulty in hearing, or complete deafness may suddenly supervene and depart as quickly. Uræmic deafness has been attributed to minute hæmorrhages in the cochlea, but, from the rapidity of recovery, it is far more probable that it generally depends upon some toxic influence acting upon the auditory centre.

2. Chronic Uræmia.—The most obvious symptoms of chronic uræmia are those connected with the respiratory and digestive systems, but several distinctly cerebral symptoms are to be noted, and there are good grounds for believing

that, in all cases, the toxic influence is mainly, if not entirely, exerted upon the central nervous system.

SYMPTOMS.—(i.) *Nervous.*—The onset is often very gradual, and may be unrecognised or attributed to some other cause. Frequently there is complaint of dull headache affecting the occipital region, or less commonly the frontal; occasionally the pain is more acute, and, when paroxysmal, it simulates violent migraine. It may be accompanied or replaced by giddiness and drowsiness, or by languor and inertia, which gravely interfere with all mental and muscular efforts. These symptoms are peculiarly persistent, though there may be brief intermissions. They may be succeeded by respiratory and digestive symptoms, or the lethargy may increase until it leads to stupor or coma, with perhaps attacks of delirium or with convulsive seizures similar to those of acute uræmia. When coma supervenes without convulsions the tongue becomes dry and brown, sordes collect upon the teeth, and the condition resembles that of an advanced stage of typhoid fever.

The mental derangements in chronic uræmia include hallucinations, terror, delusions of persecution, and excessive motor irritability. The type of delirium is generally quiet, but occasionally it may be furious. Binswanger, who describes several cases, has found that exacerbations and remissions of the renal disease keep pace with similar variations of the symptoms of mental derangement.

Severe itching of the skin is very frequent and often very distressing. In old people it leads to intense pruritus which defies all local treatment. The irritation may disturb sleep, and even when sufficiently comatose to resist all efforts at rousing them the patients may continue to rub and scratch themselves. Cutaneous hyperæsthesia with burning or stabbing sensations are sometimes met with.

It is possible that some of the eruptions which occur in uræmia are primarily due to central nervous changes; they may appear as maculæ or papulæ of a bright red colour upon the extensor surfaces of the forearms and legs, spreading rapidly over the whole body, or as an erythematous, scarlatiniform, papular, or vesicular eruption. Sometimes the rash becomes eczematous; frequently it is accompanied by severe itching.

(ii.) *Respiratory System.*—Reference has already been made (*see* article on "NEPHRITIS," vol. vi.) to the various forms of dyspnœa met with in uræmia independently of the dyspnœa due to œdema of the lung or fluid in the pleural cavity, though frequently associated with these conditions. The chief types are (1) continuous dyspnœa; (2) paroxysmal dyspnœa; (3) Cheyne-Stokes breathing. The symptoms of (1) and (2) resemble those of spasmodic asthma in their sudden onset, and they are frequently accompanied by sibilant râles, but the term "uræmic asthma" is to be deprecated. The condition does not ordinarily yield to any of the usual remedies for asthma, while relief may sometimes be afforded by measures calculated to remove nitrogenous waste material. The attacks usually occur at night, and the dyspnœa is often mainly expiratory, though there may be much inspiratory difficulty. The dyspnœa has often a peculiar hissing character, suggestive of laryngeal trouble, but it is undoubtedly due to central changes, rather than to laryngeal or pulmonary obstruction. Similar breathing may occur in connection with acute uræmia, indeed it is considered to be most common with the uræmia of scarlatinal nephritis; in chronic cases it is thought to be most frequent when the nephritis results from alcoholism. It must be remembered that attacks of dyspnœa in chronic nephritis may be due to gradual failure of the circulation, or to interference with the work of the lung, owing to engorgement or to effusion into the pleural cavity, and that these conditions are not necessarily associated with uræmia.

The well-known Cheyne-Stokes respiration frequently occurs shortly before death in cases of chronic nephritis, especially in those with secondary cardiac failure, and it is generally thought to be of toxic origin.

Persistent hiccough is another distressing symptom in severe cases tending to a fatal termination. It has been observed in numerous cases of renal cirrhosis and extravasation of urine. Although hiccough is generally regarded as a late symptom, it has occasionally been noted early, when only slight œdema of the legs was present.

(iii.) *Digestive System.*—Dyspeptic symptoms are common throughout the course of chronic forms of kidney disease. In the earlier stages of renal cirrhosis they may be of an ordinary type, but when the renal changes are more advanced nausea and vomiting become more frequent, and in their intractable character they resemble other forms of chronic uræmia. The vomiting cannot be traced to any error of diet, and it affords no relief. It often occurs in the early morning, before food has been taken, and the vomited matter may consist either of acid fluid or of alkaline fluid with an ammoniacal odour. Urea has been detected in this fluid, and the alkaline reaction has been attributed to its transformation into ammonium carbonate. It has been suggested that the vomiting is the result of irritation of the stomach by urea or ammonium carbonate, which are being vicariously eliminated. Probably this is erroneous, and the vomiting is of central origin, since when urea has been administered to animals with food, vomiting only ensues after an interval sufficient to allow of absorption.

Diarrhœa is generally a late symptom, and it

may be overlooked or rather attributed to the treatment unless it is associated with uræmic vomiting. Diarrhœa is stated to be most troublesome during the night, when six or more watery stools may be passed, though during the day there may have been only one or two actions of the bowels. This feature is of some diagnostic importance, since in advanced nephritis dysenteric symptoms with definite lesions are not uncommon.

3. Latent Uræmia.—Under this term a third variety has been described, in which all the most obvious symptoms of uræmia are conspicuous by their absence. This form is common with calculous anuria and other varieties of obstructive suppression. In these conditions death occurs in from five to nine days, and the leading symptoms are contracted pupils, a low temperature, occasional vomiting, and towards the end slight muscular twitchings and slight drowsiness. The negative characteristics are the absence of coma, convulsions, and dyspnœa, consciousness being retained until very shortly before the end.

Theories of Uræmia.—Although from the clinical side it may be convenient to include under a comprehensive term diverse symptoms which are undoubtedly the result of kidney disease, it must be remembered that the term *Urœmia* is somewhat misleading unless it is employed in a very wide sense.

Numerous theories, explanatory of the symptoms, have been suggested, and arguments can be adduced in support of most of them. Many turn upon some interference with the eliminative work of the kidney, whereby the system becomes surcharged with one or more of the urinary constituents, which may be directly toxic, or may exert a toxic influence after undergoing some chemical change in the body. The attempt to find a single theory which shall afford a satisfactory explanation of all cases seems doomed to failure in view of the complexity of the symptoms of uræmia.

The theories of uræmia may be broadly divided into two classes:—

1. Mechanical.
2. Chemical.

According to the first some definite physical change affects the nerve centres, such as œdema or minute hæmorrhages; according to the second, the symptoms are to be explained on the theory of the toxic influence produced by some retained constituent of the urine, or resulting from the action of some material formed by decomposition or metabolism within the system.

1. *Mechanical.*—Numerous minute hæmorrhages have been found in various parts of the brain, but these are so rare and so uncertain that they must be regarded as the results rather than the cause of the convulsive seizures. Traube thinks that the basis of uræmic symptoms is to be found in œdema of the brain, which from its increased volume induces anæmia. Two conditions predispose to œdema—the marked hydræmia and the increased arterial pressure. If the arterial tension is by some accidental means suddenly raised, or the density of the blood serum suddenly further diminished, the serous fluid will transude into the brain substance and cause œdema. Traube considers that the special symptoms of the uræmic attack are dependent upon the degree and localisation of the œdema in the brain: convulsions occur when the middle lobes are affected; coma when the œdema extends to the whole cerebrum. Traube's theory is supported by (1) the frequency of cardiac hypertrophy and hydræmia in cases of uræmia; and (2) by the production of coma and convulsions in dogs by the injection of water into the carotid after ligature of the ureters and of one of the jugular veins. Against this theory it has been urged that the symptoms are only produced experimentally when enormous quantities of water have been injected, and that even under such circumstances the brain may present no indication of œdema. Moreover, from the clinical side hypertrophy of the heart and hydræmia are not always accompaniments of uræmia, while post-mortem examination frequently fails to demonstrate œdema of the brain. Even when cerebral œdema has been recognised it has been thought to be the result of the convulsions rather than the cause. In spite of these objections Traube's theory has, in a modified form, found several adherents; it has been held that in scarlet fever, inflammatory œdematous conditions of the brain and its meninges co-operate with the action of some result of infection.

2. *Chemical.*—The term "uræmia" was first employed under the idea that all the symptoms depended upon the non-elimination of urea, its circulation in the blood, and its toxic action upon the central nervous system or upon other organs.

The following observations have been advanced in favour of the theory that uræmia is due to the *retention of urea*:—(i.) The appearance of marked symptoms when the secretion of urine and the elimination of urea are much reduced; (ii.) the detection of urea in the blood under these conditions; (iii.) the production of many of the ordinary symptoms, such as drowsiness, convulsions, and vomiting, when the excretion has been experimentally arrested by ligature of the renal arteries, or the ureters, or by extirpation of the kidney.

Against the retention theory it must be admitted (i.) that all symptoms of acute uræmia may be absent when complete suppression has lasted for many hours, or even days, without being accompanied by vomiting or diarrhœa, through which vicarious elimination might have been effected; (ii.) that uræmic symptoms, in their frequency and severity, do not clinically

exhibit any necessary relation to the quantity of urea excreted; (iii.) that occasionally no symptoms of uræmia may be present, even though large quantities of urea may be detected in the blood; (iv.) that when urea is given to animals with their food no symptoms are produced so long as it can be freely excreted; (v.) that frequently in dogs uræmic symptoms are not produced even by the injection of large quantities of urea into the circulation. By the extirpation of the kidneys it has been possible to estimate the amount of urea retained within the system from the time of the operation until the time of death from uræmic symptoms, and it has been found that far larger quantities may sometimes be introduced directly into the blood without any resulting uræmia.

Some observers maintain that if in animals the rapid elimination of urea is prevented by ligature of the ureters, death with uræmic symptoms occurs much more quickly than where the kidneys have been extirpated: this appears to indicate that the presence of urea is not the sole determinating factor in uræmia, but that in all probability some other deleterious substance is elaborated by the kidney.

As the result of much experimental work Frerichs suggested that the toxic agent in uræmia might be *ammonium carbonate*, resulting from the decomposition of urea in the blood, owing to the presence of some ferment. The arguments in support of this theory are (i.) the comparative ease with which urea can be transformed into ammonium carbonate; (ii.) the ammoniacal odour of the breath in cases of uræmia; (iii.) the occasional detection of small quantities of ammonium carbonate in the blood in puerperal eclampsia; (iv.) the similarity of the symptoms produced by the experimental injection of ammonium carbonate; (v.) the possible absorption of ammonium carbonate resulting from decomposition of urea within the intestine or urinary passages. The objections to this theory are (i.) that the ammoniacal odour of the breath depends upon decomposition within the mouth; (ii.) that it occurs both in health and in sickness, and is not limited to renal affections; (iii.) that in uræmic persons this transformation of urea in the blood does not occur; (iv.) that the injection of urine, which has undergone spontaneous decomposition, is not followed by uræmic symptoms unless the urine has been made septic by infection.

The difficulties connected with the two chemical theories above mentioned led Feltz and Ritter to test the effects produced by the intravenous injection of the various constituents of the urine. They concluded that the convulsions, coma, and death which ensued after the injection of fresh urine could not be attributed to increased pressure, nor to the organic constituents, and that the *potassium salts* were the most powerfully toxic of the inorganic constituents. They maintain that similar symptoms of uræmia were produced by injecting either fresh urine or distilled water containing the same proportion of salts of potassium. By one observer symptoms of respiratory trouble have been attributed to the retention of salts of potassium; while another considers potassium chlorate to be the principal cause of vomiting. In opposition to this theory of the toxic influence of potassium salts, it has been stated that in some cases of puerperal eclampsia the proportion found in the blood was below the normal amount; it must be remembered, however, that many of the clinical features of eclampsia differ from those of uræmia.

Alteration in the excretion of *chlorides* has been suggested as an explanation of uræmia. Clinically marked diminution of chlorides in the urine has been noted in acute and chronic nephrites, and the hypodermic injection of a concentrated solution of sodium chloride has been followed by clonic and tonic spasms alternating with a semi-comatose condition.

Symptoms resembling those of uræmia have been produced by irritation of the cortex cerebri by creatin, creatinin, leucin, and tyrosin, and the late Sir William Roberts regarded uræmia as the result of the accumulation of these or similar substances in the blood, that is to say, of products intermediate between urea (or uric acid) and the albuminous substances from which it originates.

Bouchard has propounded an extremely interesting theory of auto-intoxication according to which the human body manufactures poisons, which are only prevented from destroying life by their rapid elimination. The kidneys excrete toxic substances such as urea, potassium carbonate, etc., while the intestine removes compounds of potassium and ammonium. If these substances are retained in the body they are said to produce symptoms of uræmia as soon as the "uro-toxic coefficient" is attained.

Recent examination of the blood and tissues in renal diseases has given the following results: (1) In suppression there is a great increase in the nitrogenous extractives in the blood and tissues; (2) in acute uræmia without dropsy there may be a still greater increase; (3) in chronic renal disease fatal from other causes there is some increase, but not comparable with the above; (4) in renal disease, even in apparent good health, there is a certain small excess; and (5) in eclampsia the excess is trivial in amount. From these and other data Bradford maintains that uræmia cannot be held to be due to simple retention, and that in kidney disease, and after experimental lesions of the kidney, there is evidence of great tissue disintegration, and that it is more than probable that uræmia is dependent upon this, directly or indirectly. He suggests that the kidneys in some way control the metabolism of the tissues of the body,

and that, when the kidney substance was greatly diminished in amount or entirely removed, the blood not only contained a great excess of urea, but the tissues, and especially the muscles, contained large quantities of urea and other nitrogenous bodies of the creatin class. Several other observers have also noted that in uræmia there is an accumulation of creatin-like bodies.

The frequent occurrence of uræmic symptoms during the rapid disappearance of renal dropsy is extremely suggestive. It has been thought that certain toxic alkaloids or ptomaines become stored up in the dropsical fluid, and that they may under certain conditions, especially when attempts are made to reduce the dropsy rapidly, be suddenly thrown into the circulation, and thus induce convulsions and coma. Such symptoms are usually attributed to reabsorption of nitrogenous materials, but Dr. William Carter has suggested that they may be due to a progressively diminished alkalinity, or perhaps actual acidity of the blood. In support of this contention he states that although, under ordinary circumstances, subcutaneous effusions are alkaline or neutral, in Bright's disease they are frequently acid.

From the clinical side it appears almost impossible to accept any single chemical theory in explanation of all forms of uræmia. It has been argued that poisons like morphine, arsenic, or belladonna, produce very different symptoms in different individuals, or in different doses, and that therefore there is no inherent difficulty in assuming similar variations as the result of a single poisonous substance in uræmia. Although this argument is perhaps permissible with the ordinary symptoms of acute and of chronic uræmia, it is scarcely to be regarded as satisfactory when applied to the modified forms of either condition. It is more likely that these variations are due to the accumulation within the body of several poisonous products of slightly different action, than that they can be ascribed merely to idiosyncrasies or to different doses of one toxic substance.

Diagnosis. — When the previous medical history is known there is usually little difficulty in correctly estimating the symptoms of the acute form of uræmia. Changes in the urine, both in volume and in specific gravity, may have sounded a warning note, or attention may have been attracted by various premonitory symptoms, and the uræmic manifestations may have been anticipated. The chief difficulty arises in patients found convulsed, or comatose, and brought under medical notice without any definite history. Suspicion may be aroused by an ammoniacal odour of the breath, though this sign is frequently masked by the smell of alcohol, taken previous to the attack or administered with good intent by some bystander. The presence of dropsy, even slight in amount, would be a valuable indication, while high tension of the pulse, outward displacement of the apex beat, and accentuation or reduplication of the sounds of the heart, would favour the diagnosis of uræmia in a convulsed or comatose patient. If urine withdrawn by a catheter is found to be albuminous and to contain casts, the hypothesis of uræmia is further strengthened. Many of these indications may, however, be present with grave cerebral lesions, with which renal changes may be directly associated. Cerebral hæmorrhage, for example, often results from arterio-sclerosis, a sequel of renal cirrhosis, and when the sudden unconsciousness is accompanied by convulsions the diagnosis may be very difficult. Although uræmic convulsions may be unilateral, or at least more marked upon one side, as in hemiplegia, the recognition of distinct paralytic symptoms generally points to hæmorrhage rather than to uræmia. Convulsions and coma may also be due to various forms of cerebral tumour, or of meningitis, particularly tuberculous, which may occur independently in patients already affected by chronic renal trouble. Alterations of temperature afford very little help to the diagnosis; in meningitis the chart is not characteristic, in apoplexy the temperature may be normal or subnormal, though usually high in basal hæmorrhage, while in uræmia the occurrence of a high temperature is quite exceptional. Help may be afforded by an ophthalmoscopic examination, which may disclose characteristic white patches or flame-shaped hæmorrhages when acute uræmia occurs with chronic renal changes, while in many cases of meningitis and of cerebral tumour optic neuritis and other equally marked deviations from the normal may be recognised.

The convulsive seizures of uræmia may at first be mistaken for hysteria or epilepsy, but an examination of the urine and the recognition of injury of the tongue should leave no room for doubt.

The coma of uræmia may be mistaken for that due to alcohol, to opium, or to diabetes. With alcoholic coma the pupils are generally dilated, while they are contracted in opium-poisoning. In diabetes the pupils afford no help, and the examination with the ophthalmoscope may reveal changes very similar to those in chronic nephritis. The examination of the urine, of the pulse, and of the heart may serve to indicate the true cause. Reference has already been made to the difficulty of dealing with the cases of prolonged partial coma, with dry, furred tongue, in which there is marked resemblance to the late symptoms of typhoid fever.

The diagnosis of chronic uræmia may give even greater trouble than that of acute, and may only be reached when symptomatic treatment has proved unavailing. The nervous symptoms—headache, neuralgia, and languor—are characterised by being peculiarly persistent

and intractable; they differ from similar conditions of rheumatic origin in presenting but little change with climatic alterations; small traces of albumin and low specific gravity may aid the diagnosis, but the position of the apex beat, the sounds of the heart, especially the accentuation or reduplication over the pulmonary valves, and alterations in the pulse, increase of tension, or the characteristic sphygmographic tracing, are to be regarded as of greater value.

In advanced cases of chronic nephritis, shortness of breath on exertion, tendency to recurrent cough, and occasional attacks of spasmodic dyspnœa, can only be attributed to uræmia, in the absence of physical signs of cardiac or pulmonary affections, or when the symptoms persist in spite of improvement in the physical signs. Usually symptomatic treatment is first adopted, although a provisional diagnosis of uræmia may have been made, and it is only when the ordinary remedies fail to give relief that increasing confidence is felt in the theory of toxæmia, and a change of treatment may give surprising benefit.

Similarly, dyspeptic symptoms, vomiting, and diarrhœa may be mistaken for the results of local lesions of the stomach or intestines; the absence of physical signs of tumours connected with the stomach, the sigmoid flexure, or the rectum, helps to differentiate uræmia from malignant diseases of these organs, and greater certainty may be attained by the examination of the vomited materials, or by testing for free hydrochloric acid after the removal of the contents of the stomach. The wasting, pallor, and weakness of advanced nephritis resemble those resulting from malignant growths. It must be admitted, however, that in chronic uræmia the symptoms so frequently resemble those due to other causes, that, in spite of the utmost care, it may be advisable to await the results of treatment before expressing a definite opinion as to the nature of the case.

Prognosis.—The prognosis of uræmia is always serious. The immediate danger depends upon the type of uræmia, and also upon the form of renal disease. Symptoms of acute uræmia necessarily cause great anxiety; death may ensue in the course of the first convulsive attack, but it more commonly results from exhaustion, when numerous attacks are repeated at short intervals. In scarlatinal nephritis the prognosis is somewhat more hopeful than in the more ordinary form of acute nephritis; in both conditions the prognosis is influenced by the rapidity with which the coma passes off, and by the speedy re-establishment of the renal excretion. When convulsions occur with chronic nephritis they may similarly prove fatal during the first attack, and although in exceptional cases the end may be remote, it usually occurs within a few weeks or perhaps months. In such cases the prognosis will be based upon the degree of stupor or coma after the convulsive seizures, and upon the general characters of the urine.

Respiratory and digestive disturbances, indicative of chronic uræmia, always justify an unfavourable prognosis. The immediate dangers of uræmic dyspnœa are sufficiently obvious, while vomiting and diarrhœa, even when not direct preludes to convulsions or coma, reduce the strength greatly and thus increase the risks. Giddiness, headache, neuralgia, and even amaurosis indicate progressive stages of uræmia; but, unless accompanied by a rapid diminution in the specific gravity or in the amount of the urine, they may improve greatly with treatment, or they may remain with little variation for months or even years.

Treatment.—In the article on nephritis will be found a full account of the measures ordinarily adopted to prevent the occurrence of threatened uræmia; it may, however, be useful to give a brief summary here, and to mention the special treatment which may be employed for individual symptoms.

Threatened uræmia entails the use of diaphoretic measures or drugs, of hydragogue purgatives, and occasionally of diuretics. Free diaphoresis is favoured by hot-air baths, hot-water baths, and by the wet pack, hot or cold; the action of these may be promoted by copious draughts of water, taken, for instance, while having a Turkish bath. In very exceptional cases when these diaphoretic measures fail, or when they increase headache, a very small dose of pilocarpine may be injected subcutaneously to initiate diaphoresis. This drug should, however, only be employed on great emergency, and it should never be used when the patient has pulmonary œdema, and is already unconscious.

Diaphoretic drugs are seldom employed in the early stages of uræmia, as many exert a stimulating action upon the renal vessels which is undesirable. Ammonium acetate has fallen into disfavour since the suggestion that uræmia might be due to ammonium compounds in the circulation.

Of the hydragogue purgatives jalap and scammony are the most useful; the official compound jalap powder is frequently administered. Elaterin is sometimes recommended; croton oil is ordinarily reserved for patients already comatose. Calomel should be avoided when the urine contains blood, though in other cases it may be of service.

The convulsions of acute uræmia may be controlled by the administration of chloroform; chloral hydrate and potassium bromide have been given with the same object. When in spite of these measures convulsions occur in rapid succession, the subcutaneous injection of morphine has been advised. This treatment is generally thought to be extremely hazardous; many observers maintain that it increases the

tendency to fatal coma. In plethoric individuals, when diaphoretic measures and hydragogue purgatives fail to check convulsions, venesection is often of service; the removal of twenty ounces of blood, or more, may give great relief. Even with great weakness or threatening collapse, venesection may still be performed with advantage if, at the same time, an intravenous injection of normal saline solution is employed.

With coma, diaphoresis and catharsis should be continued, or chloral hydrate and potassium bromide may be given. The inhalation of oxygen has been tried, with indifferent success, to check the hissing dyspnœa.

The spasmodic dyspnœa of chronic uræmia is sometimes benefited by the administration of nitroglycerin (trinitrin) in solution, or by the inhalation of amyl nitrite; preparations of ether and other diffusible stimulants are also of service. Frequently, however, it will be found that the dyspnœa yields more rapidly when the hot air baths are employed in conjunction with hydragogue purgatives.

Similarly, although neuralgia and headache may sometimes be relieved by phenacetin or acetanilide, better results ordinarily accompany an eliminative line of treatment.

Nausea and vomiting may be controlled by ice, by diluted hydrocyanic acid, and by preparations of bismuth, but the due recognition of their uræmic character and the adoption of diaphoretic and purgative measures will often give greater relief.

Uræmic.—Caused by or characterised by uræmia (*q.v.*).

Ural or Uralium.—Chloral-urethane, a combination of chloral hydrate and urethane ($CCl_3CH.OH.NHCO_2C_2H_5$), which acts as a hypnotic; dose, 10 to 20 grains.

Uranalysis.—Urine analysis, qualitative and quantitative. *See* URINE, PATHOLOGICAL CHANGES IN; URIC ACID; etc.

Uraniscoplasty.—Reparative surgery of the palate (Gr. οὐρανίσκος, roof of mouth), *e.g.* in cases of cleft or fissure; this may take the form of suture of the palate, uraniscorrhaphy or staphylorrhaphy.

Uranism.—Sexual perversion; desire for individual of the same sex; tribadism, Lesbian love, etc.

Uranomania.—Religious mania with exaltation.

Uranoschisis.—Cleft palate (Gr. οὐρανός, the palate; σχίσις, cleft).

Urari. *See* CURARA.

Urate.—A salt of uric acid. *See* GOUT (*Morbid Anatomy, Urate of Soda*); STOMACH AND DUODENUM, DISEASES OF (*General Spmptomatology, Deposit of Urates in Urine*); URIC ACID; URINE, PATHOLOGICAL CHANGES IN (*Sediments*).

Uratic.—Belonging to or characterised by urates, *e.g.* uratic deposits in gout.

Uraturia.—A morbid state of the urine in which there is an excess of urates.

Urberoago de Alzola. *See* BALNEOLOGY (*Spain and Portugal*).

Urea.—Carbamide ($CO(NH_2)_2$), the chief nitrogenous product of the action of the kidneys, also found in the blood and lymph. *See* URINE, PATHOLOGICAL CHANGES IN (*Nitrogenous Constituents, Urea*). *See also* BALNEOLOGY (*Effects of Heat*); ECLAMPSIA (*Etiology*); ENZYMES (*Urea Ferments*); FÆCES (*Chemical Examination, Urea*); LIVER, PHYSIOLOGY OF (*Supply of Proteids*); MENSTRUATION (*Phenomena, Menstrual Wave*); NEPHRITIS (*Clinical Features of Types, Urine*); PHYSIOLOGY, BLOOD AND LYMPH (*Plasma, Chemistry of*); PHYSIOLOGY, FATE OF FOOD ABSORBED (*Liver in Relation to Proteins*); PREGNANCY, PHYSIOLOGY OF (*Changes in Urine*); SKIN, ANATOMY AND PHYSIOLOGY (*Function of Secretion, Sweat*); TEMPERATURE (*Changes in Fever, Injection of Urea*); URÆMIA; and URIC ACID.

Urechysis.—Effusion of urine into the areolar tissue of the body, *e.g.* in rupture of the urethra or bladder.

Uredo Fœtida.—A form of fungus, known also as *rust*, which makes flours and meals unfit for food.

Uremia. *See* URÆMIA.

Ureters. *See* KIDNEY, PHYSIOLOGY OF; KIDNEY, SURGICAL AFFECTIONS OF (*The Ureter*). *See also* BLADDER, INJURIES AND DISEASES OF; CYSTOSCOPE (*Method of Cystoscopy*); HÆMATURIA (*Causes, Catheterisation of Ureters*); PYURIA (*Diagnosis of Origin*); SPASM (*Ureteric Spasm*); TUBERCULOSIS (*Genito-Urinary*); URINE, PATHOLOGICAL CHANGES IN (*Significance of Albuminuria, Temporary Occlusion of Ureter*); VISCERAL PAIN (*Ureter*).

Ureteralgia.—Pain in the ureters.

Ureterectomy.—Excision of the ureter, *e.g.* in tubercular and suppurative ureteritis. *See* KIDNEY, SURGICAL AFFECTIONS OF (*Operations*).

Ureteritis.—Inflammation of the ureter. *See* KIDNEY, SURGICAL AFFECTIONS OF (*Ureter, Inflammation*).

Uretero-.—In compound words *uretero-* (Gr. οὐρητήρ, ureter) means relating to or belonging to the ureter, *e.g.* *uretero-cystostomy*

(formation of an opening between a ureter and the bladder); *uretero-enterostomy* (formation of a passage between a ureter and the intestine); *ureterolith* (calculus in the ureter); *ureteropyosis* (suppuration in the ureter), *ureterorrhagia* (hæmorrhage from a ureter), *ureterorrhaphy* (suture of the ureter), *ureterostomy* (formation of a ureteric fistula), and *uretero-vaginal* (belonging to the ureter and the vagina).

Ureterotomy.—Incision of the ureter, *e.g.* in stone in the kidney and ureter. *See* KIDNEY, SURGICAL AFFECTIONS OF (*Calculus, Treatment*); KIDNEY, SURGICAL AFFECTIONS OF (*Operations*).

Urethane.—Ethyl carbamate ($C_2H_5O.CO.NH_2$), a hypnotic drug which may be given in doses of 20 to 40 grains. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Treatment, Sleeplessness*).

Urethra, Diseases of.

INTRODUCTION 251
ANATOMY—
Male Urethra 251
Female Urethra 252
ABNORMALITIES 252
Hypospadias 252
Epispadias 252
INFLAMMATION—
Causes 253
Symptoms 253
Treatment 253
GONORRHŒA 253
Pathology 253
Course and Symptoms—
In Men 254
In Women 255
Complications 255
Sequelæ 255
Treatment—
In Men 255
In Women 256
TUBERCULOSIS 256
STRICTURE—
Varieties 257
Symptoms 257
Sequelæ 257
Diagnosis 257
Treatment 258
Instrumental 258
Operative 259
RUPTURE 260
PROLAPSE 261
TUMOURS 261
CALCULI 261
FOREIGN BODIES 262

See also ASEPTIC TREATMENT OF WOUNDS (*Urethral Operations*); CATHETERS, USES AND DANGERS OF; CYSTOSCOPE; ELECTRICITY (*Use in Stricture of Urethra*); FRACTURES (*Pelvis*); GONORRHŒAL INFECTION (*Urethra*); MICTURITION; PROSTATE GLAND; PYURIA; RECTUM, DISEASES OF (*Rectal Symptoms due to Urethral Diseases*); SCROTUM AND TESTICLE, DISEASES OF (*Scrotal Fistula, Causes*); URINE, BACTERIA IN (*Modes of Infection*).

INTRODUCTION.—The urethra or duct of the bladder differs both anatomically and physiologically in the male and female. It is more complex in the male, because it is the seminal channel, whilst in the female its only function is to discharge urine. The male urethra, therefore, is subject to a much greater number of pathological conditions than that of the female.

ANATOMY.—*The Male Urethra* measures 8 to 9 inches in length, and extends from the neck of the bladder to the urinary meatus. It is a continuous tube of mucous membrane, which is divided for descriptive purposes into the prostatic, membranous, and spongy or bulbous portions, but for the practical purposes of treatment into anterior and posterior parts, the anterior portion lying in front of the compressor urethræ, the posterior within and behind this muscle. The anterior urethra, therefore, extends from the meatus to the anterior layer of the triangular ligament, whilst the posterior urethra includes the membranous and prostatic portions. In a normal adult the posterior urethra begins at a point over 5 inches and under 7 inches from the meatus, for it is at this depth that an instrument is grasped by the compressor urethræ when it is being passed along the urethra. The urethra is a potential tube, for its walls are nearly in apposition, except during the passage of urine and semen. Its mucous membrane exhibits a number of pouches—the lacunæ of Morgagni—which are more numerous in the upper than in the lower wall; they are lined with cylindrical epithelium, and their orifices are directed towards the meatus. A blind pouch protected by a fold of mucous membrane—the valve of Guérin—lies just within the meatus in the upper wall of the fossa navicularis, and a surgeon always introduces a curved catheter into the urethra, with the point directed towards the floor of the canal in order to avoid this pouch. The ducts of Cowper's glands open upon the floor of the spongy portion of the urethra close to the membranous part. The openings are usually bilateral, and at the same level, but one duct sometimes opens in advance of the other, and occasionally they unite to form a single orifice. The membranous portion of the urethra is of great surgical importance, and, with the exception of the urinary meatus, it is the narrowest part of the urethra. It extends from the apex of the prostate to the bulb of the corpus spongiosum, and lies between the two layers of the triangular ligament, passing beneath the arch of the pubes, from which it is separated by the anterior fibres of the compressor urethræ, and

by the dorsal vessels and nerves of the penis. The urethra is curved in this situation, the lower wall being convex downwards and separated from the rectum by the triangular space of the perinæum. The mucous membrane of the membranous portion of the urethra is thrown into longitudinal folds, and contains numerous small mucous glands—the glands of Littré.

The prostatic portion passes from the bladder through the prostate from base to apex. It is about an inch and a quarter in length, and contains the verumontanum upon which are the openings of the utricle and the ejaculatory ducts, whilst on either side is the prostatic sinus into which open the many little ducts from the lateral lobes of the prostate. This portion of the urethra is the widest, and is capable of the greatest dilatation.

The Female Urethra is somewhat fusiform in shape, and in the adult is about an inch and a half in length. It opens within the vulva behind the clitoris, but in front of the vaginal orifice, the urinary meatus being the narrowest and least dilatable part of the canal. The inner surface of the urethra, as in man, presents numerous crypts of Morgagni, which are largest and most numerous near the meatus. There are also many urethral glands varying in complexity from simple depressions in the mucous membrane to true glands of the compound tubular type. Similar glands are found in the vestibule just outside the meatus, where they are called periurethral glands. The periurethral glands are scattered over the mucous membrane, but collectively they are the homologue of the prostate in the male, and calculi are sometimes formed in them. The canals of Skene are two small ducts which open upon either side of the posterior wall of the urinary meatus; they are present throughout life, but increase in size during pregnancy, and are said to attain their maximum size at the time of labour.

ABNORMALITIES. — The urethra is developed from the urogenital sinus by a complicated series of changes which allows plenty of scope for the production of various malformations. The changes are more complete in the male than in the female, and abnormalities are consequently seen more often in boys than in girls. Imperforate urethra, either partial or complete, absence of a part or of the whole urethra, double urethra, anomalous dilatation, pouchings of various kinds, and recto-prostatic communications are the less common malformations met with in practice. Hypospadias and epispadias are the most frequent and most important.

Hypospadias.—The condition of hypospadias is due to defective closure of the urethra, which may open on the under surface of the penis at any point between the perinæum and the glans penis. The slighter cases are the more common, for the hypospadias is generally limited to the anterior part of the penis, but the condition is often associated with considerable deformity of the penis, which is curved downwards during erection, or may be twisted upon itself in such a manner as to render it useless for sexual purposes. The glans, too, is often flattened; there is no frænum, and the prepuce is long, thickened, and hooded over the imperforate glans. Some of the native tribes in Australia purposely produce hypospadias by opening the urethra at the root of the penis to limit the population.

Treatment.—Operative measures are designed to remedy the deformity of the penis rather than to restore the defective urethra, for extensive plastic operations have been uniformly unsuccessful in my hands. An operation is performed most satisfactorily upon a patient aged 8 to 10 years, and when the orifice of the urethra lies just behind the corona the skin on the under surface of the penis is divided, and retracted until the urethra is freely exposed. The unclosed portion of the urethra is then dissected away, and the tubular portion is freed from its attachments until it can be drawn up and sutured in place of the defective portion which has been cut away; the skin is then brought together with interrupted sutures. In the more severe cases an attempt is made to remedy the deformity of the penis, and to render it capable of erection rather than to cure the hypospadias. And the patient is well content with the operation if this end is attained, especially if the nature of the operation be explained to him beforehand, since he is generally above the age of puberty. The skin on the under surface of the penis is divided, all bands of adhesion are dissected away without cutting into the corpora cavernosa, and the urethra is divided at the point where it forms a complete tube. The cut end of the urethra is then attached to the skin, so that the urethra opens on the under surface of the penis at a somewhat lower level than before; any redundant prepuce is removed at the same time, and the circumcision is completed in the usual manner.

Epispadias is that condition of the penis in which the urethra is defective dorsally. The fissure is often limited to the glans penis, but in the more severe cases the whole penile urethra remains as an open channel, a condition which may be associated with ectopia vesicæ. Little or nothing can be done to remedy these extreme cases, though the urethra has sometimes been drawn through the scrotum partially to close the canal. In other cases I have separated the mucous membrane from the skin along each side of the cleft, and have afterwards sutured it with fine catgut over a black rubber catheter of appropriate size, the edges of the skin being then brought together with sutures of silkworm gut. The anterior part of the epispadias may be closed in this manner, but the

posterior part usually remains open even after several subsequent operations.

INFLAMMATION.—Inflammation of the urethra, like inflammation elsewhere, is due to a variety of causes, some mechanical, but the majority due to the action of micro-organisms. The mechanical causes leading to urethral inflammation, both in the male and female, are injury produced by foreign bodies passed along the urethra from without, or by calculi, whole or in fragments, passing from within. Irritating urine may produce urethritis, either because it contains crystals as in oxaluria, or because it holds in solution such drugs as cantharides, turpentine, or squills. Urethritis, too, may be a troublesome symptom in gout, rheumatism, and herpes. The introduction of dirty sounds, catheters, and other surgical instruments is a fertile source of a urethral inflammation, which may become purulent, and may be attended with serious and even fatal results in an unhealthy person. Reference has already been made to urethral inflammations produced by syphilis (vol. ix. p. 461), and by soft sores (Art. "VENEREAL DISEASE," vol. x. p. 427).

The symptoms in these cases of urethritis consist in a smarting pain, when urine is passed, and the appearance of a discharge which is either serous or purulent.

The treatment consists in removing the cause, and rendering the urine as little irritating as possible. The patient may be ordered lactate of soda, 10 grains of urotropin, dissolved in half a pint of water, the dose to be repeated three times a day; or he may be given a mixture containing potassium nitrate, 10 grains; bicarbonate of potash, 20 grains; tincture of henbane, 30 minims; and chloroform water to an ounce. Simple and unirritating urethral injections will also afford relief, *e.g.* a teaspoonful of Condy's fluid in a pint of hot water, or 10 grains of subnitrate of bismuth in an ounce of water at 100° F.

GONORRHŒA.—A clap is the commonest form of venereal disease affecting the male urethra. It is an infective inflammation of the urethral mucous membrane, due to the inoculation of the micrococcus gonorrhœæ, or gonococcus, first described by Neisser in 1879. The primary infection in both sexes occurs commonly at some part of the urethra; in the male at the lacuna magna, rapidly spreading to the bulbous portion, or to the prostatic part of the urethra; in the female the anterior part of the urethra, the periurethral glands, the glands of Skene and of Bartholin are chiefly affected at an early period, whilst the cervical canal of the uterus is a usual seat of inoculation, the vagina only becoming affected by the extension of the suppurative inflammation.

Pathology.—Gonococci, like the poison of syphilis (vol. ix. p. 460), can only be inoculated upon the human subject, for no experiments hitherto have succeeded in transmitting either gonorrhœa or syphilis to animals. The gonococci are minute organisms shaped like coffee-beans, which can only be studied under the highest powers of the microscope (one-twelfth immersion lens and upwards). They are generally arranged in pairs with the flattened or concave surfaces adjacent to each other, but they multiply rapidly by fission, so that tetrads and multiples of four are frequent in the colonies. The organisms are always pyogenic as they occur in the urethra, though it is clear that they are not necessarily associated with the formation of pus, because when they attack the tissues secondarily, as in gonorrhœal synovitis, suppuration is often absent. The gonococci gain access to the tissues through abrasions of the superficial epithelial cells, and are passed along the superficial intra-epithelial lymphatic plexuses until they reach a leucocyte. The white corpuscles seem to have a special affinity for them, and the gonococci multiply much more readily in their substance than in the protoplasm of the epithelial cells. The micro-organisms do not penetrate at first beyond the layer of unstriated muscle which bounds the urethral mucous membrane, but the irritation which they cause produces a serous exudation in the surrounding connective tissue and a loosening and detachment of the neighbouring epithelial cells. Pyogenic organisms then add their destructive agency to that of the gonococci, and the connective tissue becomes infiltrated with pus whilst the capillaries may be thrombosed.

The diagnosis of gonococci must be undertaken with due caution, for Mr. Foulerton has shown that eighteen different species of micrococci have been isolated from the healthy or diseased urethra in the male in addition to the ordinary pyogenic cocci, and a mistake in diagnosis may lead to irreparable disaster. The three characteristics by which gonococci are known are:—(1) The occurrence in the pus cells from the urethral discharge of paired organisms shaped like coffee-beans; at least four pairs should be recognised in each cell. (2) The typical cocci should be readily decolorised when treated by Gram's method. (3) Pure growths cannot be subcultured on gelatin at 20° C., or on agar either at 20° C. or at 36° C., since the other micrococci from the urethra are said to grow at these temperatures and on these media.

Preparation and Staining of Film.—Take two clean square cover slips and place a minute drop of the secretion to be examined upon one slip. Place the other cover slip accurately upon it, and then slide the two apart to obtain a couple of "smear preparations." Place the cover-glasses edgeways until they are dry, and then put them for fifteen minutes into a mixture of equal parts of absolute alcohol and ether to coagulate the albumin and allow the film to be

stained and washed without destruction. Filter on to each film surface sufficient anilin gentian violet (which can be bought ready-made or in soloids) to completely conceal the cover-glass. Pour off the stain in fifteen minutes, dip each cover-glass for an instant into water, and then pour on Gram's solution of iodine (iodine one part; potassium iodide two parts; distilled water 300 parts). Pour off the iodine in two minutes, and, without further washing, place the cover-glass in a watch-glassful of absolute alcohol until no more violet colour comes away, when the cover-glass is drained on a piece of white filter paper, but not necessarily until the film is decolorised. This usually takes from five to ten minutes, and it may be necessary to employ a second watch-glassful of spirit. Wash in water, dry, and mount in Canada balsam, or counterstain for forty-five seconds in a saturated aqueous solution of Bismarck brown, diluted with three times its volume of water. The pus corpuscles may be stained with eosin and the gonococci with Löffler's methylene blue by putting a film into methylene blue for one minute, washing till most of the blue disappears; counterstaining in a watery solution of eosin for twenty to thirty seconds; washing, drying, and mounting in Canada balsam. The preparation in each case must be examined with an immersion lens.

Course — In Men. — Gonorrhœa is nearly always caught by direct infection and usually during sexual intercourse. The incubation varies from three to five days, though it may be as short as a few hours, and is said to have been as long as a fortnight. The poison is less easily inoculated than that of soft sores, and its incubation period is longer (*see* Art. "VENEREAL DISEASE," vol. x. p. 427).

The earliest symptoms are those of a simple urethral inflammation, a sense of heat and itching in the glans penis, with increased sexual excitability and smarting on passing urine. These symptoms increase in intensity, until in twenty-four hours the urethral mucous membrane becomes swollen and begins to discharge a secretion which is at first serous but soon becomes purulent. The acute stage is marked by painful erections which may culminate in chordee, when the penis is abnormally curved or bent during erection as a result of plastic exudation into the corpus spongiosum or into one of the corpora cavernosa. Vesical tenesmus and increased frequency of micturition are also frequent and troublesome symptoms. The acute stage lasts about a fortnight, and is often associated with much mental depression. It may be complicated by cystitis and by a sharp and painful inflammation of the epididymis. As the acute stage gradually subsides the discharge becomes thinner, scantier, and clearer, until it either disappears altogether or is merely present as a drop at the meatus on first waking in the morning. An acute attack of gonorrhœa may thus run its course in five to eight weeks, and although this occasionally happens, it is much more usual for the inflammation to undergo a series of relapses which greatly retard recovery, or it becomes chronic as a gleet. The prognosis, therefore, is not very good as regards a speedy cure, though with care and attention there is but little doubt that a gonorrhœa can be wholly cured.

The patient needs to be warned on several points in regard to his cure. He recognises clearly enough that gonorrhœa is contagious, but he does not always understand that it is auto-inoculable. He should be told, therefore, of the danger of carrying infection to his eyes or mouth by rubbing them when his fingers are soiled with the urethral discharge. He must also be told that relapses are produced by very slight indiscretions in eating and drinking, as well as by every departure from strict continence. The dangers of marrying with an uncured gonorrhœa, even though it be very chronic, should be pointed out, and he should be made aware of the fact that it is extremely difficult to determine when a cure has been effected. The gonococci have a remarkable faculty of lying dormant until they are roused into activity by any cause leading to an increase in the blood or lymphatic supply of the urethral mucous membrane. They are especially liable to remain in the follicles of the prostate, so that the prostatic threads passed at the end of micturition should always be carefully examined, and no patient should be pronounced free from gonorrhœa until a careful bacteriological examination has been made of the urethral discharge produced by the injection of a solution of nitrate of silver. The patient is first asked to pass his urine, and 10 drops of a 4 per cent solution of cocaine hydrochlorate are then injected into the urethra. Five minutes later a solution of nitrate of silver (20 grains to the ounce) is injected into the urethra, the penis being compressed at a point 2 inches behind the meatus, to prevent the fluid passing into the bladder. The solution is retained for a few seconds before it is allowed to escape, and the injection is thrice repeated, after which the penis is washed in warm water. The nitrate of silver causes an acute but temporary desquamative urethritis with considerable discharge, and if the patient is to be pronounced free from gonorrhœa, the discharge should not contain any gonococci. A more rough and ready way of endeavouring to ascertain whether a patient is cured of gonorrhœa is to ask him nearly to empty his bladder. The prostate is then manipulated through the rectum, and the remainder of the urine is then passed. This residue will contain a considerable quantity of the prostatic secretion, and if this is free from gonococci, the patient is perhaps cured.

In Women.—The symptoms of acute gonorrhœa are usually much less severe in women than in men, but the sequelæ and complications are more serious. The inflammation begins as a urethritis which soon involves the vulva and the uterine cervix. The bladder often becomes inflamed quite early in the acute stage in consequence of the shortness of the female urethra.

The symptoms chiefly complained of are tenesmus and scalding during micturition, and an examination of the vulva shows it to be in a state of acute inflammation with a purulent discharge from the orifice of the urethra. The acute stage passes off in the course of a few days, when the inflammation tends to become chronic. The chronic state lasts many months, and as it gives rise to few symptoms it may pass almost unnoticed. Pressure through the vagina along the course of the urethra will cause a little exudation of muco-pus which may contain gonococci in the earlier stages, though it is often sterile when the discharge is of long standing. Gonorrhœa in some women appears to pass at once into a chronic state without any markedly acute symptoms, so that when the patients afterwards suffer from chronic pelvic inflammation, they are unable to assign a definite beginning to their trouble.

In female children gonorrhœa takes the form of a vulvo-vaginitis, though it should be understood quite clearly that every discharge from the vulva and vagina of a little girl is not due to gonorrhœa.

Complications.—The complications of gonorrhœa common to both sexes are cystitis, though the symptoms of this trouble in men are often due to an inflammation of the posterior urethra (p. 251), the bladder itself not being involved; lymphangitis; bubo; urethral abscess; synovitis, which may lead to disorganisation of the joint, but does not usually do so; tenosynovitis, flatfoot; conjunctivitis, endocarditis, more rarely pericarditis or myocarditis, and meningitis; gonorrhœal peritonitis affects women more often than men; pyæmia is somewhat more frequent in men than in women, possibly because it is not very unusual to have a septic thrombosis in the prostatic plexus of veins.

The complications occurring in men are balanitis or inflammation of the surface of the glans penis; phimosis; paraphimosis; acute and chronic inflammation of Cowper's glands, of the prostate and of the vesiculæ seminales; epididymitis, and more rarely true orchitis, or inflammation of the body of the testicle.

The complications peculiar to women are inflammation of the numerous glands lying in the immediate neighbourhood of the urethra: chief amongst these are Skene's glands; the endo- and para-urethral mucous follicles; the vestibulo-vaginal glands and the glands of Bartholin. The gonococci often linger for long periods in these recesses, where they give so little trouble that an apparently healthy woman is able to spread the disease through their secretions. The more serious complications are associated with acute and chronic inflammation of the pelvic organs and of the connective tissues surrounding them, foremost amongst these being metritis, salpingitis, perimetritis, and pelvic cellulitis.

The sequelæ common to men and women alike are sterility; stricture of the urethra, and less commonly of the ureters; hypertrophy of the bladder and pyelitis; urinary abscess; extravasation of urine; urinary fistula.

Treatment.—The treatment of gonorrhœa is both important and difficult. In an ideal state the early and happy marriage of every male would abolish the disease in a single generation; but as this is impracticable, it should be remembered that a diseased woman is more likely to infect a drunken man, and one who has repeated connection than another who takes his pleasure more soberly; that one attack of gonorrhœa predisposes to others, and that even slight sexual indulgence, though it be with a healthy woman, is able to reawaken the acute symptoms in a man who has only been partially cured of the disease. Gonococci deposited upon a mucous membrane are more likely to gain access through an abraded than through a healthy surface, but they do not pass into the lymphatics instantaneously. Washing and micturition immediately after a suspicious coitus may therefore remove them; injections are less to be relied upon, for they may wash the micro-organisms into the urethra, and may act adversely by injuring the surface epithelium of the urethral mucous membrane.

The first symptoms of infection show that the time has arrived for the adoption of more active measures, and it may be premised that those who can rest and modify the routine of their lives are more amenable to treatment, and are less likely to suffer from complications and sequelæ, than those who continue their usual avocations. The hygiene of the acute stage consists in bodily rest, low diet, the avoidance of stimulants, regulation of the bowels, and cool sleeping-rooms. The urethra should be freed as far as possible from the secretion, and where the older surgeons were content to effect this object by flushing and by stimulating injections, modern surgeons endeavour to soothe the inflamed parts and to render the discharge sterile. Nitrate of silver was formerly used to a large extent in the treatment of gonorrhœa from its earliest to its latest stage; but various organic compounds of silver are now being adopted with beneficial results. Protargol is the most widely used, itrol or citrate of silver, and albargin, a preparation of silver nitrate and gelatose, are more recent forms. The advantages claimed for these salts over the nitrate of silver are that they are less painful; that they are markedly destructive

to gonococci, and that they penetrate the urethral mucous membrane without injuring it or causing irritation. The disadvantages are the instability of the substances which causes them to decompose on warming and even on exposure to light.

Injections should be ordered as soon as the discharge is recognised. They should consist of a half per cent solution of protargol given thrice daily, the morning and mid-day injections being retained for ten to fifteen minutes, and the evening one for twenty to thirty minutes. These injections may be increased in strength to 3 per cent of protargol, and they are to be continued until the discharge is free from gonococci. The protargol is then to be replaced by an astringent injection, *e.g.* subnitrate of bismuth, 10 grains; tincture of opium, 10 minims; tincture of catechu, 15 minims; distilled water to three ounces. Urotropin to the amount of 10 grains, three times a day, dissolved in half a pint of water, may be given throughout the disease, replacing the older treatment by copaiba and sandal-wood oil.

Chronic gonorrhœa used to be called gleet, but the symptoms are now known to be associated with a chronic inflammation of the anterior or posterior urethra. The morning drop at the urinary meatus is often the only symptom of chronic anterior urethritis, though the urethroscope shows numerous localised patches of hyperæmia or erosion even as low as the bulbous portion of the canal, whilst the passage of a sound may demonstrate a stricture of large calibre.

The treatment of chronic anterior urethritis consists in healing the erosions, curing the catarrh, and dilating the commencing stricture.

Chronic posterior urethritis shows itself by the recurrence of the discharge after apparent cure without sufficient cause; by attacks of epididymitis long after the period of infection; by prostatic inflammation and spasm of the compressor urethræ muscle many months after the cessation of treatment; by pain of a vague and neuralgic character in the genito-urinary tract, and by sexual hypochondriasis, which is often so marked that Ricord is reported to have said jestingly he expected the retribution for his ill-spent life would be the peopling of his everlasting abode with importunate patients suffering from a gleet with this symptom.

The treatment of posterior urethritis consists in reawakening an acute inflammation in the affected part in the hope that the thickened tissues may thus be restored to their natural condition. This is best effected by the local application of silver nitrate, sulphate of copper or thallin, the latter in a 2 per cent solution. The application must be applied locally and skilfully, neither in front of the compressor urethræ nor too far back, lest it enter the bladder. The nitrate of silver and sulphate of copper solutions vary in strength from 1 to 10 per cent, according to the tolerance of the patient. When the prostate is involved the patient should be placed recumbent. The meatus and glans penis are then cleansed with a solution of protargol, the anterior urethra is filled with a $\frac{1}{2}$ to $\frac{3}{4}$ per cent solution of cocain, after which from five to ten ounces of protargol solution should be injected into the bladder to be afterwards passed voluntarily by the patient. The injection may be repeated three times a week unless there are symptoms of local inflammation.

Treatment in Women.—The patient should be kept in bed on a milk diet during the acute stage, the bowels being regulated by saline aperients. The genital organs at first should be irrigated frequently with warm solutions of boroglyceride (which is prepared by heating together in a water bath 2 parts of boric acid and 3 parts of glycerine) of the strength of a drachm to the pint. Injections may be used as soon as the acute symptoms have subsided. A 4 per cent solution of protargol: 1·5000 of biniodide of mercury or 1·4000 of formaldehyde form useful and efficient injections. They should be employed whilst the patient lies upon her back, and when mercurial injections have been used the vagina should afterwards be washed out with a warm salt solution, because in many women there is a tendency for a pool of solution to remain in the vagina so long as they lie on their backs, and from this pool an undesirable amount of absorption may take place. The vaginal douche, however, is only an accessory to the thorough application of a strong solution of protargol or of nitrate of silver to the whole of the affected mucous membrane. This application is made by placing the patient in a semi-prone position, and using a duck-bill speculum. The mucous membrane is wiped clean, and the silver nitrate solution of the strength of 20 to 60 grains to the ounce—or of a 10 per cent solution of protargol—is applied with a swab. Especial attention is devoted to the urethra, Skene's glands (p. 252), the ducts of Bartholin, and the cervix uteri. The urethra and canal of the cervix are best treated by instillation of the solution: Skene's and Bartholin's glands by injection through a small syringe with a slender nozzle. A 5 to 10 per cent solution of ichthyol glycerine may be applied on pledgets of cotton-wool, which should be left *in situ* during the intervals between the application of the germicidal solution. Curetting and douching of the uterine cavity should only be employed in the most advanced and chronic stages of the disease, lest the local inflammation set up by the operative measures should lead to the extension of the inflammation to the Fallopian tubes and ovaries.

TUBERCULOSIS OF THE URETHRA.—Tuberculosis of the urethra is always secondary to infection

of other parts. It affects the urethra either as an inflammation of the mucous membrane—urethral tuberculosis—or as an inflammation of the connective tissue surrounding the urethra as it lies in the perinæum, or more strictly localised to the prostatic urethra tuberculous periurethritis. As in other mucous membranes the tubercular inflammation manifests itself in the form of chronic inflammation, miliary tubercles, shallow but circular ulcers, or caseating deposits. The inflammation has occasionally been followed by stricture. The symptoms are those of an ordinary chronic urethritis: a mucopurulent discharge, increased frequency of micturition, sometimes with pain and sometimes associated with the passage of blood. Treatment consists in administering cod-liver oil, the ordinary hygienic measures now in use for the treatment of phthisis, and the removal of the discharge by unirritating urethral injections. Urotropin may be administered to keep the urine acid (grs. x three times a day in half a pint of water), and iodoform bougies are often of service in the local treatment of the urethritis.

STRICTURE OF THE URETHRA.—True stricture of the urethra—the *organic stricture* as it is often called—is a narrowing of the urethra caused by a poliferation of inflammatory cells in the submucous tissue, the cells becoming converted into scar tissue which afterwards shrinks. Any chronic inflammation of the urethra therefor may lead to a stricture, and the commonest forms of inflammation are gonorrhœal; injury due to external violence, to the passage of instruments or calculi along the urethra; soft sores, chancres, and phagedæna. Acute retention of urine is sometimes caused by a temporary narrowing of the canal due to inflammatory swelling of its walls—*inflammatory stricture*—and sometimes from spasm of the muscles surrounding the canal—*spasmodic stricture*. Inflammatory stricture is the result of acute urethral inflammation, of direct injury to the mucous membrane, and it is often associated with an old-standing organic stricture. Spasmodic stricture is usually reflex in character, and it causes the retention of urine so often seen after the operation for piles, and after injury to the perinæal region even though the urethra has not been hurt. Like the inflammatory stricture, this reflex spasm of the urethral muscles is often associated with an organic stricture.

Symptoms.—The symptoms and their severity vary greatly in different people, but the most marked, and that which most often brings the patient to a surgeon, is some alteration in the flow of urine. The stream, instead of passing at once in a full flow and stopping completely, presents various modifications. It is often smaller than usual, and may be twisted or forked. The act of micturition takes longer, it is more frequent, and it is painful. The last few drops of urine can only be expelled by straining, and even then the urethra is not completely emptied, for the patient complains that the urine dribbles away, and that his trousers get wet if he is at all in a hurry. There is often a slight muco-purulent discharge from the meatus associated with the presence of erosions of the urethral mucous membrane in the immediate neighbourhood of the stricture. The outflow of semen is retarded in the same manner as that of the urine, and various difficulties arise during intercourse which lead in many cases to a condition of sexual hypochondriasis.

Sequelæ.—Many strictures remain untreated, sometimes because they are unrecognised, but usually because the patient is too careless to undergo the prolonged course which is necessary to cure them. The sequelæ, therefore, are numerous and well recognised. The urethra immediately behind the stricture becomes dilated and ulcerated; a calculus may be lodged in this portion of the urethra, or it may yield and allow of the extravasation of urine into the scrotum and beneath the deep layer of the superficial abdominal fascia. More frequently, however, only a few drops of decomposing urine leak through the softened mucous membrane, and a perinæal abscess is formed leading to a urinary fistula.

The prostate may become congested by the chronic inflammation, and it may either be hypertrophied or suppurate.

The urine does not pass freely out of the bladder, which in consequence is often incompletely emptied. The urine decomposes, and a condition of chronic cystitis is set up with a deposit of mucus and phosphates which may give rise to such discomfort as to require the performance of a perinæal section. The additional force needed to expel the urine through the narrowed urethra is obtained by hypertrophy of the muscular walls of the bladder, but eventually this hypertrophy ceases, and the bladder becomes thinner whilst its mucous membrane is pouched, the pouches often acting as traps where small calculi may grow into larger ones.

The hypertrophy of the bladder prevents the easy passage of urine along the ureters, which in consequence become dilated and tortuous, whilst the backward pressure acting upon the pelves of the kidneys causes them to dilate. Hydronephrosis results, and if the urine becomes purulent a multiple suppurative nephritis or surgical kidney ends the life of the patient.

Diagnosis.—The only means of determining whether a patient has a stricture is to pass an instrument, and the proper instrument to pass is a bougie and not a catheter. The acorn-headed black bougies are the best for exploratory purposes. A No. 12 English should first be passed, and care should be taken that it is

absolutely clean, quite supple, and well lubricated. Glycerine or castor oil will be found more useful than vaselin or lanolin. The patient should be recumbent on a couch, the surgeon standing upon his right side. The instrument is passed onwards deliberately, continuously, and without using any force, and it should be withdrawn as slowly and gently as it has been passed. The instrument will pass into the bladder with the greatest ease if there is no stricture, though the patient may complain of a little smarting or even faintness as it passes through the triangular ligament, and of a desire to pass water when the end of the bougie touches the trigone. If a stricture be present the bougie will be more or less completely arrested during its passage, the actual seat of arrest being situated most often at the bulbo-membranous part of the urethra, the next commonest seat of stricture being within two inches of the urinary meatus; whilst the prostatic urethra is only strictured as a result of direct injury. Strictures are often multiple, and when this is the case the posterior stricture is usually tighter than the anterior. An attempt should be made to ascertain the size of the stricture as soon as its position has been determined. This estimation is made by passing a black olivary bougie of large size, and replacing it by successively smaller sizes, until an instrument can be passed through the stricture; and it is always advisable to begin with a large bougie and work downwards rather than to begin with a small size and work up to bigger ones, since by this method it is easy to underestimate the size of a stricture, because the passage of successive instruments causes spasm or congestion of the injured urethra.

Treatment.—A stricture sometimes leads to acute retention of urine, even though it has not previously been troublesome. Such a condition is readily caused by anything which will produce a temporary congestion of the urethral mucous membrane or a reflex spasm of the muscles surrounding the urethra, or even of the muscularis mucosæ itself. A hot bath and a full dose of Dover's powder will often enable a patient to pass his water in such cases, but if these measures fail, the passage of a No. 8 or 9 black olivary catheter can generally be readily effected if the instrument be passed gently but continuously and rather quickly along the urethra, for any jerky movement or any bungling or uncertainty on the part of the surgeon will increase the spasm.

The broad principles to be grasped in the treatment of an organic stricture are, first, that the canal of the urethra be sufficiently enlarged to allow of the free flow of urine, and, secondly, that its calibre should afterwards be maintained. The urethra is restored to a sufficient size either by instrumental dilatation or by division of the strictured portion. Dilatation is the better method in most cases, though it has well-marked limitations. Some patients are quite unable to tolerate the passage of instruments; in others the stricture contracts again as quickly as it is dilated — *resilient stricture*; other strictures are so firm and gristly as to be incapable of dilatation, whilst others again are practically impermeable. As a general rule strictures situated within the meatus are better divided than dilated. Dilatation is performed either gradually or continuously; gradual dilatation is the method most often employed at the present time, because it does not oblige a patient to discontinue his ordinary avocations. A clean black olivary bougie is passed through the stricture, of as large a size as can be introduced, and is immediately withdrawn, the patient being told to come again for a second sitting after an interval of five or seven days. At the next interview a bougie of the same size as the one last passed is again introduced, and when it is withdrawn it is replaced by the next size larger. A similar method is followed on successive occasions, until a No. 10 English can be readily passed into the bladder. Further dilatation to admit a No. 15 or 16 English is accomplished by the use of a highly polished and conical steel sound, whose curve is graduated through four sizes of the French gauge from the point to the thickest part. The patient should be told that it may take from five to six months to dilate a moderately tight stricture, but he has the satisfaction of knowing that when the dilatation is fully accomplished the occasional passage of a full-sized instrument will be sufficient to prevent recontraction. The directions I usually give to a patient who has been treated by gradual dilatation are to pass a bougie every Sunday morning for three months from the time he last sees me; on every alternate Sunday for a further period of six months; then once a month for nine months; and finally on every quarter day, but he is not to feel satisfied unless he can continue to pass the full-sized instrument on each occasion. The rationale of the cure is not fully understood, though there is little doubt that it acts by altering the blood and lymph supply in the affected part of the mucous membrane.

Sizes of Catheters and Bougies.—The sizes of English catheters and bougies are purely empirical, for they differ slightly according to the maker. They are numbered from 1 to 12, and correspond roughly with Nos. 3 to 21 of the French scale. The filière Charrière of the French notation is the more satisfactory, as it is based on a scientific system. It extends from 1 to 30, each number denoting the size of the circumference of the instrument in millimetres, thus No. 1 catheter or bougie measures one millimetre or one-twenty-fifth of an inch round, No. 6, six millimetres, and so on.

Continuous dilatation is less often employed.

It consists in putting the patient to bed, introducing a catheter into the bladder, and tying it in for twenty-four hours, when it is replaced by another two or even three sizes larger than the first. It is often more comfortable for the patient if the catheter first tied into the bladder be a size smaller than the stricture admits readily. The catheter must be removed if the patient has a rigor and if, without a shivering fit, he becomes feverish. The end of the catheter should only lie just within the bladder, and for this reason the ordinary red or black rubber catheters are better than the olivary catheter whose eye is necessarily at some distance away from the end. The urethra should be gently irrigated with a 1 : 10,000 solution of biniodide of mercury, at a temperature of 100° F., every time the catheter is changed. The treatment usually lasts a fortnight, and the patient is confined to his bed. The after-treatment is the same as after the continuous method. Continuous dilatation is used when the interrupted method has failed, when the patient has only a limited time at his disposal, as in the case of an officer on leave or a mate at home between two voyages, when the stricture is complicated with a cystitis which renders it advisable to drain the bladder, and as a preliminary to internal urethrotomy.

The operative treatment of stricture is conducted on the same lines as that of dilatation. The urethra is restored to its natural size by division of the constriction, and it is afterwards maintained at this size by the passage of instruments. The mere division of a stricture without adequate after-treatment is useless. Two methods of operative treatment are in use at the present time: division of the stricture from within the urethra or internal urethrotomy, and division by cutting through the penile or perineal structures from without—external urethrotomy. Both methods have their strong advocates, but neither can be employed to the exclusion of the other. Internal urethrotomy requires that the stricture shall admit some instrument as a guide to the urethrotome, whilst external urethrotomy compels the patient to remain in bed for a considerable length of time.

Internal Urethrotomy.—The cases suitable for internal urethrotomy are those in which the stricture is situated close to the urethra, narrow and well-localised strictures, *e.g.* annular and bridle strictures, strictures in which dilatation has been tried and failed, either because they are resilient (p. 258), or because they are too fibrous or gristly. It is undesirable to operate in this manner upon patients who have an infective urethritis.

Internal urethrotomy is performed according to Civiale's method, in which a concealed knife is passed through the stricture, and the constriction is divided from behind forwards, or it is done by Maisonneuve's method, in which the incision is made from before backwards by a blade running in a metal guide, the knife being passed along a groove in the guide which is first introduced through the stricture.

When Civiale's method is adopted the stricture is continuously dilated until a No. 5 English catheter can be passed. This catheter is left in until the time of the operation when it is withdrawn, and the urethra is carefully cleansed by irrigation with a solution of biniodide of mercury (1 : 5000) at a temperature of 100° F. The sterilised urethrotome is passed along the urethra until the surgeon is satisfied that it has entered the bladder. The instrument is withdrawn until the bulb touches the posterior edge of the stricture, the cutting edge of the knife being directed towards the floor of the urethra. The blade is then projected to the required distance, and is drawn smartly through the stricture until it is fully divided, when the knife is retracted into its sheath before the urethrotome is withdrawn. If the strictures are multiple they are all divided at the same sitting. A steel director is afterwards passed along the urethra to determine whether the strictures have been satisfactorily divided, but not to dilate, for if it does not pass easily, the urethrotome must be reintroduced. The bladder is then washed out with a saturated solution of boric acid, at a temperature of 110° F., and the patient is directed to retain his urine if possible for a period of eight hours.

If Maisonneuve's method is adopted it is usual to employ Tevan's modification. A filiform guide-bougie with a screw top is first passed through the stricture, and the thin grooved staff of the urethrotome is screwed on to it and passed on through the stricture. The staff is held steadily, and the *bistouri caché* is passed along the groove until its hood is arrested by the stricture. The constriction is then divided by a series of short cuts with the knife, the blade being allowed to recoil between each cut until the stricture is divided. The subsequent steps are the same as in the previous operation. In either case it will be sufficient to begin dilatation with a catheter on the tenth day after the operation.

External Urethrotomy.—The methods chiefly employed are those of Syme and Wheelhouse, and of these I prefer Wheelhouse. External urethrotomy is required when the stricture is practically impassable, when the stricture is narrow and the bladder requires draining in consequence of long-standing cystitis having filled it with foul urine and deposits of phosphates and mucus. The method is also serviceable when the stricture is complicated with false passages and urinary fistulæ.

In *Wheelhouse's operation* the patient is prepared in the usual manner, his skin at the seat of operation being made as sterile as possible.

He is anæsthetised, and placed in the lithotomy position, Wheelhouse's staff being passed along the urethra until its point touches the stricture, the groove on the staff looking towards the floor of the urethra. The surgeon cuts down upon the groove, making a skin incision of about an inch and a half in length. The assistant then rotates the staff to engage its hook in the upper angle of the wound, and draws it upwards to stretch the urethra in this position. A pair of pressure forceps are placed upon each edge of the wound to act as retractors, and by this means the urethra immediately in front of the anterior end of the stricture is converted into a lozenge-shaped space. The surgeon takes a fine probe and carefully explores the lower angle of this lozenge to ascertain the position of the urethra, and he may have to explore many false passages before he discovers it. When it is found he passes a probe-pointed director onwards until it lies freely in the urethra beyond the stricture. The stricture is divided by a straight bistoury passed along the groove in the director, and when the division is complete a probe-pointed gorget is pushed along the urethra into the bladder. A full-sized silver catheter is passed through the meatus over the gorget into the bladder, the gorget is withdrawn, the pressure forceps are taken off, and a dressing is applied to the wound which is left unsutured. The catheter is tied into the bladder, but its end is not plugged, as it is important that the urine should run away freely from the bladder. The catheter is retained for three or four days, and after its removal a steel sound is passed every third day until the external wound is completely healed. The after-treatment of the case is the same as that described at page 259.

In Syme's operation the patient is anæsthetised, and the staff in passed before the patient is put into the lithotomy position. The narrow terminal portion is passed through the stricture until its shoulder is stopped, the patient is then trussed, an incision is made exactly in the median line, and is carried through the urethra until the staff is exposed. The stricture is divided by passing a bistoury along the groove in the thin part of the staff. The staff is withdrawn, and a full-sized catheter is passed into the bladder, if necessary with the aid of a blunt-pointed gorget in the wound. The subsequent treatment is that described above (p. 259). It often happens that the patient's condition renders it impossible to keep a catheter in the bladder, and in such cases a piece of catheter may be inserted into the bladder through the perinæal wound, special precautions being taken to prevent the drain dropping into the bladder, as it may then be lost sight of and form the nucleus of a vesical calculus long after the operation.

Cock's operation opens the urethra behind the stricture and without a guide, by cutting accurately in the middle line of the perinæum, whilst the operator has the index finger of his other hand in the rectum. The urethra is opened in front of the prostate and a catheter is passed into the bladder through the wound, is cut short and tied in with tapes. Mr. Jacobson points out that this operation is not easy for one who only practises it occasionally, and that it is a more severe operation than the size of the wound would suggest.

Strictures of the urethra have sometimes been enlarged by forcible splitting, and in other cases electricity has been employed for the purpose of electrolysis, but both these methods have now fallen into disuse.

Rupture of the Urethra.—The urethra is readily ruptured by such direct violence as falling astride a bar or from a kick in the perinæum, but it is sometimes the result of indirect violence or it is a complication of fractured pelvis. The rupture is either partial or complete, and may coexist with a mere contusion of the perinæum.

The symptoms are generally severe pain, with greater or less difficulty in passing water. A tense and tender swelling forms in the perinæum, and there may or may not be bleeding from the orifice of the urethra.

The treatment in the slightest forms should be expectant, as it is possible that the urethra may only be bruised. The patient is put to bed, and the swollen perinæum is carefully watched to see that no extravasation of urine or suppuration takes place. The perinæal hæmatoma should be incised if the temperature rises or if there is evidence of extravasation. More usually, however, there is no doubt as to the character of the injury, and the surgeon should then make an immediate attempt to pass a full-sized silver catheter, which should be tied in if he is so fortunate as to be successful. A median perinæal incision should be made if the rupture is associated with much bruising. When it is impossible to pass a catheter the patient should be placed in the lithotomy position, and a grooved staff should be passed along the urethra until it reaches the seat of injury. An incision is then made in the middle line until the staff is exposed, and the ruptured urethra can be explored. It is easy enough to find the anterior end of the torn urethra through which the staff projects, but it may be extremely difficult to find the posterior portion. The search, however, may be rendered easier by remembering that the posterior portion of the urethra in such cases usually lies nearer to the anus than the penile part, whilst pressure upon the bladder may cause a few drops of urine to trickle from the torn end. A catheter should, if possible, be passed from the meatus into the bladder; but if this is impossible the wound may be left unclosed and lightly packed with gauze, for the bladder will tend to empty itself through the torn end of the urethra without any dangerous

distension, and a catheter may be passed after a few days' interval. The use of hot fomentations to the perinæum will often be a useful dressing, as it tends to promote the flow of urine. Suprapubic drainage of the bladder may be performed if it is really necessary, the drainage being maintained for about ten days until a catheter can be passed through the urethra.

The prognosis of ruptured urethra, even in its slighter forms, is extremely bad, for the laceration nearly always leads to a most intractable and deeply seated stricture, which makes it necessary for the patient to maintain instrumental dilatation for many years, or even during the rest of his life.

PROLAPSE OF THE URETHRA.—Prolapse of the urethra is not uncommon in little girls as a result of general weakness, local irritation at the neck of the bladder and repeated straining due to a chronic cough or habitual constipation. It is occasionally met with in elderly women associated with senile changes and laxity of the genital passages, and it is seen as a rare accident in healthy women during middle life. The prolapse is generally chronic, but it may be acute.

The symptoms are increased frequency of micturition, with scalding, and the passage of a little blood. Examination of the vulva shows the presence of a red mass which may be furrowed longitudinally, and presents a central depression forming the orifice of the urethra. The prolapse may be mistaken for a nævus, polypus, or urethral caruncle.

Treatment.—Simple replacement is often sufficient to cure the prolapse, especially if the parts be regularly washed with cold water. The occasional application of solid nitrate of silver to the mucous membrane causes sufficient constriction to prevent recurrence in many of the chronic cases. Satisfactory results are also obtained by drawing forwards the prolapsed and œdematous mucous membrane, and then snipping it off.

TUMOURS OF THE URETHRA.—The innocent tumours of the urethra are neither common nor important, and the primary malignant growths are very rare in both sexes. The simplest innocent tumours are found by the urethroscope in the male urethra. They are simply masses of granulation tissue in the neighbourhood of strictures, or associated with chronic inflammations of the urethra. Small polypi are found in the prostatic portion of the urethra independently of any urethral inflammation, and occasionally a villous tumour makes its presence known by hæmorrhage when it is found in the penile portion of the urethra near the meatus. I have seen from time to time a pedunculated fibrous tumour growing from the lips of the meatus.

A urethral caruncle is the best known urethral tumour growing in the female. It is very vascular, is painful when it becomes inflamed, and it often causes increased frequency of micturition. The treatment consists in dissecting it out, for mere ligature and snipping off is useless, as it quickly grows again.

Malignant tumours of the urethra are much rarer than innocent growths. The carcinomata are primary or secondary. Primary cancers in men occur in two forms. A squamous epithelioma growing in old men; it springs from the urethra often at a considerable distance from the meatus, and is to be clearly distinguished from the comparatively common cancer of the penis. A more rare villous carcinoma of the urethra which occurs in younger men, the growth having a histological structure not unlike that of a "duct cancer" of the female breast. This form of tumour spreads rapidly as a destructive papilloma of the penis. Secondary carcinomata involve the urethra by extension from the bladder, prostate, and penis in men: from the uterus and vulva in women.

Sarcoma of the urethra occurs in girls and women. It is part of a general sarcomatous growth affecting all the connective tissues, which are involved in the complicated developmental processes associated with the formation of the cloaca. The growth is polypoid in character, and has a greater tendency to ulcerate in the adult than in the child. The prognosis is more favourable in later than in early life, for the growth can be removed more completely in women than in children.

CALCULI.—The first and only symptom of stone in a child may be the acute retention of urine due to its impaction in the urethra, but in adults there is usually evidence of a calculus before it becomes impacted, and the symptoms are much less acute, whilst the retention may be very incomplete. In adults, too, a stone which has thus become lodged in the urethra, especially if it lies in the prostatic portion, may gradually increase in size until it takes the shape of the urethra for an inch or more. Calculi which have been retained in the urethra in this manner for long periods of time are often multiple. In some cases a calculus may become lodged behind a stricture, and may receive successive incrustations of phosphates from the ammoniacal urine passing over it until it attains a considerable size. The presence of such a calculus increases the tendency to ulceration and pouching of the urethra behind the stricture, and is often associated with extravasation of urine or a urinary abscess. The diagnosis of a urethral calculus is not attended with any great difficulty. It can be felt when it lies in the penile portion of the canal, whilst the passage of a sound will usually demonstrate its presence when it is situated nearer the bladder.

Treatment.—When the stone lies near the meatus and is of a regular shape it can be removed by means of a pair of urethral forceps, and when it lies farther back the passage of a

sound will often drive it into the bladder whence it should be removed by crushing or suprapubic lithotomy. Prolonged and violent attempts should never be made to extract calculi, as much damage may be done to the urethral mucous membrane. If the calculus cannot easily be withdrawn through the meatus or pushed into the bladder it should be removed by external urethrotomy (p. 259), the incision in the skin and urethra being afterwards sutured in the hope of obtaining union by first intention. It is better, I think, not to tie a catheter into the urethra after the operation unless it is clear that the mucous membrane has been much injured, either by the irregularity of the stone or by the attempts which have been made to remove it.

FOREIGN BODIES.—Foreign bodies are more often found in the male than in the female urethra because the shortness of the urethra in women allows them to pass readily into the bladder. In both sexes foreign bodies are often passed into the urethra, and show a remarkable tendency to escape from the hold of the patient and to travel rapidly backwards towards the bladder. They usually cause much pain with the muco-purulent discharge characteristic of urethritis, and their presence is easily recognised by the passage of a sound. No general rule can be laid down for their extraction, and the ingenuity of the surgeon may be taxed to the uttermost to remove them with as little injury as possible to the urethra.

Urethral.—Belonging to the urethra; *e.g.* a *urethral polypus* (pedunculated tumour growing from or protruding from the urethra, such as a caruncle), *urethral fever* (high temperature following the introduction of a catheter, etc.). *See* CATHETERS, USES AND DANGERS (*Urinary Fever*); CAUTERY (*Uses, Urethral Caruncle*); GONORRHŒAL INFECTION (*Complications*); TABES DORSALIS (*Symptomatology, Urethral Crises*).

Urethralgia.—Pain in the urethra, especially of a neuralgic nature.

Urethrameter or Urethrometer.—An instrument for gauging the diameter of the urethra. *See* ASEPTIC TREATMENT OF WOUNDS (*Disinfection of Instruments*).

Urethritis.—Inflammation of the urethra. *See* URETHRA, DISEASES OF.

Urethro-.—In compound words urethro- (Gr. οὐρήθρα, the urethra) means relating to the urethra, *e.g. urethro-bulbar* (relating to the urethra and the bulb of the corpus spongiosum), *urethrocele* (protrusion of the urethral mucous membrane), *urethro-cystitis* (inflammation affecting both the urethra and the bladder), *urethrorrhaphy* (closure of a urethral fistula with sutures), *urethrostenosis* (narrowing of the urethral canal), *urethrostomy* (making an artificial meatus urinarius in the perineum), and *urethro-vaginal* (belonging to the urethra and the vagina).

Urethrotomy.—The operation for the division of a urethral stricture; it may be external or internal, and in the latter case an instrument called a *urethrotome* is used. *See* URETHRA, DISEASES OF (*Stricture, Operative Treatment*).

Urgency Certificate. *See* INSANITY, GENERAL TREATMENT OF (*Scotland, Emergency Certificate*); LUNACY (*Laws*).

Urginea.—Indian squill, the bulbs of *Urginea indica* and *Scilla indica*, used for the same diseases as squill; it is official in the Indian and Colonial Addendum to the B.P. of 1898; there are six preparations: (1) The *Acetum Urgineæ* (dose, 10-30 m.); (2) the *Oxymel Urgineæ* (dose, ½ to 1 fl. dr.); (3) *Pilula Ipecacuanhæcum Urginea* (dose, 4 to 8 gr.); (4) *Pilula Urgineæ Composita* (dose, 4 to 8 gr.); (5) *Syrupus Urgineæ* (dose, ½ to 1 fl. dr.); and (6) *Tinctura Urgineæ* (dose, 5 to 15 m.).

Uriage. *See* MINERAL WATERS (*Sulphated Muriated*).

Uric Acid.

INTRODUCTION 262
PHYSICAL AND CHEMICAL CHARACTERS . 263
METHODS FOR ESTIMATION . . . 264
FORMATION AND EXCRETION OF . . 264
ORIGIN OF, IN THE ORGANISM . . . 265

See also BLADDER, INJURIES AND DISEASES OF (*Calculus*); CHILDREN, CLINICAL EXAMINATION OF (*Urine*); GASTRO-INTESTINAL DISORDERS OF INFANCY (*Colic, Causes*); GOUT; HEADACHE (*Causes, Toxæmia*); LEUCOCYTHÆMIA (*Symptoms*); LIVER, PHYSIOLOGY OF (*Regulation of Supply of Proteids*); MIGRAINE (*Causes*); NEPHRITIS (*Clinical Features, Uric Acid in Blood*); PHYSIOLOGY, BLOOD AND LYMPH (*Plasma, Chemistry of*); PHYSIOLOGY, EXCRETION (*Non-Urea Nitrogen in Urine*); PIGMENTS OF THE BODY AND EXCRETA (*Animal Pigment, Uric Acid in Butterflies*); STOMACH AND DUODENUM, DISEASES OF (*Remote Symptoms, Urinary*); URINE, PATHOLOGICAL CHANGES IN (*Uric Acid*).

AMONG the products of nitrogenous katabolism uric acid occupies a very important place, not only because of the appreciable quantity in which it is usually excreted, but also on account of the marked variations in its excretion under pathological conditions. Variations in the excretion of urea, the most important nitrogenous bye-product, do not so readily attract the attention of the clinician as an increase or decrease in the excretion of such an insoluble body as uric acid. The great tendency that it has to separate out from the urine on cooling is noticed even by the individual who has had no medical

training, and he rightly associates it with the febrile state and its accompanying diminished renal secretion. Not only do uric acid and its salts tend to separate out from such a slightly acid fluid as the urine, but they also under abnormal conditions are deposited in the alkaline reacting tissues.

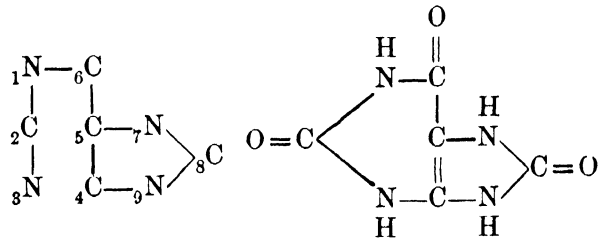
In this article special stress will be laid upon the physical and chemical characters, the probable modes of origin, and the relationship to certain important pathological conditions. In studying what may be called the physiology of uric acid, one is brought face to face with some of the most important problems in metabolism, and it is absolutely essential for all who aim at acquiring an accurate knowledge of the changes which nitrogenous foodstuffs undergo after absorption to have a good grasp of the life-history of uric acid.

Physical and Chemical Characters.—It is in an impure condition that the acid and its salts separate out from the urine, but when examined free from pigments, etc., it forms a white powder, which may easily be obtained as transparent rhombic plates. In a urinary deposit, however, uric acid is always pigmented, having carried down usually the pigment uroerythrin along with urochrome or urobilin—the so-called "cayenne pepper" deposit. It is always in crystalline form, the appearance of the crystals varying greatly from a lozenge shape, through many intermediate forms, to the barrel shape (rhombic plates or rods, daggers, rosettes, cubes, dumb-bells, etc.). If there be a percentage increase in the amount of urates in the urine, although there may be no deposition at body temperature, a precipitate of a yellow to a reddish-brown colour gradually separates out as the urine cools—the "brick-dust" deposit. This, of course, disappears when the urine is warmed above body temperature. In an ammoniacal urine the acid separates out in the form of an acid ammonium urate, sometimes masses of dumb-bell crystals or spherical accumulations of needles. Acid sodium urate, although usually in amorphous form when occurring in a urinary deposit, is now and then met with in crystalline form as spherules with processes—"thorn apple" crystals. In all uric acid or urate deposits the normal pigment of the urine, urochrome, is present as the colouring agent, often but not always associated with uroerythrin.

There is a less soluble salt of uric acid than the biurate, namely, the quadriurate, which is formed by the action of the diphosphates of the alkalies on the biurate. This triacid urate ($NaH\bar{U}$, $H_2\bar{U}$) usually occurs in amorphous form, now and then as a gelatinous cloud, which on microscopic examination is seen to be composed of radially striated spheres. It is very easily decomposed, for example, by means of hot water or neutral salt solutions, into the biurate and uric acid if no additional base be present. The biurates separate out either in the form of an amorphous deposit, small spherules, collections of small acicular or prismatic crystals.

In order of solubility the salts of lithium come first, then potassium, sodium, ammonium, calcium, and magnesium. In all cases the neutral salts are most soluble, then the biurates, while the least soluble are the quadriurates. Uric acid is less soluble than any of these, as one would expect, one part requiring approximately 1600 parts of boiling water or 16,000 parts of cold water to keep it in solution. The neutral salts of the alkalies do not occur in the urine as they so rapidly undergo decomposition.

From the formula of uric acid it is apparent that there are two urea residues, with a central three-carbon chain, and so it has been termed a diureid of trioxyacrylic acid; but from E. Fischer's researches its constitution has been brought more into line with other important bodies of a similar nature occurring in the organism, the so-called xanthin series. Uric acid may be regarded according to this nomenclature as a trioxypurin, a derivative of the hypothetical 9C—N ring "purin." Thus,



(Uric acid).

That is to say, this acid may be regarded as a 2, 6, 8 trioxypurin, xanthin as a dioxypurin, and hypoxanthin as an oxypurin. It is of very great assistance to possess this knowledge of the close chemical relationship existing between these bodies when the physiological connection between them is studied.

As there is, of course, the possibility that in the animal organism there are means of *synthetically* producing uric acid from simpler bodies, it is important to discover how it may be synthetically formed outside the organism. Horbaczewski formed it synthetically from urea and glycocoll, an exceedingly important synthesis from the physiological standpoint.

$$3CO(NH_2)_2 + CH_2.NH_2.COOH = C_5H_4N_4O_3 + 2H_2O + 3NH_3.$$

It was also formed synthetically from trichlorlactic acid, or its amide and urea. Both these syntheses are of great interest, as there is no reason why they should not be carried out in the animal organism.

Thus from a chemical knowledge of the means by which the acid might be formed without the aid of living tissues, investigators began to pay

special attention to those substances occurring in the organism from which purin bodies could be obtained, and also to those which might play a part in a synthesis similar to those just mentioned. Physiological work along those lines will be described when the mode of origin of uric acid is taken up.

The final products of decomposition of the acid are carbonic acid, water, and ammonia, but there is a variety of paths along which this disintegration may take place before arriving at the final stage. When peroxide of lead is used as the oxidising agent, the most important products are carbonic acid, oxalic acid, urea, and allantoin; while if nitric acid be employed, urea and alloxan are formed in the first place, then the latter breaks down on heating into carbonic acid and oxalylurea, the latter finally splitting up into oxalic acid and urea.

Tests employed for the Detection of Uric Acid and its Salts

(*a*) The *microscopic appearance of the crystals*, their *pigmentation* with uroerythrin or urochrome, *and their solubility on heating in alkaline solution* are characteristic.

(*b*) They form definite *crystalline compounds with nitric and oxalic acids.*

(*c*) *Murexide Test.*—If a small quantity of the acid, or one of its salts, be placed in a porcelain lid, and then a few drops of nitric acid be added, on careful heating, either on water bath or over small open flame, a reddish-coloured residue is left, which gives a purple red colour with ammonia, or a bluish violet with a drop of caustic soda (added when residue is cold). On heating the colour disappears.

(*d*) On heating a small quantity of the acid with an excess of *Fehling solution*, *cuprous oxide* separates out, but if the cupric salt be relative to the uric acid small in amount, then a white precipitate of *cuprous urate* forms.

(*e*) It forms *double salts with magnesium and silver*, which possess a characteristic gelatinous appearance. One of the quantitative methods (Ludwig-Salkowski) depends upon this reaction.

(*f*) It is separated out completely from its solutions on *saturation with ammonium chloride* in ammoniacal solution. Another quantitative method (Hopkins) depends upon this reaction.

Methods for Uric Acid Estimation.—There are two which are commonly employed, namely, those of Salkowski and Hopkins, and, as the latter is the more suitable for comparatively rapid clinical work, it alone will be described here.

Take 30 grammes of finely powdered pure ammonium chloride in a beaker, add 100 c.c. of urine, stirring the mixture for some time until solution is complete, then add sufficient ammonia to render it distinctly ammoniacal in odour. Place mixture aside for three to six hours, filter, then wash filter paper with boiling water until all the ammonium urate has been dissolved. Add to this solution sufficient hydrochloric acid to make it slightly acid in reaction, evaporate down on water bath until volume is about 10-20 c.c., then add 1-2 c.c. of conc. hydrochloric acid and leave mixture overnight. After the deposit has been collected on an ash-poor filter paper, and washed chlorine free with distilled water (measured amount), then with alcohol and ether, it is finally dried to constant weight at 110° C.

The ammonium urate of uric acid deposit may be titrated with warm sulphuric acid and permanganate solution, but only after all trace of the chloride has been removed by washing.

Formation and Excretion of Uric Acid.—It is to be met with in largest quantity in the excreta of birds and snakes, where it forms the main nitrogenous waste-product excreted by the kidneys. In man the excretion varies from 0·2-1 gm., the average on mixed diet being about 0·6-0·7 gm. in twenty-four hours, but under certain abnormal conditions more than 1 gm. may be excreted. It is frequently but not always present in the urine of carnivora, the amount as a rule being very small; in herbivora it is probably always to be discovered, although also only in small quantity. It has been extracted from various organs, but only in very small quantities, at least in mammals, traces having been detected in the liver, spleen, pancreas, and perhaps also in the normal blood. In the liver of birds it is present in appreciable quantity, certainly more than in the other organs.

The *influence of diet* upon the excretion is undoubted, although the reasons for the effects produced are still subjects of controversy. Flesh diet causes a rise in uric acid excretion, while a vegetable diet is accompanied by a fall. Where there is a breaking down of tissues, especially of those rich in nucleins (proteid substances containing a purin radicle), there is a rise in uric acid relative to the urea. Just as nucleins or their decomposition products derived from tissue metabolism ("endogenous") produce this increased excretion, so also does a diet rich in those proteids ("exogenous"). An increase has also been noticed two to five hours after food has been taken, containing neither nucleins nor purin bases. In the last-mentioned case, of course, the mode of formation may be of the same nature as in the others, namely, the result of nuclein katabolism occurring during the digestion leucocytosis.

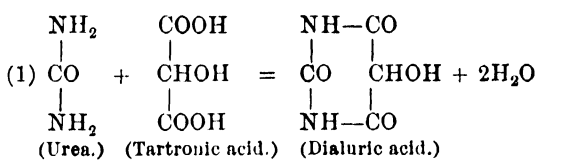
The effect of such variations in the diet as increase in the amount of water or alkalies is comparatively slight, and may be explained in various ways, *e.g.* a purely diuretic effect or an increase in nitrogenous metabolism.

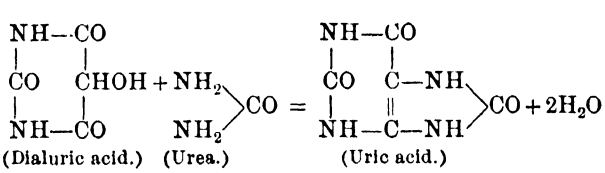
Under mixed diet the ratio of uric acid to urea is from 1 : 50 to about 1 : 70, but there are often marked variations. In the newly born the ratio is 1 : 13.

Origin of Uric Acid in the Organism.—Although in all probability the synthetic mode of origin in mammals is of minor importance when compared to the purin source, still it is one which is of great interest, and as it seems to be the main one in birds where uric acid constitutes the principal nitrogenous effete product, it will be taken up first of all. The earlier observations on birds and snakes dealt mainly with the question of the seat of origin, either by noticing the effects of removal of different organs, or by examining various tissues in order to discover whether any particular one was richer in uric acid than the others, and hence more likely to be the seat of origin. Von Schroeder found that even after removal of the kidneys in birds and snakes, uric acid accumulated in the blood and tissues, thus proving at least that the kidneys were not the sole seat of uric acid formation. Meissner found uric acid both in the blood and liver of birds, but most largely in the latter, and so concluded that it probably was the seat of origin. Von Knieriem found that amido-acids, such as glycocoll, aspartic acid, and leucin produced an increase; but neither ammonium chloride nor sulphate produced an appreciable effect upon the excretion. Subsequently, however, von Schroeder found that, just as in mammals, the only ammonium salts that could be synthetised were those which could be converted into the carbonate in the organism, this being the reason why von Knieriem obtained no effect from giving the sulphate and chloride. The last-mentioned investigator surmised that ammonium lactate might be the important forerunner, but it was not until Minkowski published his very important work that our knowledge of the subject was based on sure experimental evidence. In an elaborate series of experiments on geese he showed that, if the liver were removed in its entirety, practically all the uric acid disappeared from the urine, while its place was taken by ammonium sarcolactate. The reason that he gave for the persistence of a small uric acid excretion even after removal of the liver was that another mode of origin existed, similar to that in mammals, namely, from nuclein sources, and this hypothesis he based upon results obtained by von Mach, who gave, on Minkowski's suggestion, hypoxanthin to geese before and after removal of the liver. He found that under both conditions this purin body could be converted into uric acid.

Although the importance of those investigations can scarcely be over-estimated, one must remember that the mere fact that the birds lived for so short a period after the operation (6 to 18 hours), makes the experiments less conclusive than they would otherwise be. Hoppe-Seyler and Araki suggested that the results might be due to the increased production of sarcolactic acid as a result of the disturbance in oxidation processes, and that the rise in ammonia was required to neutralise the abnormal amount of acid. Minkowski answered those objections by showing that an operation of almost equal severity, entailing as great a general disturbance, did not produce an increased sarcolactic acid excretion. Lang showed that after removal of the liver from geese, not only was there an increase in the ammonia excretion, but also a decided rise in the mono-amido acids.

In support of Minkowski's views the author has shown that acids on being given to birds produce a very marked diminution in uric acid and a corresponding increase in ammonia, similar to the effects produced by acids in carnivorous mammals when the urea is replaced in part by ammonium salts. He was unable to find a direct transformation of hypoxanthin to uric acid, at least certainly not to the same extent as von Mach describes, and all his experiments went to prove that in birds the purin mode of origin was of minor importance compared to the synthetic one from ammonium salts (*vide* Wiener). It is possible that the nitrogen of the purin bodies may be split off in some cases in the form of ammonia, and may then, of course, be synthetised. Wiener, in a recent paper on the synthesis of uric acid, refers especially to the nitrogen-free partner in the synthesis, and concludes that tartronic acid may be the body, the process probably running as follows. Tartronic acid unites with one molecule of urea to form dialuric acid, and this in its turn combines with a second molecule of urea to form uric acid, thus—





Whether this possible synthesis is the actual one occurring in the organism of the bird is still doubtful, and although this author's experiments seem to favour the theory that a similar synthesis occurs in man, still, as he himself remarks, its importance compared to the purin origin is probably not great. In mammals there can be no doubt that the purin-holding substances constitute the main source of uric acid, and Minkowski in his most recent investigations comes to the conclusion that in dogs, at least, the synthetic mode of origin is of extremely slight importance, if it exists at all. The purin mode of origin is undoubtedly the most important one in mammals. Thus after a diet rich in nucleins (nucleo-proteids)

there is a marked increase in the amount of uric acid excreted, both absolute and relative to the urea. This transformation can take place outside the organism, as for example when spleen or any tissue rich in nucleins is kept in contact with oxygenated blood at body temperature. A rise in uric acid excretion after a meal free from nucleins is probably due to an increased nuclein metabolism in the tissues (*e.g.* digestion leucocytosis).

Horbaczewski was the first to refer to endogenous sources of uric acid. He drew attention to a possible connection between increase in number of leucocytes and increased uric acid production, but his conclusion that all forms of leucocytosis are accompanied by this uric acid rise is certainly incorrect. Leucocytosis accompanies pathological conditions of the most diverse nature, so that it was scarcely reasonable to expect that their effect on metabolism would be the same. One would expect that if the rise were due to the purin derivable from the nucleins present in the leucocytes, this would be most marked when the number of leucocytes was falling. One would also expect that a rise in P_2O_5 would accompany the increased purin excretion, seeing that the former must be set free after nuclein disintegration, and this does not occur in all cases, *e.g.* leucocythæmia. That variations in the absolute number of leucocytes in the body, or more likely alterations in the metabolism of the white blood corpuscles, occurring with or apart from an absolute increase in their number, may affect the purin excretion is, however, very probable, and that the purin-holding foodstuffs do so is undoubted.

The excretion of uric acid in gout is a subject upon which opinions differ greatly, and the reader must be referred to the special article on "Gout."

The difficulties that are met with in trying to discover the significance of an alteration in the amount of uric acid excreted are intelligible when one remembers that there may be an increased *production of uric acid* with no effect on the excretion, because it may be broken up into urea in the liver, or the variations in the quantity excreted may simply be due to a more rapid or slow removal by the kidneys.

Uricacidæmia or Uricacidemia.—The presence of uric acid in the blood in such amount as can be demonstrated; the possible but not the probable cause of gout. *See* Uric Acid; Gout; etc.

Uricedin.—A proprietary preparation recommended in gout; it contains sulphate chloride, and citrate of sodium, and some potassium and calcium salts.

Urinal.—A vessel for the reception or for the inspection of urine; also a convenient place, in cities, public buildings, etc., in which urine can be passed. *See* Bladder, Injuries and Diseases (*Extroversion of the Bladder, Non-Operative Treatment*); Sewage and Drainage (*Water Closets*).

Urinalysis.—The chemical testing of the urine. *See* Urine, Pathological Changes in.

Urinary.—Belonging to the urine or to the organs which secrete, transmit, or store it up. *See* Urination; Urine, Pathological Changes in; Bladder; Kidneys; Urethra; etc.

Urination, Disorders of.

Incontinence of Urine in Children . 266
Dysuria in Children 267
Increased Frequency of Micturition in Adults 267
Incontinence in Adults . . . 268
Retention of Urine 268
Anuria 269

See also Micturition; Neurasthenia (*Clinical Features*); Urine, Pathological Changes in; etc.

Incontinence in Children.—In the physiological act of micturition the natural stimulus and evacuation of the bladder is a degree of mechanical distention of the organ. When this reaches a certain degree afferent impulses pass through the spinal cord and the brain, which in turn sends out efferent impulses to the bladder, bringing into play the natural muscular mechanism which effects the expulsion of urine. After the third year of life the urine may be held during sleep for eight or nine hours, and at other times for two or three hours. If a child cannot control his bladder during his waking hours by the time he has reached his third year he may be regarded as suffering from incontinence (Thomson). Incontinence may be due to—

1. Some defect in the wall of the bladder or urinary passage in any part of its course.
2. An alteration in the character of the urine, or
3. A change in the nervous mechanism which governs micturition.

Enuresis due to Defect in the Bladder or Urinary Apparatus.—This may be due to some malformation, *e.g.* a small meatus, extroversion of the bladder, epispadias, or hypospadias. Long or adherent prepuce is also a common cause, and among rarer conditions we find polypoid growths in the bladder or at the urethral orifice. Cystitis is not a common affection in children, but when present always gives rise to incontinence of urine. Included in this category also is the atonic condition of the sphincter of the bladder which may be present in any acute or chronic debilitating illness. Intestinal worms and scybalous masses in the bowel may also determine increased frequency

of micturition. In intractable cases a careful visual examination of the urinary apparatus, vulva, etc., should be made.

Enuresis due to Alterations in the Character of the Urine.—In every case of incontinence of urine in children the urine should be carefully examined, special attention being paid to the degree of acidity, and the presence of any abnormal constituents. Oxaluria, phosphaturia, or uric acid in excess may be the determining cause. If the presence of one or other of these is determined, attention must, of course, be directed to the diet and the state of the gastro-intestinal tract.

A Change in the Nervous Mechanism which governs Micturition.—As the controlling nervous mechanism of micturition is a complex one, we find that there are many factors which may effect a disturbance of the act. These may be roughly grouped as follows:—

1. Defects in the higher controlling centres in the brain.

2. Disease in the spinal cord (lumbar region) which involves the reflex centres for micturition.

3. Derangement of the peripheral nervous mechanism in the wall of the bladder.

Epileptics, imbecile and mentally deficient children, are on this account prone to suffer from incontinence. Fright is another factor which occasionally induces an inability to retain urine for the normal time. Any disease of the spinal cord may give rise to want of control over the bladder. This disease may primarily involve the lumbar centres, or secondarily from interference with the conducting paths in the cord. We must admit, however, that in many cases of incontinence in children no lesion can be discovered to account for the symptoms, and in such cases we are forced to take refuge in the theory of disturbed nerve action from unascertained causes. This disturbance may manifest itself either in delicate children or in those in apparently robust health. In some cases there is some inability to retain the urine during the day as well as at night. In most cases, however, the incontinence is nocturnal only. In some cases there is a history of bed-wetting from early infancy; more frequently the disease has developed after the first few years of life.

Treatment.—It will be obvious from what has gone before that the treatment must be etiological. Every attention should be paid to ascertain what part of the mechanism of micturition is at fault in any case. If the urine is obviously faulty diluents and alkalies with a change of diet may be all that is necessary. Any source of local irritation in or about the bladder, or peripheral source of irritation, *e.g.* in the intestines, should be removed. The ordinary cases, due to unascertained causes, are the most intractable. Careful attention to regular habits of micturition are essential. The medicinal remedies of most value are belladonna, ergot, and cantharidis. Belladonna is best administered in the form of the tincture, 20 to 60 minims at bedtime, according to the age of child. If this fail, ergot, in 20-minim doses twice or thrice daily, may succeed, or a cantharidis blister over the sacrum may be tried. The diet should in all cases be investigated. A purely milk diet may give a very good result in cases which resist all medicinal remedies. The application of the cold douche to the spine is a means of treatment that is much advocated by some authorities. The child before being put to bed should sit in a bath or tub, and from one to two gallons of cold water poured down the spine from the nape of the neck. It is then rubbed dry and put to bed (Carmichael).

Dysuria in Children.—Pain in micturition in children is usually dependent on one of the following causes:—

(*a*) Phimosis or preputial adhesions.

(*b*) Vulvitis.

(*c*) Uric acid or phosphates in excess in the urine.

The child has a constant desire to pass water, and screams with pain when she tries to do so. Treatment must be directed to the cause. When due to the presence of uric acid in excess the condition may be accompanied by a true renal colic.

Increased Frequency of Micturition in Adults.—This may be dependent on a defect in the bladder, a condition spoken of as irritability of the bladder. It may be due to various forms of inflammation of the bladder wall, calculi, foreign bodies, or tumour growths. Again, it may be due to a stricture of the urethra. A very frequent cause in old age is enlargement of the prostate gland, more especially in cases attended with enlargement of the middle lobe. Renal disease is also a cause, more especially chronic nephritis and pyelitis. In addition to these various diseased states involving the urinary apparatus, increased frequency, often attended by some incontinence, may arise from various constitutional derangements which are attended by the passage of an abnormal urine. The most striking illustrations in this group are diabetes mellitus and insipidus, and hysteria. It is probable that some cases are to be explained by the existence of early vascular disease throughout all the organs and tissues of the body. In such cases the vaso-motor control of the circulation is defective, and variations in the calibre of the renal vessels occur with more frequency than in perfectly normal conditions, with the result that a large amount of limpid urine of low specific gravity is passed. Increased frequency of micturition varies in degrees; it may exist as a single symptom, but more usually it is accompanied by other symptoms which aid the diagnosis. The diagnosis of the cause is made from the following:—

1. From a careful examination of the history,

the duration of the complaint, the age of the patient, the history of previous trouble with his urinary apparatus.

2. A digital examination per rectum, to distinguish the presence or absence of disease of the prostate gland, or any disease of the rectum which may also act as a causal agent.

3. A careful examination of the urine, more particularly noting the specific gravity, and the presence of sugar or albumin, indicating structural or functional derangement of the kidneys and other organs.

4. If the foregoing are negative, attention should be directed to a general medical examination of the patient, paying particular attention to the state of the heart, blood-vessels, and blood-pressure. Definite changes may then be found long before the urine reveals the presence of any abnormal constituent.

The treatment will, if necessary, vary with the cause. The only point calling for further note is number four. The line of treatment should be, (*a*) judicious use of aperients; (*b*) attention to the functions of the skin, *e.g.* by the use of Turkish baths and the like; and (*c*) medicinal remedies, more especially iodide of potassium in small doses.

Incontinence and Overflow of Urine in the Adult.—A true inability to retain water on the part of the bladder is a very rare occurrence, and is chiefly found in cases of disease of the nervous system, and is then usually accompanied by indications of paralysis in other parts of the body. The increased frequency of micturition in people over sixty years of age or thereby, which involves passing water every hour or two both by day and night, is not really incontinence, but the reverse. The defect in these cases is an inability on the part of the bladder to empty itself, the cause being usually obstruction from an enlargement of the prostate gland with secondary weakness in the wall of the bladder. There are three methods of treatment applicable, depending on the stage and severity of this disease. These are—

1. In the early stages educate the patient not to cease the attempts at micturition until every drop of water has been passed that can possibly be done by a sustained effort. This is a most important therapeutic point, and if early attended to much benefit will be derived.

2. The use of a soft or flexible catheter used under the strictest aseptic precautions.

3. Operative measures directed to the removal of the prostate gland, or that part of it which is the cause of the mechanical obstruction. This subject is further considered in the section on "Prostate Gland." In these cases the muscular tone of the bladder wall may sometimes be improved by the use of general and nervine tonics or by electricity, or various massage treatment carried out by an expert in the Turkish system of massage.

Incontinence from Disease of the Nervous System.—This is an important group of cases, of which typical illustrations are tabes dorsalis, general paralysis, and indeed all chronic diseases approaching their natural termination. Having regard to the nervo-muscular mechanism involved in the act of micturition, we can readily understand that a defect may be due to—

(*a*) A lesion in the brain cortex, accompanied by dulling of the sensibility (paralysis of sensation), or inability to give rise to adequate efferent stimuli (motor paralysis). This factor is perhaps the most important one in explaining incontinence of urine towards the end of any chronic exhausting disease.

(*b*) A defect in the conducting columns of the cord, such as is present in cases of tabes and other chronic diseases of the cord.

(*c*) Interference with the normal activity of the sphincter centre and micturition centre in the lumbar region of the cord, *e.g.* in cases of paraplegia, traumatic or otherwise, and also in tabes, etc.

The temporary nature of the bladder symptoms in some of these cases is best explained on the assumption that the nutrition of the nerve cells and fibres is from time to time interfered with, mainly from a spasm of their nutrient vessels. It is important to appreciate this point, because it indicates the necessity of treatment of these cases being conducted on general medical lines, and not on strictly neurological lines, as is frequently the case.

A word may be said as to the occurrence of disorders of urination in the adult female. The chief point that requires to be added to what has been said has reference to the gravid uterus. In the early and later months of pregnancy increased frequency of micturition may be a prominent symptom. If the symptoms become prominent we should suspect a complication, *e.g.* retroflexion of the gravid uterus, or other complications, such as a dermoid, or ovarian or uterine tumour. The position of the urethra in women makes it more easy for pressure to induce a slight degree of irritation followed by incontinence. In these cases treatment must be directed to the causal conditions.

Retention of Urine.—In the majority of instances this is dependent on mechanical obstruction, the most common causes being an enlarged prostate, and a stricture of the urethra, the result of previous urethritis. An impacted calculus and tumour growth are rarer causes of retention. Retention of urine must not be confounded with suppression, the latter being the result of defective renal activity, as a result of which no urine is produced and the bladder remains empty. Mention should be made of cases of hysterical retention of urine. Young women especially are the subjects of this disorder. No urine may be passed for a few days, or even longer, and on examination the bladder

may be enormously distended and contain several pints of urine. It is a wise rule in these cases not to withdraw the whole of the urine at one operation. By so doing the risk of the occurrence of bleeding from the vessels of the bladder wall, which have been suddenly relieved of their strain, is minimised. The treatment of ordinary cases of retention is the treatment of the disease causing it. A catheter of appropriate size and kind should be used. Among other remedies mention should be made of—(*a*) the use of a hip-bath at a temperature of 100° to 110° F., (*b*) hot fomentations over the pubic region, (*c*) the use of active purgative medicines, and (*d*) the use of morphia as a temporary measure. If opium be used it should be used in full doses.

ANURIA.—Absence of urination may be due to suppression, or it may mean that the ureter of a working kidney has become completely blocked, while the other kidney has from some cause been thrown out of action. The nature of the injury, or the character of the disease causing this, varies greatly. They may be classified as follows (Newman):—

1. Reflex inhibition of the functions of the opposite healthy kidney.
2. Impaction of a stone at a previous date, followed by hydronephrosis or atrophy of the kidney on the opposite side to the one recently attacked.
3. Other unilateral disease of the kidney, such as tuberculous nephritis, pyonephrosis, or congenital absence of one kidney.
4. Bilateral disease of the kidneys, including the various forms of Bright's disease, cystic degeneration, etc.

The diagnosis of these conditions is established from—(*a*) the history of the case, (*b*) careful bimanual examination of the region of the kidneys, and (*c*) the presence of uræmic or other symptoms indicating a severe toxic poisoning.

Urine, Pathological Changes in.

i. INTRODUCTORY 270
ii. GENERAL PROPERTIES—
Quantity, Specific Gravity, Freezing-Point, Reaction, Toxicity, etc. 271
iii. INORGANIC CONSTITUENTS—
Chlorides, Sulphates, Phosphates, Metals, Ammonia . . . 275
iv. OXALURIA 278
v. PROTEIDS IN URINE—
Albuminuria, Albumosuria, etc. . 279
vi. THE CHIEF NITROGENOUS CONSTITUENTS—
Total Nitrogen, Urea, Uric Acid, Xanthin Bases, Kreatinine . 285
vii. SOME ABNORMAL NITROGENOUS CONSTITUENTS—
Diamines, Leucin and Tyrosin . 288
viii. SUGARS IN URINE—
Glucose, Lactose, Pentose . . 289
ix. THE ACETONE GROUP—
Acetone, Aceto-Acetic Acid, β Oxybutyric Acid 291
x. AROMATIC SUBSTANCES—
Ethereal Sulphates and Glycuronates, Indicanuria, Alkaptonuria 293
xi. NEUTRAL SULPHUR—
Hydrothionuria, Cystinuria . 294
xii. LIPURIA—
Chyluria, Cholesterin in Urine . 295
xiii. PIGMENTS—
Including Melanuria . . 296
xiv. BILE-PIGMENT AND ACIDS . . 298
xv. PNEUMATURIA 299
xvi. URINARY SEDIMENTS . . . 299
xvii. URINARY CALCULI . . . 304
xviii. DRUGS, POISONS, AND ACCIDENTAL CONSTITUENTS OF URINE . 306

See also ABDOMINAL TUMOURS, DIAGNOSIS OF; ACROMEGALY (*Symptoms, Urine*); ADRENAL GLANDS (*Physiology, Effect on Renal Secretion*); ALCOHOLISM (*Chronic, Symptoms, Incontinence of Urine*); ANÆMIA, PERNICIOUS (*Symptoms, Urine*); BLACKWATER FEVER; BLADDER, INJURIES AND DISEASES OF; BRAIN, TUMOURS OF (*Localising Symptoms, Urine*); CATHETERS, USES AND DANGERS OF; CHILDREN, CLINICAL EXAMINATION OF (*Urine*); CHOLERA, EPIDEMIC (*Symptoms, Urine, Suppression*); CHOREA (*Symptoms, Incontinence of Urine*); CRYOSCOPY; DIABETES INSIPIDUS; DIABETES MELLITUS; DIPHTHERIA (*Associated Symptoms, Albuminuria*); DIURETICS; ENZYMES (*Urinary*); ERYSIPELAS (*Clinical Features, Urine*); FILARIASIS (*Chyluria*); GOUT (*Urine in Gout*); GROIN (*Anatomy, Extravasation of Urine*); HÆMATURIA; HÆMATOPORPHYRINURIA; HÆMOGLOBINURIA; HEART, MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Kidneys*); HYDATID DISEASE (*Echinococci of Urinary System*); HYSTERIA (*Disorders of General Nutrition, Urinary Formula*); HYSTERIA, SURGICAL ASPECTS OF (*Retention of Urine*); JAUNDICE (*Symptoms, Urine*); KIDNEY, PHYSIOLOGY OF; KIDNEY, SURGICAL AFFECTIONS OF; LEUCOCYTHÆMIA (*Symptoms, Characters of Urine*); LIVER, DISEASES OF (*Acute Yellow Atrophy, Urine*); LIVER, DISEASES OF (*Cirrhosis, Characters of Urine*); LIVER (*Hyperæmia, Symptoms, Urine*); LIVER (*Tropical Abscess, Symptoms, Urine*); LIVER (*Hydatids, Rupture into Urinary Passages*); MALARIA (*Clinical Manifestations, Urine in*); MALINGERING (*Genito-Urinary*); MENINGITIS, TUBERCULOUS (*Symptoms, Retention of Urine*); MICTURITION; MORPHINOMANIA (*Diagnosis, Urine*); NEPHRITIS; NOSE, POST-NASAL ADENOID GROWTHS (*Symptoms, Nocturnal Incontinence of Urine*); PARASITES (*Helminths, Bilharzia*); PHYSIOLOGY, EXCRETION (*Urine*); PONOS (*Odour of Urine in*); PREGNANCY, PHYSIOLOGY OF (*Changes in Urine*); PROSTATE

GLAND; PUERPERIUM, PHYSIOLOGY (*Excretory System*); RHEUMATISM, CHRONIC (*Treatment, Attention to Excretory Organs*); RICKETS (*Clinical Features, Urine*); STOMACH AND DUODENUM, DISEASES OF (*Symptomatology, Secretory*); SUPPURATION (*Diagnosis, Microbes in Urine*); TEMPERATURE (*Changes in Urine in Fever*); TETANY (*In Toxic Conditions, Albuminuria*); TOXICOLOGY (*Carbolic Acid, Symptoms*); TOXICOLOGY (*Alcohol*); TYPHOID FEVER (*Etiology, Symptoms, Complications*); TYPHUS FEVER (*Symptoms*); URÆMIA; URIC ACID; URINATION; URINE, BACTERIA IN; UTERUS, MALFORMATIONS OF (*Development of Genito-Urinary Organs*); and VAGINA, DISORDERS OF (*Urinary Fistulæ*).

I. INTRODUCTORY

EXAMINATION of the urine is one of the most ancient of methods employed in the diagnosis of disease, and advancing knowledge, far from diminishing, has greatly enhanced the value of the indications which it affords.

Whereas the early physicians, from Hippocrates downwards, were perforce content with what could be learnt by such simple means as inspection and smell, in more recent times analytical methods, both qualitative and quantitative, have been applied to the recognition and estimation of the various constituents of this highly complex mixture, and the microscope, spectroscope, and polarimeter, not to mention other instruments, are all brought into requisition for the elucidation of certain special points.

By such means a great mass of knowledge has been accumulated regarding the composition of the urine in health and the changes which it undergoes in disease, but it will only be possible to speak, within the limits of the present article, of such points as are of special importance and interest from a clinical rather than a pathological standpoint, and to describe briefly such simple methods of analysis as are employed in clinical work. For discussions of the more complex problems which arise, as also for the more elaborate methods in use in the laboratory, it will be necessary to refer the reader to the various treatises devoted to the subject.

So many of the waste products of metabolism are chiefly or wholly eliminated in the urine, that the changes in the amounts of its various constituents, and the presence in it of substances which are not present under normal conditions, afford very important indications of disturbances of the complex chemical processes at work in the organism.

The individual constituents are derived from various distinct sources. Some of them are products of the metabolism of the tissues of the body itself, waste products of the combustion of the muscular, fatty, and other structures. Others, again, are substances introduced into the alimentary canal, and which, although they are thence absorbed, are not incorporated into the tissues but find their way either unchanged or in altered forms into the renal excretion. These two classes of substances overlap to a considerable extent, and not a few of the constituents of the urine are partly endogenous, *i.e.* products of tissue metabolism, and partly exogenous, that is to say, constituents of the food which pass through the body unchanged. Some of these latter, as for example certain inorganic salts and many drugs, fulfil highly useful purposes in their passage.

In yet another class may be included certain products of the action of the bacteria which are so abundantly present in the alimentary canal, either upon the materials of the food or upon the constituents of the bile, and the quantities of such products excreted in the urine afford valuable information as to the state of affairs within the alimentary canal. Among such derivatives of the food may be classed the members of the indoxyl group, which are excreted by the kidneys in the forms of indoxyl sulphates and glycuronates, and other aromatic compounds which also appear as conjugated sulphates. On the other hand urobilin, the chief member of the latter group, has for its parent substance the pigment of the bile.

Among the products of the body tissues which appear in the urine in disease are some which do not appear to be formed in the normal organism, and others which must be looked upon as intermediate products of metabolism which, in consequence of the abnormal conditions which prevail, have escaped the further changes which they normally undergo, and are therefore excreted as such. However, it is not yet possible to make any complete classification of the urinary constituents according to their places of origin, since we are still in ignorance of the sources and modes of formation of not a few of them.

Of no less clinical importance than the evidences of disordered metabolism which are obtained from the study of the renal excretion are the indications which are afforded of disease in the urinary tract. Thus the power of the kidneys to hold back the albumin and globulin of the serum is easily impaired by morbid changes in those organs, with the result that these proteids are excreted in the urine in larger or smaller amounts. It is in this connection, too, that the microscope is of special service in urinary diagnosis, for, with the exception of some kinds of bacteria, all organised cells or groups of cells present in the urine have their origin in the kidneys, ureters, or some other portion of the genito-urinary tract, or in cavities in communication therewith. Thus by means of the microscope we are able to distinguish between hæmaturia due to hæmorrhage from the kidneys or from below them, and hæmoglobinuria resulting from the presence of free blood pigment in the plasma. Again, epithelial cells of various kinds are met with, from the

shapes and sizes of which it is often possible for the trained eye to recognise the portion of the genito-urinary apparatus from which they are derived.

Bacteria of various kinds are met with in the urine either as the result of infection of the urinary tract from without, or of diseases of the kidneys and passages, or, as appears to be the case in typhoid fever and some other infective diseases, of their expulsion from the body by way of the kidneys. In the former case the presence of bacteria is usually associated with evidences of cystitis or other local inflammatory processes, whereas bacteriuria of the latter kind may be attended by no signs of cystitis or pyelitis. Even here the large numbers of bacteria present probably result from their multiplication in the urine itself, only a small proportion of those expelled having actually passed the renal barrier.

One of the most remarkable features of the urine is the manner in which its many constituents are all held in solution, and do not mutually precipitate each other, a condition of affairs which is essential to the perfect performance of the functions of the human urinary apparatus. However, very slight deviations from the normal conditions suffice to interfere with the solution of some of its constituents, as is well exemplified by the so frequent deposition of uratic sediments when the urine cools to the temperature of the air, and the effect of increased or diminished acidity in favouring the deposition of crystalline uric acid on the one hand and of earthy phosphates on the other.

Hence it is by no means uncommon to meet with some or other of the constituents in the form of sediments, either crystalline or amorphous, which sediments may have been formed either before or after the liquid has left the bladder.

Under certain circumstances small concretions are formed in the kidneys, which may either remain in the renal pelvis, or pass along the ureters to the bladder. Either in the pelvis of the kidney or in the bladder they may increase in size by the deposition upon them of successive layers of the original or of some other material; and by their presence, or in their transit from place to place, they give rise to the familiar symptoms and morbid changes which are associated with calculous disorders.

Some accidental constituents derived from foods or drugs produce conspicuous effects upon the urine with which it is important to be familiar, if only that their want of importance may be recognised. In this connection certain colouring matters, such as methylene-blue, may be referred to. The coloration produced by methylene-blue has recently been utilised for the determination of the integrity of the kidneys, by measuring the time which elapses between the injection of a given quantity of the pigment and the appearance of a green tint in the urine. Again, the detection of certain drugs may be of service in ascertaining whether a patient has been taking the medicines in question.

II. General Properties of the Urine

A necessary preliminary to any examination of the urine for clinical purposes is to take note of certain of its general properties—such as its colour, whether it be clear or turbid, whether it has any striking or peculiar odour, its reaction to litmus paper, and its specific gravity, which last, when taken in conjunction with the amount of the daily excretion, affords a rough indication of the quantity of solids which it contains.

Quantity.—The collection and measurement of the total urine passed in the course of the day of twenty-four hours is not only important on account of the variations in quantity observed, but is always desirable in order that from the collected total a fair sample specimen may be obtained, and is essential when any quantitative estimations of its constituents are to be carried out.

The average amount of urine passed by a healthy adult in twenty-four hours is taken as being from 1500 to 1700 cubic centimetres, or about 2½ to 3 pints. Thomas states that an adult man excretes about 1 c.c. hourly for each kilogramme of body-weight. Women excrete somewhat less urine than men, and children pass considerably smaller quantities than adults.

The quantity varies at different periods of the day, being least during the hours of sleep and greatest after the chief meal or after the free drinking of liquids. Active exertion, by increasing the activity of the sudoriferous glands, tends to notably diminish the output of urine. Apart from any obvious modifying conditions, the quantities habitually passed by different healthy individuals differ somewhat widely, and one occasionally meets with persons who, without any obvious departure from the standard of health, habitually excrete quantities considerably below those usually regarded as normal.

C. E. Simon draws an interesting comparison between the estimates arrived at from observations on normal persons in different countries, which reveal differences which he ascribes in part to habits of life and in part to climatic influences.

Nervous influences also modify the urinary excretion. Emotions such as fear or anxiety are apt to lead to an increased output, and the same may be observed in cases of hysteria, although hysterical patients occasionally exhibit actual anuria which may persist for some time.

In fevers the excretion of urine is usually diminished, partly, no doubt, as a result of cardiac weakness and consequent impaired circulation in the kidneys. During convalescence, on the other hand, polyuria is often observed. In acute nephritis the urine is scanty and actual

suppression may occur, but in chronic interstitial nephritis the output is usually markedly increased, and the same is often observed in the later stages of chronic parenchymatous affections and in many cases of lardaceous disease of the kidneys.

The influence of ascites in diminishing the flow of urine, and in so establishing a vicious circle, is well recognised, and this influence is probably due to pressure upon the abdominal vessels and a resulting diminution of the flow of blood through the kidneys.

As is only to be expected, when large quantities of water are being lost by the bowels in diseases attended by diarrhœa the quantity of urine shows a conspicuous diminution, and actual suppression of urine may result from obstruction of the ureters, as in cases in which one kidney has been previously rendered useless and the ureter of the only active kidney becomes blocked by a calculus.

Polyuria is a conspicuous symptom of *diabetes mellitus*, and far larger quantities still are passed in the remarkable cases which are grouped together under the name of *diabetes insipidus*. In not a few nervous diseases polyuria may occur, and whereas in hysteria and under the influence of emotions the excess is usually transitory, when the nerve centres are the seats of organic disease it tends to have a more permanent character.

In conclusion, mention must be made of the intermittent passage of large quantities of urine as a valuable diagnostic sign in cases of advanced unilateral hydronephrosis with intermittent emptying of the sac.

Specific Gravity.—As so often carried out with isolated specimens of urine, the determination of the specific gravity loses much of its value. Much more useful results are obtained when a specimen is taken from the collected urine of twenty-four hours, and more useful ones still when the specific gravity is calculated for an average daily excretion of 1500 c.c.

Low specific gravity may be due to excessive excretion of water, the total solids being normal in amount, but when the quantity is not increased it indicates a diminished output of solid ingredients. Similarly an unusually high specific gravity may indicate diminished water or an excessive excretion of solids.

In health this factor varies considerably at different periods of the day, to a great extent inversely to the quantity of urine passed.

The mean specific gravity of the urine of healthy adults is about 1020. Under abnormal conditions it may fall as low as 1002, or may rise above 1040.

For clinical purposes the specific gravity of urine is determined by means of the urinometer, an instrument too familiar to call for description. The urine to be tested should be allowed to cool to the temperature of the air, and should be gently poured, so that frothing is avoided, into a clean cylindrical vessel of considerably greater diameter than that of the urinometer. The instrument is then allowed to sink slowly into the liquid, and after it has come to rest it should be slightly depressed and allowed to float up again. The reading should then be taken from the lower meniscus.

As a rule polyuria is attended by diminution of specific gravity, but to this rule *diabetes mellitus* offers a conspicuous exception, and the association of excessive excretion with high specific gravity, 1030-1040, in itself affords strong evidence of the presence of sugar. On the other hand, it must not be forgotten that sugar may be present in a urine the specific gravity of which is not above the normal, and the routine testing for sugar should not be omitted on that account.

In *diabetes insipidus* the specific gravity of the urine may be but little above that of pure water, and in some cases of chronic interstitial nephritis also very low readings are obtained. The concentrated and highly albuminous urines of patients with acute nephritis have, on the contrary, a high specific gravity, and the administration of certain drugs which are excreted in the urine also has an elevating effect.

In the latest stages of chronic diseases, when metabolism is at a low ebb, the urine passed is often scanty in amount and at the same time of low specific gravity, owing to the scantiness of the solid constituents.

Freezing-Point; Cryoscopy of Urine.—The property of urine most recently utilised for clinical purposes is the freezing-point, and as the alterations in the freezing-point under morbid conditions have been widely studied, since attention was first called to them by A. von Koranyi in 1887-88, it will be well to give some description of the apparatus devised by Beckmann which is employed in such determinations.

The liquid to be tested is placed in a large test-tube, which can be closed by a cork with two bores. Through one bore is introduced a special thermometer of great delicacy, graduated to hundredths of a degree between + 1 and − 4° C. Through the other orifice is introduced a metallic stirrer, by means of which the liquid around the thermometer is kept in constant motion during the determination. The tube is enclosed in a somewhat larger one which acts as an air-jacket and ensures equal cooling of the liquid contained in the inner tube. Both tubes are then placed in a vessel containing water which has been cooled to some five or six degrees below 0° C. by the addition of sodium nitrate or some other salt which produces a low temperature by its solution.

The first effect is a lowering of the temperature of the liquid in the inner tube below its true freezing-point. Then follows a rise, and

when this rise has reached its limit the reading of the thermometer gives the true freezing-point of the solution.

The determination of the freezing-point of a solution supplies a measure of its osmotic pressure, which again is proportional to the number of molecules of the substance in solution in a unit volume of the solvent. Thus equi-molecular solutions of different indifferent substances lower the freezing-point to equal extents, and when two such substances are in solution together, the lowering of the freezing-point which results is equal to the sum of the depressions which the two substances would individually produce.

In the case of acids, bases, and salts, the lowering of the freezing-point is markedly in excess of that which corresponds to the number of molecules present, an anomaly which is explained by supposing that in solution they are dissociated into their component ions, which must be supposed to produce the effect of independent molecules.

Thus the depression of the freezing-point of the urine below 0° C. corresponds to the total number of dissolved molecules or ions, and when the excretion of twenty-four hours is measured, affords an index of the total number of molecules or ions of all kinds passed in that period. The most abundant constituents will naturally be responsible for most of the effect, and these are sodium chloride and urea.

Von Koranyi attaches much importance to the ratio between the lowering of the freezing-point and the excretion of sodium chloride, and believes that a disturbance of their relation affords a very valuable indication of backward pressure in the kidneys in cases of cardiac disease.

He found that the normal freezing-point of the blood was −0·56° C., whereas the normal freezing-point of the urine of twenty-four hours was between −1·3°-2·2°. The variations of the ratio of freezing-point to sodium chloride excretion in twenty-four hours was much less conspicuous under normal conditions, viz. from 1·23-1·69.

In cases of renal disease the molecular excretion may be considerably diminished, indicating a failure in the renal functions, and this deficiency may reach such a point that the freezing-point of the urine may actually be higher than that of the blood of the same patient. Thus Lindemann, who found the normal freezing-point of the urine to vary between −1·30° and −2·30° C., states that in inflammatory diseases of the kidneys, in which average quantities were passed in the twenty-four hours, the depression of the freezing-point is usually less than one degree.

Lindemann also found that in interstitial nephritis the phenomenon is much less marked than in the parenchymatous variety, and again that in acute nephritis the progressive depression of the freezing-point of the urine until ultimately normal figures were reached afforded a valuable index of the process of repair in the kidneys.

It will not be desirable in the present state of our knowledge to enter here upon any detailed consideration of the somewhat complicated questions which arise in connection with this method of urinary investigation, which is as yet in its infancy, nor to discuss the theories upon which von Koranyi explains the results which he has obtained in cases of cardiac disease with secondary disturbances in the kidneys. These will be found set forth at length in his original papers, and an interesting summary of them, in our own language, was given by Huddleston in the *Philadelphia Medical Journal* in 1900.

The most valuable results are obtained by the comparison of the freezing-points of the blood and urine respectively, and also by comparing the urine obtained from each kidney separately by catheterisation of the ureters.

Reaction.—The acid reaction of normal urine is not due to the presence of a free acid, but to acid salts of which the mono-sodium phosphate is the most important. The degree of acidity is to a large extent determined by the relative amounts of mono- and di-sodium phosphates present, and when a certain proportion is reached the urine will have an amphoteric reaction, that is to say, will redden blue litmus, and render red litmus blue. This is the condition with what are known as neutral urines, but which are not neutral in the strict sense of the term.

The reaction of the urine varies at different periods of the day, and the excretion of alkaline urine, rendered turbid by precipitation of earthy phosphates, during the morning hours is not uncommon, even with healthy persons. Diminished acidity is also observed an hour or two after a meal.

A diet rich in meat tends to increase the acidity of the urine, whereas a vegetable diet has a contrary effect, and the urine of vegetivorous animals is habitually alkaline. The administration of mineral acids raises the acidity of the urine to some extent, and alkalies have a still more conspicuous effect in lowering acidity, or, if taken in sufficient quantities, of rendering the urine alkaline. The sodium and potassium salts of such organic acids as citric and tartaric, which become converted into carbonates in the body, also diminish the acidity of the urine.

Absorption of alkaline exudates and transudates is accompanied by diminished acidity or even alkalinity of the urine, and conversely the formation of such effusions, with a corresponding withdrawal of alkalies from the blood, tends to render the urine more acid.

Concentrated urine, such as that passed during fevers, is usually abnormally acid, and a high degree of acidity favours the deposition of urates and the separation of crystalline uric acid, but the excretion of alkaline urine is of more importance from a clinical point of view.

Alkalinity may either be due to the presence of an excess of fixed alkali or to the formation of ammonium carbonate from the decomposition of urea. The passage of urine which is alkaline from fixed alkali has no clinical importance, unless the effect of the administration of alkaline hydrates, carbonates or salts of vegetable acids, can be excluded. Apart from such causes, the persistent excretion of alkaline urine may have its origin in anæmic conditions, malnutrition, and other debilitated states, and especially those in which the nervous system is affected. This symptom is usually removed by tonic treatment rather than by the administration of mineral acids. On account of the precipitation of the earthy phosphates this condition is often inaccurately spoken of as phosphaturia. Ammoniacal urine, which may be recognised by the blueing of moistened red litmus paper held above the surface of the liquid as well as by its odour, is only of clinical importance when it is passed as such. It then indicates infection of the urinary tract and decomposition occurring in the bladder or elsewhere. Such urines are usually rich in mucus-like substance, and deposit crystals of triple phosphate.

Turbidity.—Normal urine is clear and transparent, but on standing it deposits a slight cloud, the so-called nubecula, which is well seen in the apex of a conical glass. Under morbid conditions the urine may be turbid when passed or it may become so on cooling, in the latter case from the separation of amorphous urates. The commonest cause of turbidity of the urine as passed is the precipitation of earthy phosphates which occurs when the liquid is rendered alkaline, and which is not infrequently observed during the alkaline tide which follows a meal or as the result of ammoniacal decomposition.

A common variety of minor turbidity results from the presence of excess of mucus-like substance; the most extreme degree of opacity results from the abundant presence of finely divided fat in the condition known as chyluria.

Odour.—The faint odour of normal urine is familiar to all and is characteristic, as also is the offensive smell of urine which is undergoing ammoniacal decomposition. When a communication exists between the bladder and bowel the urine may have a fæcal odour, and under certain conditions, which will be referred to later, it may emit sulphuretted hydrogen in quantities sufficient to be recognised by the nose. In diabetes and some other conditions the presence of acetone may impart a characteristic odour to the urine.

The effect of eating asparagus has been ascribed by Nencki to the presence of methylmercaptan. The odour of copaiba is easily recognised, as also the smell of violets given off by the urine of patients taking turpentine.

Colour.—The normal yellow colour of urine is apt to be greatly modified under many morbid conditions. Such changes are in part due to alterations in the relative amounts of what may be called the true urinary pigments, viz. urochrome, urobilin, uroerythrin and hæmatoporphyrin, all of which may be present, at least in traces, in the urine of healthy individuals, and in part to the presence of other colouring matters which are not normally present in the urine.

The presence of abundance of urobilin imparts a rich orange colour, and very large quantities may give a brown tint. Uroerythrin gives a redder orange colour, such as is so often exhibited by the urine of patients with febrile disorders or with organic diseases of the liver. A peculiar orange colour is seen when bilirubin is present in small amount, but, owing to the admixture of biliverdin, urine containing bile often has a greenish-brown tint.

The administration of santonin or of chrysophanic acid, which is contained in rhubarb and senna, causes an intensification of the yellow colour, which is changed to pink by the addition of alkalies.

Dark brown urines are not uncommon. The commonest causes of this colour are the admixture of bile-pigments, or of blood-pigment in the form of methæmoglobin. Urines rich in indican, which is itself colourless, often have a dark brown colour which is ascribed to higher oxidation products of indol.

In carboluria the urine has a brown colour with a tinge of green, due to the oxidation of hydroquinone, and a similar aromatic oxidation product causes the dark brown colour seen in the rare condition known as alkaptonuria. In melanuria the urine as it darkens passes through a brown stage, and mention must be made of the brown colour of the urine in some cases of pernicious anæmia, which is usually ascribed to urobilin, but is probably not entirely due to the abundance of that pigment present.

Pink or red urines most commonly owe their tint to an admixture of blood or of free oxyhæmoglobin. A port-wine tint is usually exhibited in hæmatoporphyrinuria, and rosaniline and some other aniline derivatives given as drugs or contained in sweets may impart a pink colour to the urine. Special mention should be made of the eosin-like pigment which is sometimes present in sweetmeats, which renders the urine pink with a bright green fluorescence.

Black urines are usually extreme examples of one of the above anomalies. In melanuria, alkaptonuria, and in extreme cases of carboluria the urine becomes black in time, and in some

cases of hæmatoporphyrinuria blackness is produced by other less known dark pigments which are usually present with hæmatoporphyrin in such cases.

Urines rich in biliverdin may have a rich green colour, but green and blue urines usually owe their tint to smaller or larger quantities of methylene-blue, which is sometimes given as a drug and is sometimes contained in sweetmeats.

Suspended indigo may produce a blue scum upon the surface of an alkaline urine, and occasionally imparts a bluish tint to the whole.

Very scanty pigmentation may be due to a deficiency of urochrome, and the urine of young infants is often almost colourless. A similar want of colour may be due to dilution, as in all kinds of polyuria, and especially in *diabetes insipidus*, in which dilution reaches its most extreme degree.

Toxicity.—When urine is injected into the circulation of animals it causes their death, but different species of animals differ in the ease with which they succumb to such poisoning. Bouchard found that 45 c.c. of normal human urine per kilo of body-weight was fatal to rabbits. The nature of the constituent or constituents which produce this effect has been much discussed, and is still to some extent an open question. The only point clearly established is that the toxic properties reside, in the main, in the inorganic constituents, and that of these the potassium salts are at any rate the most potent in this respect. The various organic constituents, such as urea, uric acid, kreatinine and pigments have been shown one by one to be incapable of producing the effect, at least in such amounts as are present in the quantities of urine experimented with, but it is held by some that although the toxicity is mainly due to potassium, other constituents contribute to the fatal results. Herringham in a recent research found a close correspondence to exist between the toxicity of the urine and the amounts of potassium contained in it, and similar results had previously been obtained by other observers. Charrin and Roger, on the other hand, failed to trace such a correspondence. The toxic symptoms are, moreover, such as result from poisoning with salts of potassium.

III. The Inorganic Constituents of Urine

It will be convenient to commence the review of the several constituents of the urine in their clinical bearings with the consideration of the simplest of all, viz. the inorganic constituents, in which group may be included hydrochloric, sulphuric, and phosphoric acids, and the metals sodium, potassium, calcium, magnesium, and iron.

The quantities of these which are contained in the urine are subject to considerable fluctuations under different conditions, and in the case of most of them the influence of diet is so great that it is necessary to take this into account before drawing any conclusion as to the influence of morbid conditions upon their excretion. To arrive at any valid results it is therefore important to know the amount of the particular acid or base that is being studied in the ingesta as well as in the egesta, both liquid and solid.

The Chlorides.—The great bulk of the chlorine excreted in the urine is in the form of sodium chloride, and only a small proportion is combined with other bases. Even under normal conditions the daily excretion varies somewhat widely, but quantities corresponding to 10-15 grammes of sodium chloride may be taken as the normal average.

The chief factor which determines the variations observed in health is the quantity of sodium chloride taken in and with the food. On a diet poor in salt, or during a period of fasting, the output is conspicuously diminished, whereas when the food is rich in salt the reverse is the case. During the day the quantity excreted in the urine is greater than during the night, and this even in spite of a meal rich in salt taken late in the day.

The drinking of large quantities of water increases the excretion, and a similar increase results from a diet rich in potassium salts or from the administration of such salts as drugs. This increase Bunge ascribes to the combination of some of the chlorine of the sodium chloride of the blood with potassium, and the elimination of the potassium chloride so formed as of a foreign substance.

In chronic diseases the chloride excretion varies, as a rule, with the nature and quantity of the food taken. Von Noorden has observed diminished excretion after copious hæmorrhages, which he attributes to the loss with the blood and the retention of the amount of sodium chloride required for the supply of the new plasma which replaces that which has been lost. The variations observed in other forms of anæmia may be attributed to diet, and in pernicious anæmia to the condition of inanition which supervenes.

In connection with the excretion of chlorides in gastric diseases it must be remembered that several factors may contribute to influence the output, such as the quantity and quality of the food taken, the amount of hydrochloric acid in the gastric juice which may be lost by vomiting, and the interference with absorption which may result from pyloric obstruction. Von Noorden calls attention to the importance of the relation between the excretion of chlorides and that of urea in diseases of the stomach. A simultaneous diminution of both points to simple inanition, whereas scanty chlorides with abundant urea point to breaking down of the proteid tissues, which are poor in sodium chloride, and such a breaking down occurs in

the cachexia which results from malignant growths.

In renal diseases, such as acute and chronic nephritis, the chlorides are excreted in diminished quantity, as also are the other inorganic salts.

Of peculiar interest is the influence of fevers, and especially of acute pneumonia, upon the output of chlorides in the urine. Redtenbacher, in 1850, emphasised the remarkable diminution of the urinary chlorides in acute pneumonia, and since then his observation has been abundantly confirmed by a number of observers. Among these Lionel Beale, Frankel, Röhmann, Terray, von Moraczewski, and R. Hutchison may be mentioned, and it is upon Hutchison's observations that the following account is chiefly based :—

The diminution, even amounting to complete disappearance of the urinary chlorides, is by no means confined to pneumonia, but is met with in other acute fevers. In fevers of long duration, such as typhoid, it is only met with during the earlier days of the attack. In malaria, on the other hand, the excretion of chlorides is increased instead of being diminished. In pneumonia the diminution persists for a day or two after the crisis, and is sometimes followed by an excretion of more than the normal amounts, quantities considerably in excess of the intake with the food.

Hutchison could trace no relation to the height of the fever nor to the extent to which the lungs were involved in different cases, and whilst the chlorides undergo such marked fluctuations nothing comparable occurs with the other inorganic salts, the phosphates and sulphates.

A comparison of the intake and output shows that the diminished excretion is due to an actual retention of chlorides, for it can be shown that the absorption from the alimentary canal is not impaired and that no adequate vicarious excretion occurs by the skin or other channels.

The total excretion in the sputum is small, although the sputum is rich in sodium chloride. The inflammatory exudate, on the other hand, is not very rich in chloride, and the quantity contained in it will not account for the retention as was formerly supposed. Nor can the retention be ascribed to lesions of the kidneys, for nothing comparable is seen even in acute nephritis which causes a diminution of the other inorganic salts of urine also.

Hutchison, whilst he was unable to obtain evidence of the accumulation of chlorides in any special organ in cases of pneumonia, found that the fixed tissues as a whole were apparently somewhat richer in these salts than usual. He brings forward evidence against the view that the retention of chlorides is merely secondary to that of water, and that it simply represents the quantity required to form with the retained water a saline solution of the normal concentration. In the estimation of the urinary chlorides advantage is taken of the well-known precipitation as chloride of silver, and several volumetric methods have been devised for this purpose which will be found described in works on urinary analysis.

Sulphates.—The normal daily output of sulphates in the urine corresponds to some 1·5-3 grammes SO_3. Of the sulphates the greater part occur as simple metallic salts, whereas a far smaller portion is in combination with aromatic substances, as ethereal or aromatic sulphates, of which the indoxyl-sulphate, the so-called urinary indican, affords the most familiar example. Not all the sulphur contained in urine is in the form of sulphates, some always appearing as neutral or unoxidised sulphur.

Of the total sulphates some portion is derived from the food, but the bulk is a product of the breaking down of the tissue proteids, some of which are much richer in sulphur than others. Sulphates administered as drugs appear for the most part as such in the urine.

No such clinical interest attaches to the variations in the total sulphates of the urine as to those in the chlorides. As may be inferred from their proteid origin, they tend to vary concurrently with the nitrogen excretion, the relation of the total nitrogen to the total H_2SO_4 being about as five to one. Accordingly, an increased excretion accompanies febrile disorders and results from active muscular exertion, and, as Garratt has shown, occurs at an early period of such exertion. An increased output has also been observed in leukæmia.

Of far greater clinical importance is the proportion of ethereal to simple sulphates, as affording evidence of the amount of proteid decomposition in the alimentary canal, but of this it will be necessary to speak later in connection with the excretion of aromatic substances.

Phosphates.—Of the phosphates of urine the major portion is derived from the food, the minor from tissue metabolism. The phosphoric acid is combined with several bases, partly as mon-acid and partly as di-acid phosphates. The total excretion amounts to some 2·5-3 grammes P_2O_5 in the twenty-four hours, but varies considerably with the nature of the diet taken.

The phosphates of the alkali metals, especially of sodium, exceed in quantity the phosphates of calcium and magnesium or earthy phosphates.

A diet rich in alkaline phosphates markedly increases the urinary output, whereas the phosphates of calcium and magnesium are to a large extent excreted by the intestine. An animal diet causes a greater excretion than a vegetable one, and the urine of vegetivorous animals is notoriously poor in phosphates. A very small portion, a few milligrammes daily, of the phosphoric excretion is in the form of glycerophosphates.

In such renal diseases as acute and chronic parenchymatous nephritis the output of phosphates is often markedly diminished.

In studying the variations in the excretion of that portion of the total phosphates which is endogenous, and derived from the metabolism of substances rich in phosphorus, such as nucleo-proteids and lecithin, it is important to exclude the greater fluctuations which result from differences in diet, and the evidence which is forthcoming upon this matter is not of very great clinical importance. In fevers there is a like diminution as compared with the output of nitrogen, although the total excretion may be above normal.

In bone diseases, such as rickets and osteomalacia, we might expect to find a conspicuously increased excretion of phosphates, but no such distinct effect is observed. However, in this connection it must be borne in mind that the intestine appears to be the main channel of the excretion of calcium phosphate, and it may be that to this is due the absence of any noticeably increased output in the urine.

Many observations have been made as to the phosphate excretion in cases of central nervous diseases, which affect structures rich in lecithin and might be expected to exert a distinct influence, but the results have not been striking or uniform in character.

In *diabetes mellitus* there is usually a markedly increased excretion of phosphates, and a condition which has been called phosphatic diabetes is described in which, in association with the symptoms of diabetes, there is an excretion of large quantities of urine rich in phosphates.

Much importance has been attached by Zülzer, whose views are supported by the investigations of Edlefsen, to the relative excretion of phosphoric acid and of nitrogen, as affording evidence of the share taken by the different tissues in the metabolic processes in any given case, a relatively large excretion of phosphoric acid pointing to the breaking down of those tissues which are rich in phosphorus, in other words in nucleo-proteids and lecithin.

The name "phosphaturia" is often loosely applied to the spontaneous deposition of earthy phosphates in the urine, a condition which affords no indication whatever of the amount of phosphate excreted but is merely the result of a lowered acidity.

The passage of amphoteric or alkaline urine may be due to the presence of an excess of alkaline salts in the blood, either as a result of their administration by the mouth or as a pathological condition, or to the ammoniacal decomposition of the urine in the bladder or elsewhere. It is upon such causes, and not upon excessive secretion, that the deposition of earthy phosphates upon urinary calculi depends.

The estimation of the phosphates in urine is carried out by titration with a standard solution of a uranium salt, after the addition of acetic acid and sodium acetate.

Metals.—Under normal conditions, and upon an ordinary mixed diet, the salts of sodium are in excess of those of potassium in the proportion of about 5-3. The abundant presence of sodium salts in the food is largely responsible for this excess. A conspicuous diminution of the sodium excretion is observed in fevers, which diminution is chiefly referable to the retention of sodium chloride under such conditions. The administration of salts of potassium is productive of an increased excretion of sodium, and Bunge's explanation of this fact has been already referred to in the section relating to chlorides.

The variations in the excretion of calcium and magnesium have not as yet acquired any special clinical interest. The fact that the intestine is the main channel of their excretion deprives the results of urinary analyses of much of their importance, and perhaps explains to some extent the indefinite results of such analyses in rickets and osteomalacia. A markedly diminished excretion of calcium has been observed in the course of pregnancy.

Traces of iron are normally present in the urine, but the form in which it occurs is at present unknown. It is probably excreted as an organic compound. Whatever its nature, the iron-containing constituent is thrown down with the crystals of uric acid, in which the presence of this metal can readily be demonstrated, and more abundantly in those spontaneously formed than in those precipitated by the addition of acids.

Ammonium.—Seeing that ammonia is readily formed by the decomposition of urea, it is necessary that any observations on the quantity of ammonium salts in urine should be carried out upon quite fresh specimens. The total ammonia excreted by a healthy adult is some 0·7 gramme in the twenty-four hours. Some of this is derived from the food, drink, and even from the air inspired, but the greater part is a product of proteid metabolism. The administration of mineral acids to man and to flesh-eating animals increases the output of ammonia, which instead of undergoing a further change to urea serves to neutralise the free acid introduced, and so the loss of fixed alkalies, which would otherwise result, is avoided. With vegetivorous animals this is not the case, for some reason not clearly understood, and the acid introduced is by them excreted in combination with potassium and sodium.

The excretion of ammonia is increased in fevers, and conspicuously so in *diabetes mellitus* under certain conditions. This increase, which may amount to as much as four to six grammes in the twenty-four hours, cannot be ascribed in any but small part to the diet rich in proteids which is usually taken by such patients, and is apparently due to the formation of aceto-acetic

and β oxybutyric acids, which are neutralised by ammonia, just as are mineral acids given by the mouth.

Such greatly increased output of ammonia is apt to precede the onset of diabetic coma. The clinical significance of the increased output of ammonia is, therefore, very similar to that of the iron reaction in diabetic cases, and it has been urged that an estimation of its amount supplies a valuable guide as to the advisability of undertaking any operative procedure in such cases.

Stadelmann recommends that when the excretion of ammonia is in excess alkalies should be administered in sufficient quantities to neutralise the abnormal acids formed.

The estimation of ammonia in urine is affected by the simple method of Schlösing. To a known quantity of the urine lime is added, and the containing vessel is allowed to stand in a closed chamber, such as an exsiccator, over a measured quantity of a decinormal solution of sulphuric acid, which absorbs, and is partly neutralised by, the ammonia given off. The amount so neutralised is estimated by titration with a standard alkaline solution.

IV. Oxaluria

Calcium oxalate is a constant constituent of human urine, and it seems clearly established that part of the amount present is a product of tissue metabolism, whereas a larger part is directly derived from the food introduced into the alimentary canal.

Of recent years many observers have occupied themselves with the study of this subject, and the researches of Salkowski and his pupils call for special mention, and in this country the work of Dunlop. Such investigations have been greatly impeded by the tedious and unsatisfactory methods formerly in use for the estimation of oxalic acid in urine, and the most recent researches gain in importance from the employment in them of a new and improved method of estimation devised by Salkowski.

The evidence for the endogenous origin of part of the oxalic acid in urine is in part derived from observations upon fasting dogs, and in part from the investigations of Lütje and Lommel upon the urine of human beings taking an oxalate-free diet, such as milk, for considerable periods. It has been shown that carbohydrates and proteids are not the sources of the endogenous oxalic acid, but, on the other hand, that in all probability gelatin is its chief parent substance, whilst kreatin and glycocol are also entitled to be included among the materials from which it is formed.

That the excretion of oxalic acid is influenced by the oxalates contained in the food, *i.e.* the existence of an alimentary oxaluria, has been questioned by some, but it has been established beyond any doubt that the vegetable constituents of the diet are its chief source. Some vegetables, such as rhubarb, spinach, and tea, are specially rich in oxalic acid, and therefore have a conspicuous influence upon the excretion.

The term oxaluria is, somewhat incorrectly, applied to the deposition of calcium oxalate from the urine in crystalline form, whereas it should, strictly speaking, be limited to the excretion of oxalic acid in undue quantities. However, of the conditions which tend to produce excessive excretion, apart from dietary causes, we know little as yet, and the careful observations of Lommel and others have failed to confirm the prevalent belief that such increase is apt to occur in diabetes, and in association with jaundice. It is, therefore, with the tendency to the deposition of calcium oxalate that we are here concerned, a tendency which acquires great clinical importance from the liability which it entails to the formation of calculi in the urinary passages.

The oxalic acid of urine is in the form of the insoluble calcium salt, and among the causes which favour its deposition therefrom its excessive excretion is certainly one, seeing that the power of the urine to hold it in solution is easily overtaxed. That the urine is able to hold it in solution at all is in part due to the presence of the acid phosphates. Klemperer and Tritschler have shown that the acid phosphates of sodium, calcium, and magnesium all exert a solvent action, the sodium salt being the least, and the magnesium salt the most potent in this respect. Other magnesium salts, such as the chloride and sulphate, also favour the solution of calcium oxalate, and it would appear that a high degree of acidity of the urine and a relatively large proportion of magnesium compounds in it, are among the causes which specially tend to keep the oxalate in solution.

Dunlop found that urine which deposited crystals was usually rich in calcium oxalate, but the observations of Klemperer and Tritschler show that a continuous deposition of crystals may also occur even from urine which is poor in this substance.

The microscopical characters of deposits of calcium oxalate will be described in the section dealing with urinary sediments. Leucocytes are often present in considerable numbers in association with them, and the urine may contain albumin or even blood, as in the not very uncommon cases in which hæmaturia follows the eating of rhubarb in considerable quantities. Dull aching pain in the loins, culminating in the intense pain of renal colic, is another symptom which calls for mention.

Begbie described a group of symptoms which he regarded as characteristic of a so-called "oxalic acid diathesis," among which he included dyspepsia, hypochondriasis, neurasthenia, and neuralgic pains in various situations. That such symptoms are not uncommon in association

with the deposition of calcium oxalate is an undoubted fact, but there is much room for doubt whether the primary phenomenon in such cases is an increase of oxalic acid in the blood. Dunlop maintains that the symptoms enumerated are merely those of hyperacid dyspepsia, and that the increased excretion of oxalate which accompanies them is due to the solution of unusually large amounts of the calcium oxalate of the food by the unduly acid gastric juice, and a resultant increase of absorption. In spite of the advocacy of Cantani and others, the tendency at the present day is rather to attach diminished importance to the association of this group of symptoms with oxaluria.

It must be remembered that the crystals are sometimes only deposited from the urine after it has been passed, and that their presence does not necessarily imply that they were formed within the urinary passages. It is therefore important, in any given instance, to ascertain whether or no this is the case by the examination of freshly passed specimens.

Our first aim in the treatment of oxaluria must obviously be to diminish the output of calcium oxalate, as the smaller the quantity present the greater the chance of its being held in solution. With this object we should eliminate from the diet those vegetable foods which are specially rich in oxalic acid, such as rhubarb, spinach, and tea. It will also be well to avoid gelatin, the taking of which has been clearly shown to increase the output. If Dunlop's views are correct, the administration of hydrochloric acid after food, which has frequently been recommended, will rather tend to do harm by increasing the absorption of oxalate from the gastric contents.

Klemperer and Tritschler recommend the taking of articles of diet rich in magnesium, such as rice, peas, farinaceous food, and coffee, and also advise the daily administration of small doses of magnesium sulphate, with a view to increasing the magnesium of the urine as compared with the calcium. The treatment of this condition has hitherto left so much to be desired that one gladly welcomes any therapeutic plan which holds out any prospect of success.

V. Proteids in Urine

Although their chemical relationship to each other renders natural the grouping together, under a common heading, of the substances belonging to the class of proteids which are met with in the urine under various morbid conditions, we should be justified, on clinical and pathological grounds, in placing them in quite distinct categories, seeing that the diagnostic indications afforded by the occurrence of the several members of the group are wholly different.

Certain proteids are normal constituents of the blood plasma which it is one of the most important functions of the renal epithelium to retain in the circulation, whilst allowing free passage to water and to the waste products of metabolism which require to be eliminated from the blood. Others, again, are foreign substances which only find their way into the blood, in any but insignificant quantities, under abnormal conditions, and upon these the renal epithelium exerts no such retentive action.

Thus whilst albuminuria, *i.e.* the excretion in the urine of easily appreciable quantities of serum-albumin, and paraglobulin, unless it be due to admixture below the kidneys, points to some failure of the renal epithelium to perform its allotted task in a satisfactory manner, albumosuria has no definite relation to the condition of the kidneys, but results from morbid processes elsewhere in the body, processes which lead to the formation of products the excretion of which in the urine reflects no discredit upon the kidneys.

It is, of course, true that the primary cause of albuminuria lies behind the kidneys in many instances, as for example in cases of valvular disease of the heart, but in such cases the effect results from secondary changes brought about in the kidneys, as, for example, by circulatory derangements producing venous congestion and impaired nutrition of the glomerular epithelium. The albuminuria is due to renal disorder, although such disorder is secondary and not primary.

Recent researches, and especially those of K. A. H. Morner, leave little room for doubt that minute traces of albuminous material are present even in normal urine, but whether or no such proteid comes through the kidneys or is derived from some lower portion of the urinary tract, its presence does not in any way detract from the diagnostic importance of such quantities of albumin as can be detected by the ordinary methods employed in clinical work.

Albuminuria.—A number of tests are available for the detection of amounts of albumin which are clinically significant, of which the following are amongst those in most general use:—

The Heat Test.—A specimen of urine, which if turbid should have been passed through a filter, is placed in a test-tube, and if alkaline should be rendered feebly acid with acetic acid. The upper part of the column of liquid is then boiled. If an opalescence turbidity or precipitate is formed this may be due either to albumin or to earthy phosphates, owing to changes in the arrangement of bases in the phosphates. If now a few drops of acetic acid be added a phosphatic precipitate will at once be redissolved, whereas an albuminous precipitate tends to become more flocculent. The boiling should be repeated after the addition of each drop of acid.

A few drops to 1 c.c. of nitric acid may be

added instead of acetic acid, after boiling; but if the urine be boiled after the addition of nitric acid, albumin if present may escape detection, owing to the formation of a soluble compound with the acid.

If carefully performed, the possible sources of error being taken into account, the heat test suffices for all ordinary purposes, but when there remains any doubt its indications should be checked by a second test.

Heller's Test.—The urine is allowed to flow gently on to the surface of nitric acid in a test-tube. The pouring is best effected by placing the urine and the acid in two tubes which are inclined to each other at a very obtuse angle, or the acid may be introduced beneath the urine by means of a finely pointed pipette. If albumin be present an opaque white layer forms at the junction of the two liquids. Some pigmentation is usually observed owing to the oxidation of indigo-yielding substances. If nucleo-albumin be present a less defined and less opaque ring appears *above* the junction of the liquids. Another possible source of error is the precipitation of a ring of uric acid, which also lies above the junction, so that if the urine be albuminous two distinct rings, one due to uric acid and the other to albumin, are sometimes visible. The uric ring is removed by heating. Occasionally a ring of precipitated nitrate of urea is formed, but this is easily recognised by its obviously crystalline character. Both these latter sources of error may be eliminated by repeating the test with diluted urine. Lastly, the precipitation of copaiba resin must also be referred to.

The test is a delicate one, and as little as 0·002 per cent of albumin can be readily detected by its means. Still greater delicacy can be obtained by using a wine-glass or small beaker in place of a test-tube, and so observing a thicker layer of liquid.

In addition to serum-albumin and paraglobulin, hetero- and proto-albumoses yield positive results with this test.

Salicyl-Sulphonic Acid.—This test was independently suggested by Roch and MacWilliam. A few drops of a saturated solution of salicyl-sulphonic acid are added to the urine in the cold. If a precipitate forms it is due to albumin or to albumose, but the albumose precipitate disappears when the liquid is heated, whereas that of albumin persists. The test, although almost too delicate, is very useful for clinical purposes, and it has the advantage that with the exception of the proteids, including nucleo-albumin, no other substances present in the urine are precipitated by the reagent employed.

Picric Acid Test.—This test is extensively used. It may either be carried out as a ring test by pouring a saturated solution of picric acid upon the surface of the suspected urine, when if albumin be present a precipitate forms at the junction of the liquids, or the urine may be allowed to fall drop by drop into the picric acid solution, when the paths of the drops will be marked by opaque tracks if the urine contain albumin. Albumoses are also precipitated, but their precipitates disappear on heating, and certain non-proteid constituents, such as uric acid and creatinin, yield precipitates with picric acid. In connection with albumoses it must be pointed out that, as Huppert has shown, normal urine yields a flocculent precipitate when boiled with picric acid. When this test is employed it is necessary that the picric acid should be in excess.

Potassium Ferrocyanide Test.—Acetic acid is added to the urine in excess, and afterwards a few drops of a solution of potassium ferrocyanide; if albumin be present a dense white precipitate is formed. Albumoses and nucleo-albumin yield similar precipitates, but the albumose precipitates redissolve on heating.

If the acetic acid alone causes a turbidity from the precipitation of urates or the mucin-like substance, this should be filtered off before the ferrocyanide is added.

The Quantitative Estimation of Albumin.—For an accurate determination of the amount of albumin (serum-albumin and paraglobulin) in urine, it is necessary to separate and weigh these proteids, but for clinical purposes the method of Esbach affords results which are sufficiently reliable to meet the requirements. Into a specially graduated tube, which is provided with a stand, urine, rendered acid with acetic acid if necessary, is poured up to a certain mark, and a solution of 2 per cent citric acid and 1 per cent picric acid is added until a second mark is reached. The tube is then closed with an indiarubber cork and carefully shaken, so as to insure mixture of the liquids without the production of froth. After standing for twenty-four hours the height of the precipitate is read off by means of graduations which indicate the parts of albumin *per mille*. Variations of temperature constitute the chief source of error in the results.

If the urine be very rich in albumin, it requires to be diluted to a known extent before being placed in the albuminometer.

Significance of Albuminuria.—Except in those cases in which the albumin present is derived from lower portions of the urinary tract or is due to admixture of semen or vaginal discharges, readily detected albuminuria may, as has been already stated, be held to indicate some abnormal condition in the kidneys.

Speaking generally, it may be said that serum-albumin makes its way into the urine more easily than paraglobulin, a difference which may result, as Halliburton has suggested, from a greater size of the molecules of the latter proteid. Undoubtedly the relative quantities of the two vary widely in different cases, and it

may be taken that a copious excretion of globulin points to a more severe degree of renal disease. When, however, we attempt to draw more detailed clinical inferences from this quotient it soon becomes apparent that the data available are as yet insufficient for the purpose, and the results obtained are apt to be contradictory. Halliburton, summing up our knowledge of these matters, after mentioning the points already referred to. adds that when both proteids are present the proteid quotient in the blood corresponds roughly to that in the urine, but that the latter is the more variable of the two, and that their variations do not necessarily correspond. It has been stated that the proportion of globulin is notably high in cases of lardaceous disease of the kidneys. Occasionally a globulin has been found alone in the urine, as in a remarkable case recorded by Byrom Bramwell and Noel Paton, in which it was deposited in crystalline form.

A rough notion of the amount of globulin present may be obtained, as Sir William Roberts pointed out, by allowing the urine to fall drop by drop into distilled water, when, if the proportion of globulin is considerable, the path of each drop, as it sinks through the water, is marked out by a milky streak, owing to the fact that this proteid is insoluble in pure water.

For accurate determinations of the relative amounts it is necessary to extract and weigh the albumin and globulin separately. To all the ordinary albumin tests the two proteids behave alike, and they are precipitated together.

The conditions under which albuminuria occurs may be grouped under several fairly distinct headings.

1. The albuminuria associated with organic disease of the kidneys, such as acute and chronic nephritis.

In acute nephritis the amount of albumin passed is usually large, and in the later stages of the disease, if the condition has become chronic, even larger proportions of albumin may be reached, and the urine may become almost solid when boiled. In such cases the presence of abundant tube-casts and their characters, as well as the general symptoms, are of even more importance than the amount of albumin in forming a diagnosis.

In chronic interstitial nephritis the excretion of albumin is as a rule scanty, except in the later stages when signs of cardiac failure are superadded, or when there is evidence of supervening parenchymatous trouble. In lardaceous disease of the kidneys the amounts of albumin excreted differ widely in different cases, perhaps in connection with the presence or absence of parenchymatous changes.

Other gross renal lesions, such as infarctions, may also give rise to albuminuria, and in all cases urine containing blood will naturally yield the reactions of albumin.

In ordinary cases of acute nephritis 1 per cent of albumin is frequently present, but this quantity is often greatly exceeded, and amounts exceeding 5 per cent are sometimes observed.

2. Circulatory disturbances are among the frequent causes of albuminuria, which is one of the ordinary accompaniments of impeded venous outflow from the kidneys. The result is the same whether the impeded outflow be due to a general backward pressure in the venous system, such as occurs in connection with valvular heart disease or with emphysema of the lungs leading to hypertrophy and dilatation of the right ventricle, or whether it be due to a more local venous obstruction caused by pressure on the renal veins or inferior vena cava exercised by the pregnant uterus, abdominal tumours, or large accumulations of ascitic fluid. In such cases the escape of albumin through the kidneys may be attributable in part to the increased *vis a tergo*, and in part to changes in the glomerular epithelium resulting from deficiency of oxygen in the blood.

A diminished supply of blood to the kidneys may produce a similar effect, and to this cause has been ascribed the albuminuria which may accompany diseases attended with profuse diarrhœa, such as Asiatic cholera, but here toxic influence may very probably come into play.

3. In fevers, albuminuria of slight degree is a very common symptom, and not only in such fevers as are apt to set up an acute parenchymatous nephritis, such as scarlatina and diphtheria. Such albuminuria may be in part due to circulatory disturbances, but the production of a toxic nephritis in the fevers referred to, and the occasional occurrence of nephritis as a complication of other febrile disorders, suggests a toxic origin, and the cloudy swelling so often observed in the kidneys post-mortem bears witness to changes produced in the renal epithelium by diseases of this class. Febrile albuminuria is to some extent dependent upon the height of the fever, and still more upon the length of the period over which it extends. Thus in typhoid fever a slight albuminuria is extremely common.

4. When the ureter has been temporarily occluded, as for example by a calculus, the removal of the obstruction is followed by albuminuria. According to Max Hermann, such albuminuria has its origin in secondary disturbances in the renal circulation resulting from the distension of the pelvis of the kidney by the urine which accumulates behind the obstruction.

5. A very interesting group of cases of albuminuria embraces those connected with nervous disturbances, such as an epileptic fit, or more frequently a series of such fits occurring in rapid succession, or with graver accidents occurring in the central nervous system, such

as cerebral hæmorrhage. In considering such cases, which are not altogether easy of explanation, the possible existence of antecedent albuminuria must not be forgotten, and the special liability to cerebral hæmorrhage of patients who suffer from interstitial nephritis. In connection with epilepsy, the occasional occurrence of albuminuria in apparently healthy persons after violent exercise may be referred to.

6. Various poisons have the power of producing albuminuria, either by exerting a hæmolytic action and causing excretion of hæmoglobin, or, as is the case with cantharides, turpentine and phenol, by setting up a toxic nephritis.

7. There remains to be considered the so-called functional or physiological albuminuria. That minute traces of albumin are present even in normal urine has already been mentioned, and if, as Morner evidently believes, such traces have a renal origin, it would appear not unlikely that somewhat larger amounts, recognisable by the ordinary clinical tests, should occasionally be excreted apart from any serious deviation from conditions of health. Moreover, we might expect that individual differences would exist in the power of the kidneys to prevent the passage of albumin from the blood into the urine.

Such a view gains in likelihood from the fact that functional albuminuria is usually intermittent, being called into existence by definite causes which differ in different cases. Sometimes it is absent while the patient is recumbent, and reappears on his resuming an erect posture; in other cases albuminuria only occurs after meals, in which connection it may be noted that the very free taking of uncoagulated albumin by the mouth is capable of producing a temporary albuminuria in a normal individual. Sometimes it follows a cold bath, and sometimes severe muscular exertion—causes which are active excitants of hæmoglobinuria—a suggestive fact.

On the other hand, such influences exert a conspicuous effect upon the excretion of albumin by patients with actual renal disease, and there are many who hold that the so-called functional albuminuria is always pathological, and that persons who exhibit it, if not actually the subjects of renal disease, are likely to develop structural changes in the kidneys in the course of time. Possibly among the cases so classed some are of one kind and some of another. In childhood and early youth the tendency to albuminuria not infrequently ceases entirely, but occasionally evidences of cardio-vascular changes are present which probably indicate an actual renal lesion.

The slight albuminuria which often accompanies the passage of oxalate crystals calls for special mention, as it may be that this is due to mechanical injury by the crystals, the slightest degree of the chain of events which may culminate in the profuse hæmaturia which sometimes results from the free consumption of rhubarb.

The question of physiological albuminuria is still an open one, concerning which investigators are divided into widely separated camps, but its importance, especially in relation to questions of life insurance, admits of no question.

8. Lastly, albuminuria may occur independently of renal lesions as the result of admixture of albumin below the kidneys derived from the renal passages or bladder, or from the admixture of other secretions. Urine which contains pus is always albuminous even when the formed elements have been filtered off, and there are many sources from which pus may find its way into the urine, as in pyelitis, cystitis, tubercular disease, and gonorrhœa. Chyluria offers another example of non-renal albuminuria. The admixture of semen is another cause, which may be recognised by the microscopic detection of spermatozoa, and in females admixture of vaginal secretion is a not uncommon cause of the presence of small quantities of albumin in the urine.

The Mucus-like Substance of Urine.—That a true mucoid material occurs in small quantities in urine, and constitutes the main proteid constituent of the nubecula, has been demonstrated by K. A. H. Mörner, but the bulk of the substance found in urine which has properties like those of mucus is not of this nature. Huppert identified it as a nucleo-albumin, but in his more recent investigations Morner has shown that there exist in urine certain substances, viz. chondroitin-sulphuric acid, nucleic acid and taurocholic acid, which have the power of combining with albumin which may be present, with the formation of chondralbumins, nucleo-albumins, and tauralbumins. The albumin of normal urines is in such combinations, and as the combining substances are in excess, the addition of more albumin leads to further precipitation with acetic acid until all is exhausted, after which the excess of albumin remains uncombined. If any of the above acids be in excess their combination products may also be in excess, which probably explains the increase of mucin-like material in cases of jaundice, which may be attributed to an increased output of the bile acid.

The presence of such compounds is indicated by the formation of a turbidity when the urine is acidified with acetic acid in the cold. The precipitation is favoured by dilution of the urine, as the salts present tend to prevent it, and the same procedure removes the risk of the precipitation of urates by the acid.

The faint ring, situated 0·5 to 1 cm. above the junction of the liquids when Heller's test is performed, has been already alluded to, and this affords a further indication of the presence of the mucin-like substance.

Increased excretion of such material has been

observed in a variety of conditions, such as vesical catarrh, leukæmia, and nephritis, as also in various forms of poisoning and in fevers such as typhoid and pneumonia. Its occurrence in jaundice has already been mentioned. Further investigations will doubtless teach us more as to the clinical significance of its occurrence and its connection with renal disease on the one hand and disease of the mucous membrane of the urinary passages on the other.

Fibrinuria.—When hæmorrhage occurs into the urinary passages the presence of clots in the urine may be observed, and in a few instances coagula have been passed which contain only a few scattered blood corpuscles, as, for example, in a case of parenchymatous nephritis near its fatal ending, observed by Friedrich Müller, and as a result of cantharides poisoning in cases recorded by Senator and by Bartels.

In a case recently recorded by Trischitta, the urine, which was quite liquid when passed, readily coagulated after emission into a jelly-like mass, which could only be decanted with difficulty. The patient was a woman aged twenty-nine, who had suffered from pain in the region of the right kidney, but no satisfactory explanation of the fibrinuria was forthcoming.

It is, however, in chyluria that fibrinuria is most conspicuous, and in this condition the urine will sometimes clot so as to form a mass resembling blanc-mange.

Albumosuria (Peptonuria).—There is some confusion in the use of the terms albumosuria and peptonuria, which arises from the application of the name peptone to different substances.

The peptone of Kuhne, the ultimate product of the digestion of proteids, is characterised by the fact that although it yields the biuret and other proteid reactions, it is not precipitated by saturation of its solutions with ammonium sulphate. Several investigators have sought for such true peptone in normal and morbid urines without success, and the statement is usually made that it does not occur in the renal excretion unless after direct injection into the circulation. However, in a quite recent paper, Ito claims to have detected the peptone of Kühne, in association with albumose, in cases of pneumonia before and during the stage of resolution, in a case of advanced pulmonary phthisis, and also in puerperal cases.

The condition spoken of as peptonuria is characterised by the presence in the urine of a proteid substance agreeing in properties with the peptone of Brucke, and which is more correctly described as a deutero-albumose. Whereas some writers prefer the more correct name of albumosuria for this condition, others limit the use of this designation to cases in which proteids are present, which resemble in their properties the digestive proto- and hetero-albumoses. The proteid resembling hetero-albumose, which is now usually spoken of as the Bence-Jones proteid, from the name of its discoverer, has a special diagnostic importance, but is of very rare occurrence. On the other hand, no very definite clinical distinction can as yet be drawn between cases in which proto-albumose occurs and the much commoner ones in which the proteid present has the characters of deutero-albumose.

Detection of Albumoses in Urine.—When present in sufficient quantities the albumoses may be detected by tests applied directly to the urine, provided that albumin be absent. When albumin is present it requires to be got rid of as a preliminary measure before the tests for albumoses are applied.

The fact that a urine which is free from albumin yields the biuret test with copper-sulphate and an alkaline hydrate must not be relied upon as indicating the presence of albumose, as some non-proteid constituents of urine yield a pink tint with these reagents, and the chief of these is urobilin.

Nitric acid added drop by drop precipitates the Bence-Jones proteid and proto-albumose, and the precipitate so formed redissolves on heating and reappears on cooling. Deutero-albumose, on the other hand, is not so precipitated unless sodium chloride has been previously added. A precipitate of urates thrown down on the addition of the acid may simulate an albumose.

Salicyl-sulphonic acid is a very useful reagent for the detection of albumoses, as the precipitates which they yield are dissolved on heating and reappear on cooling.

For the preliminary removal of albumin mere boiling does not suffice, and the removal is best effected by precipitation with ferric chloride from the neutralised urine.

For the precipitation of albumoses, where scantily present, phospho-tungstic acid or tannin may be employed, or Devoto's method, which is based upon the fact that albumin and globulin become completely insoluble when boiled with a saturated solution of ammonium sulphate, whereas the precipitate of albumose so obtained may be redissolved in water. This method has the advantage that no special proceeding for the removal of albumin is required. The recognition of the Bence-Jones proteid will be spoken of later.

Clinical Significance of Albumosuria.—By the researches of a number of observers, among whom von Jaksch, Hoffmeister, Devoto, and Sidney Martin may be mentioned, a mass of information as to the clinical significance of albumosuria or peptonuria has been collected. As has been already pointed out, the presence of albumoses is not indicative of renal disease, but of their presence in the blood. Huppert has well pointed out that the excretion of albumoses in the urine indicates the breaking down of albuminous tissues somewhere in the body. Thus albumosuria may occur during the

early days after delivery, when its appearance may be attributed to the rapid involution of the uterus. In another class of cases it results from the tissue changes which occur in an infarcted area, from which the blood-supply has been cut off by the lodgment of an embolus in the artery which feeds it. The rapid breaking down of tissues which occurs in the liver in cases of acute yellow atrophy and phosphorus-poisoning may also cause albumosuria, and it has occasionally been met with in cases of cirrhosis hepatis. A like result may follow from degenerative changes in malignant growths, and in cases in which the intestines are the seats of extensive ulcerative lesions.

When exudations rich in leucocytes are undergoing absorption the conditions favourable to the production of albumosuria are naturally present, and both in cases of pneumonia during the stage of resolution, and in connection with suppurative processes, such as empyema and bronchiectasis, the phenomenon is often to be observed. These are indeed the most important varieties of albumosuria from the clinical standpoint.

It should be mentioned that some observations which have been recorded are not easily to be explained on the above view, as, for example, the occasional detection of albumoses in urine in the earlier stages of pneumonia, and Fischel's observations on the occurrence of albuminuria in pregnancy for some weeks before delivery.

Histon has been detected in the urine by Krehl and Matthes in cases of peritonitis, pneumonia, erysipelas, and scarlatina, and by Kolisch and Burian in a case of lymphatic leukæmia.

The Bence-Jones Proteid.—In the rare cases in which this substance is met with in urine it is usually present in large amount, and may be recognised by methods which are of easy application in ordinary clinical work. In Bradshaw's case it was partly in the form of a solid sediment. Unlike deutero-albumose it is precipitated on heating the urine, which becomes turbid at the low temperature of about 40° C. On further heating a bulky flocculent precipitate is formed which clings with peculiar tenacity to the sides of the containing test-tube. Precipitation is complete at about 58°-60° C. On further heating, and especially if a few drops of acetic acid have been added, the precipitate almost completely or completely disappears, and the urine is left nearly or quite clear. The low temperature at which the precipitate forms and the behaviour on further heating are quite characteristic and serve to differentiate the Bence-Jones proteid from all other proteids met with in urine. With Heller's nitric-acid test the substance yields a ring as do albumin and globulin.

The nature of the Bence-Jones proteid has been specially studied by Magnus Levy, who arrived at the conclusion that, in spite of its resemblance to hetero-albumose, its proper place is among the albumins rather than the members of the albumose group. It is interesting to note that Zülzer has found a proteid with identical properties in the urine of dogs poisoned with pyridin.

The clinical importance of the Bence-Jones proteid is due to its association with myelomatous and myelosarcomatous growths in the bony skeleton, such growths having been present in cases which have come to post-mortem examination, and have been successfully diagnosed during life on the ground of the presence of this substance in the urine. Until the last few years only some five cases of its occurrence were known, but of recent years the subject has received much attention from Bradshaw and others in this country, and also from continental observers, with the result that fresh examples have rapidly multiplied. These later observations have tended to supply confirmatory evidence of the special diagnostic significance of the condition.

Hæmoglobin.—The last proteid to be considered is hæmoglobin, but as hæmaturia and hæmoglobinuria have both been treated of in special articles (*q.v.*) they need only be briefly referred to here. It will suffice to recall the fact that hæmaturia implies hæmorrhage from some portion of the urinary tract, whereas the cause of hæmoglobinuria lies behind the kidneys, in the presence of free hæmoglobin in the blood plasma.

Urines which contain hæmoglobin yield the ordinary tests for albumins, and the failure to obtain positive results by these tests suffices to exclude its presence.

The blood-pigment may be present in the urine either as oxyhæmoglobin or as methæmoglobin, and often both are present together. The presence of methæmoglobin is recognised by the dark absorption band in the red which forms the most conspicuous feature in its spectrum, and the familiar absorption bands of oxyhæmoglobin are usually conspicuously visible, especially in urines which have a pink colour.

Methæmoglobin may become transformed into oxyhæmoglobin when the urine is allowed to stand, and the colour of the liquid changes, at the same time, from brown to pink. In alkaline urines, which often have a peculiar livid tint, some reduction of oxyhæmoglobin takes place in the deeper layers, which often exhibit the bands of reduced hæmoglobin and of oxyhæmoglobin in superposition. The spectroscopic test is of great value for the recognition of blood-pigment, but it must be borne in mind that, when only a trace is present, and that in the form of methæmoglobin, the absorption bands may be very faint and quantities may be over-

looked which are readily detected by the guaiacum test.

This latter test is of great delicacy, but may mislead, owing to the fact that other substances met with in urine yield a similar blue colour, but a negative result is of much value in excluding the presence of hæmoglobin, always providing that the reagents are freshly prepared and active.

A few drops of tincture of guaiacum are added to some ozonic ether (an ethereal solution of hydrogen peroxide), and the mixture is allowed to run upon the surface of the urine. If blood be present a blue ring appears in the ether near the junction of the liquids, and on gently shaking the whole layer of ether becomes blue.

The urine of patients who are taking iodide of potassium yields the blue colour with great intensity, but the reaction is more slowly developed.

Heller's test is performed as follows:—To some urine in a test-tube an equal volume of liquor potassæ is added, and the mixture is boiled. The flocculent phosphatic precipitate which forms is coloured brown, and on further boiling usually assumes a pink tint. At this stage, as Arnold has pointed out, the spectrum of hæmochromogen, which is easy of recognition, is clearly seen when the phosphatic precipitate is examined with the spectroscope. When this is the case the presence of blood-pigment may be confidently affirmed.

Chrysophanic acid, which occurs in the urine of patients taking rhubarb or senna, imparts a pink tint to the phosphate precipitate, which however, shows no definite absorption bands; hæmatoporphyrin is also carried down, but is seldom present in such quantities as are likely to lead one into error.

The microscopic detection of red blood corpuscles is the only certain test of hæmaturia, as distinguished from hæmoglobinuria.

Pyuria.—The presence of pus in the urine forms the subject of a special article, *q.v.*, vol. viii.

VI. The Chief Nitrogenous Constituents of Urine

The Total Nitrogen.—A healthy adult excretes some 10-16 grammes of nitrogen in his urine in the course of twenty-four hours. Of this the great bulk (84-90 per cent) is contained in urea, and the remainder is distributed amongst a number of other constituents, such as uric acid, kreatinine, the xanthin bases, ammonia, indoxyl compounds, and the pigments, and, under morbid conditions, leucin, tyrosin, the diamines, cystin, and albumin. The nitrogenous constituents are for the most part products of proteid metabolism, and their amount is largely determined by the quantity of nitrogenous materials contained in the food, provided that fats and carbohydrates are at the same time given in sufficient amounts. However, the output of urea which follows a meal is not directly derived from that meal, but chiefly from the metabolism of the tissue proteids. Under ordinary conditions, when a fixed diet is given a condition of nitrogenous equilibrium is established, the output of nitrogen in the urine being equal in amount to the intake after subtraction of the quantity contained in the fæces; or, in other words, the nitrogen in the urine and fæces equals that taken in the food. If, within certain fairly wide limits, a person in whom such nitrogenous equilibrium has been established is placed upon a diet richer or poorer in proteids than that which he was previously taking, the organism will adapt itself to the altered conditions within a few days, and nitrogenous equilibrium will be re-established upon a higher or lower plane.

The administration of excess of carbohydrates and, in a less degree, of fats to a person in nitrogenous equilibrium tends to spare the tissue proteids, and reduces the nitrogenous output, and a diminution of these constituents of the diet has a contrary effect. In fevers, as was first shown by Huppert, the output of nitrogen is greatly increased beyond that of healthy persons on a similar diet, and an increase is observed in other conditions which lead to increased proteid metabolism, such as *diabetes mellitus* and malignant disease. Although urea contributes so large a proportion of the total nitrogen, the results of estimations of these two factors do not altogether agree, seeing that under a variety of conditions the relative proportions of the several nitrogenous constituents of the urine undergo conspicuous variations. This is specially marked in certain grave hepatic diseases, in which the output of urea may undergo a remarkable relative diminution, whereas there is usually a compensatory increase of the ammonium salts.

In renal disease a diminution of the total nitrogen below the quantity corresponding to the amount ingested results from a diminished eliminative power of the kidneys. Von Noorden and Ritter found that this retention of nitrogenous products was not continuous in chronic renal diseases, but that a period of low excretion is followed by one of normal or increased output. This may account for the conflicting results which have been obtained in observations which have usually extended over short periods only. Von Noorden points out that in cases of chronic parenchymatous nephritis the retained nitrogen may amount to several grammes per diem, and that if such retention were continuous enormous accumulation would result, seeing that the excretion by other channels, such as the skin, is very limited. Vogel and Schmoll found that a similar diminished excretion of total nitrogen occurs in gouty persons apart from any clinical evidence of renal disease. Magnus Levy con-

firmed this for the convalescent period, but observed, on the other hand, a greatly increased output of nitrogen during the acute attack.

For the estimation of the total nitrogen excretion Kjeldahl's well-known method is employed.

Urea.—Some 30 grammes of urea are excreted by a healthy adult in the twenty-four hours, forming about 2 per cent of the urine.

The estimation of urea has become a common clinical proceeding, and most hospital wards are provided with the necessary apparatus for the purpose, but it is permissible to doubt whether the results obtained are, as a rule, of much clinical value. It will be clear from what has gone before that, in forming any conclusions from the urea excretion, the amount of nitrogen contained in the food of the patient must be taken into account, and in renal cases this is as a rule much below the usual quantity. It is hardly necessary to point out that unless the total urine of the twenty-four hours has been collected and measured, and unless the specimen employed has been taken from this total, the results obtained are of no value at all.

In fevers the output of urea is markedly increased, and a still more conspicuous increase, up to 100 grammes or more in the day, is observed in *diabetes mellitus*. The increase in fevers is usually associated with a diet poor in nitrogen, whereas in diabetes the contrary is the case. However, the great increase in the latter disease is far too pronounced to be wholly attributable to the proteid diet, and can only be referred to an abnormally active tissue metabolism. In fevers the increase doubtless has a similar cause, but it is not proportionate to the height of the fever, and in diseases ending in a crisis, such as pneumonia, the maximum excretion may occur shortly after the crisis.

Diminished excretion of urea is observed in some nervous diseases and has been often noticed in Addison's disease, in which it perhaps has a nervous origin. Otherwise a marked diminution of the urea output is usually due to one of two causes, viz. diminished formation, such as results from certain diseases of the liver, which organ is the chief seat of urea formation, or diminished excretion, such as occurs in various forms of renal disease.

The diminished output of urea in hepatic diseases reaches its most conspicuous development in cases of acute yellow atrophy, in which the excretion may be practically abolished. It is seen in a less pronounced form in acute phosphorus poisoning, and in some cases of cirrhosis hepatis, in which disease, as is well known, the hepatic parenchyma may undergo marked degenerative changes. Under such conditions the abnormal nitrogenous substances leucin and tyrosin may make their appearance in considerable quantities, and the decrease of urea is usually attended by an increased excretion of ammonia, which may either be due to a failure of the conversion which it normally undergoes in the liver, or may result, as in diabetes, from the formation of abnormal acids.

As regards parenchymatous and interstitial nephritis, what has been said of the total nitrogen excretion applies equally to that of urea, which may be below the normal, or even increased at different periods in a given case. Some importance has been attached to the relative excretion of phosphates and of urea in renal disease, as affording evidence of the portions of the renal epithelium chiefly affected. Seeing that the excretion of phosphates takes place from the glomeruli, and that of urea from the renal tubules, we should expect a nephritis which is chiefly glomerular to specially affect the phosphatic output and one chiefly tubular that of urea. Such differences have been actually observed.

The method employed for the estimation of urea in clinical work is based upon its decomposition by sodium hypobromite with the liberation of nitrogen and carbon dioxide. The absorption of the carbon dioxide is ensured by the presence of an excess of sodium hydrate in the hypobromite solution, and the nitrogen is collected and measured over water. The hypobromite solution employed should always be freshly prepared.

Various forms of apparatus have been devised for such estimations, and these will be found described in works on urinary analysis. In order that the results obtained may have an approach to accuracy, the readings of the column of nitrogen should be corrected, at least for the temperature of the air, if not for barometric pressure and the tension of aqueous vapour. The apparatus is usually so graduated that the percentage of urea in the urine tested may be read off directly.

Uric Acid.—The excretion of uric acid by healthy adults varies within somewhat wide limits, viz. from 0·2 to 1·25 grammes in the twenty-four hours. The amount is greater with a flesh than with a vegetable diet, and recent researches render it highly probable that, as Horbaczewski first pointed out, the nuclein of the food and tissues is the source of the uric acid excreted in the urine, and that it is not a product of proteid metabolism in general. Glandular structures such as thymus, which are rich in nuclein, have a specially conspicuous effect in increasing the excretion of uric acid when they are taken as foods. Owing to their sparing solubility uric acid and its salts are apt to proclaim their presence in the urine and tissues, and have consequently attracted much attention from clinical observers and chemical pathologists alike.

Whilst it is the view of the majority that the deleterious effects which uric acid produces in the organism are limited to those which result

from its deposition, or that of its salts, in solid form, there are some who maintain that this substance exerts a toxic action even when in solution in the body fluids, and that to its presence there a large number of morbid symptoms are due. Haig lays great stress upon the relative variations in the output of uric acid and urea, as affording evidence of the retention of uric acid in the tissues, or the expulsion therefrom of quantities previously retained.

The excretion of uric acid is increased in febrile diseases, and a marked increase has been sometimes observed in cases of pernicious anæmia. The most conspicuous increase is met with in leukæmia, but even in this disease quantities as large as 4 or 5 grammes in the twenty-four hours, although occasionally met with, must be regarded as exceptional. More usually the output is about twice the maximum normal amount. In leukæmia there is a relative as well as an absolute increase, and the proportion of urea to uric acid may be as low as 9 : 1 instead of 50 : 1, which is about the average normal ratio.

Owing to the part which uric acid undoubtedly plays in the pathology of gout, a peculiar interest attaches to its excretion in that disease, but the results of investigations directed to this point are far from being as conclusive as could be wished, or as was foreshadowed by some of the earlier investigations in which the unsatisfactory method of Heintz was employed. The variations observed are by no means extreme, and the results are by no means uniform in different cases. Moreover, the wide individual differences in the uric acid excretion of normal subjects render it peculiarly difficult to estimate the importance of any but very marked variations. Indeed von Noorden, writing in 1893, expressed the opinion that the figures obtained in gouty cases show no important differences from those obtained with healthy persons and sufferers from various diseases other than gout. A study of the results obtained by trustworthy methods of estimation, and especially those of Fawcett, point to a low average excretion of uric acid by gouty patients in the intervals between attacks, and an increased output during acute attacks. The results obtained in cases of chronic nephritis are not more conclusive as regarding the retention of uric acid by diseased kidneys.

The deposition of amorphous urates and of crystalline uric acid from the urine has no necessary connection with excessive excretion, although this is doubtless one of the causes of such deposition. The other causes which contribute to the result will be spoken of in the sections which deal with urinary sediments and with calculi respectively. The estimation of uric acid has of recent years been deprived of much of its difficulty, and in Hopkins's method we have a means of making such estimations simply and with as much accuracy as is attained to with more complex methods. It is based upon the fact that the uric acid is completely precipitated in the form of ammonium urate when the urine is saturated with ammonium chloride. From the precipitate of ammonium urate, dissolved in a small quantity of hot water, the uric acid is set free by means of hydrochloric acid. The precipitated uric acid is collected upon a filter and washed, and afterwards dissolved by means of sodium carbonate and titrated, in the presence of sulphuric acid, with a vigintinormal solution of patassium permanganate.

The Xanthin Bases.—Several different bases allied to uric acid and often classed with it under the general name of alloxuric substances, constitute a very small fraction of the total nitrogenous constituents of urine. Of these xanthin is the most important, and has been met with as a crystalline sediment, and in a very rare variety of calculus.

Much study has been devoted, of recent years, to the variations in the excretion of such bases, sometimes by methods which are not satisfactory. As yet the fluctuations in their amounts have hardly acquired any definite clinical importance, and the methods employed for their estimation are complicated and difficult.

Like that of uric acid, the output of the xanthin bases is increased by a diet rich in nuclein, and in connection with leucocytosis. In leukæmia a marked increase has been frequently but not constantly observed, and it should be mentioned that Babinsky found their amounts excessive in cases of acute nephritis. The increase described by Kolisch as occurring in gout has not been met with by other observers.

Kreatinine.—As compared with those of uric acid, the variations in the excretion of kreatinine in the urine have attracted but little attention from clinical workers. It is never thrown down as a sediment from the urine, and no untoward results can be ascribed to its presence in excess.

Neubauer found that in health the kreatinine excreted by the kidneys in periods of twenty-four hours varied between 0·6 and 1·3 grammes, or was, roughly speaking, 1 gramme. Macleod has recently obtained higher figures, viz. an average of 2·098 on flesh diet, and 1·064 on a diet free from kreatine.

The kreatinine of urine is partly exogenous and partly endogenous, the former portion being derived from the kreatine of food, the latter being a product of tissue metabolism. It is therefore important that in studying the variations of the endogenous excretion the patient should be placed upon a kreatine-free diet.

An increased output has been observed by Grœco and Moitessier after violent muscular exertion, and it may be stated generally that the amount is increased when metabolism is active, and diminished when it is sluggish, and

the excretion shows a parallelism to that of urea. During acute fevers, such as pneumonia and typhoid fever, the excretion of kreatinine is increased, but it falls below the normal during convalescence from these diseases. In anæmic states, in marasmus, and in association with tuberculous processes, a diminution has been observed.

In cases of progressive muscular atrophy, and in pseudo-hypertrophic paralysis, a diminished output has been observed, but Macleod has failed to find such diminution in cases of muscular atrophy. Macleod has recently demonstrated a very conspicuous diminution in cases with marked splenic enlargement. This diminution, which attains to as much as 50 per cent of the normal excretion on kreatine-free diet, was equally conspicuous in cases in which the leucocytes of the blood were diminished in number, and in those in which there was marked leukæmia.

The presence of kreatinine in the urine is easily demonstrated, but its mere detection is of no clinical value. For its quantitative estimation advantage is taken of the fact that it forms a sparingly soluble compound with zinc chloride. For a description of the processes available the reader is referred to works on urinary analysis.

It is interesting to note that it was the green fluorescence observed when working at the estimation of kreatinine by a zinc chloride method, that led Jaffé to the discovery of urobilin, to the compound of which pigment with zinc chloride that fluorescence is due.

VII. Some Abnormal Nitrogenous Constituents. Diamines, Leucin, and Tyrosin

Diaminuria.—Of recent years two of the members of the class of diamines, viz. cadaverine or pentamethylene diamine, $H_2N(CH_2)_5NH_2$, and putrescine or tetramethylene diamine, $H_2N(CH_2)_4NH_2$, have been repeatedly met with in the urine in cases of cystinuria, and sometimes in the fæces of cystinuric patients also. The original case in which this association was observed by Baumann and Udranszky was peculiar in the constancy with which the diamines were present both in urine and fæces over a period of years. In some of the recent cases they have only been found at intervals, and in some not at all in a few observations. In no other instance has the same high proportion of diamines been found in the fæces. The two bases have sometimes been found in association, more often cadaverine has alone been found, and very seldom putrescine alone has been isolated. In the case referred to the quantities obtained amounted to some 0·2-0·4 grammes of the benzoyl compounds of the mixed diamines in the urine of twenty-four hours. In another case examined by P. J. Cammidge and the present writer similar quantities were obtained from the urine on two occasions only during a series of examinations extending over about a month.

In no other condition have these diamines been found in amounts capable of detection in the urine of a single day, but in large quantities of the urine of patients with pernicious anæmia, collected and concentrated down, William Hunter was able to detect their presence in smaller quantities. From normal and most morbid urines they appear to be wholly absent.

It is impossible to avoid the conclusion that some intimate connection exists between the excretion of cadaverine and putrescine and cystinuria, but no approach has yet been made to a solution of the problem of the nature of this connection.

The occurrence of the diamines in the fæces of their patient in larger quantities than in his urine naturally led Udranszky and Baumann to attribute their formation to bacterial action in the intestine, seeing that they are well-known products of bacterial activity. There are, however, no grounds for supposing that cystin is formed in the intestine, and there is no question that the excretion of cystin may continue unchecked when the diamines are absent for a time both from urine and fæces. Morcigne and C. E. Simon have, on the other hand, suggested that in these cases the diamines, as well as cystin, may be products of tissue metabolism.

Two methods are in use for the detection of diamines in urine. That of Udranszky and Baumann involves the shaking of the total day's urine with benzoyl-chloride and an excess of sodium hydroxide solution. From the precipitate formed, which consists in part of earthy phosphates, the benzoyl compounds of the diamines are extracted with hot alcohol. On pouring the alcoholic solution into water the benzoyl diamines, if present, separate in crystalline form.

In the alternative process of Stadthagen and Brieger the diamines are obtained in the form of crystalline picrates.

Leucin and Tyrosin.—These are abnormal constituents of urine which are usually met with in association. Both are members of the class of amido-acids, but while leucin belongs to the fatty series, being amido-caproic acid ($C_6H_{13}NO_2$), tyrosin is an aromatic compound, viz. par-oxyphenyl-amido-propionic acid. Both are products of proteid decomposition.

It is especially in cases of acute yellow atrophy of the liver that these substances appear in large amounts in the urine, so that their presence becomes an important clinical feature of that disease. In cases of acute phosphorus-poisoning they are occasionally met with, but in smaller quantities. Their occurrence in phosphorus-poisoning has even been denied, and von Jaksch has very seldom met with them in such cases.

In severe febrile disorders, such as variola,

they have also been found, as also in the urine of pernicious anæmia by von Noorden and Laache, and in leukæmia by Prus.

In acute yellow atrophy of the liver as much as 1·5 gramme of tyrosin has been present in the urine of twenty-four hours.

The view generally accepted of the cause of their so abundant presence in this disease is that leucin and tyrosin are intermediate products of proteid metabolism, and that under normal conditions the nitrogen contained in them passed through the stage of ammonia, to take part in the formation of urea in the liver. When, however, the liver cells have undergone extensive destruction these further changes are escaped, and leucin and tyrosin appear in the urine. This view receives much support from the very conspicuous diminution of the urea excretion in the disease in question. Von Noorden, on the other hand, is inclined to attribute their appearance to the action of bacteria in the tissues, and ascribes the smaller quantities found in phosphorus-poisoning and severe anæmias to absorption of leucin and tyrosin from the intestine, and failure of the ordinary destructive processes.

Under normal conditions tyrosin administered by the mouth certainly does not appear as such in the urine, and even the benzine ring contained in it is broken up. The only exception to this rule is in the rare condition known as alkaptonuria, which will be described in a later section (p. 294).

When tyrosin is present in considerable amounts some of it is usually deposited from the urine as a crystalline sediment, whereas leucin is very seldom so deposited.

For their detection the following method may be employed:—The urine of twenty-four hours is precipitated with basic lead acetate and filtered. From the filtrate the lead is removed by passing a stream of sulphuretted hydrogen through it. The lead sulphide is filtered off, and the liquid is evaporated to a syrupy consistency. On standing leucin and tyrosin crystallise out, the former in needles, the latter in greenish spherules. It is usually better to remove the bulk of the urea present in the residue with a small quantity of absolute alcohol. The residue is then boiled with dilute ammoniacal alcohol, the solution is filtered, evaporated to a small bulk, and allowed to stand.

Their crystalline forms usually suffice for the recognition of leucin and tyrosin.

The needles of tyrosin are insoluble in acetic acid, but dissolve in ammonia and in hydrochloric acid; but if the quantity suffices, and if full certainty is desired, some of the special chemical tests should be applied.

VIII. Sugars in Urine

Glucose.—Seeing that diabetes mellitus and glycosuria have been dealt with in separate articles, it will be unnecessary to enter here upon any discussion of the conditions which lead to the presence of glucose in the urine, and it will only be necessary to refer to the chief tests for its detection which are in use for clinical purposes.

The simplest of these, known as Moore's test, is not a very delicate one, but is sometimes useful when the reagents for better tests are not available. To some of the suspected urine, placed in a test-tube, some liquor potassæ is added, and, after mixing, the upper portion of the column of liquid is boiled. If sugar be present a deep brown colour is produced by its decomposition. It must be remembered that even normal urine shows a distinct darkening of tint under such treatment, and only the development of a pronounced brown colour affords evidence of the presence of sugar.

In the remaining tests advantage is taken of one or other of the conspicuous properties of glucose, viz. its power of reducing metallic salts, of undergoing fermentation, of rotating the polarised ray, and of forming with phenylhydrazin an insoluble crystalline compound, glucosazone.

The reduction tests are of great service, but it must not be forgotten that they only afford evidence of the presence of a reducing substance, which is not necessarily sugar, but which, if present in large amount, is probably of that nature.

Trommer's test is performed as follows:—The urine is rendered strongly alkaline with liquor potassæ and a solution of cupric sulphate is added, drop by drop, until the precipitate formed is not wholly redissolved. If sugar be present a reduction of the copper salt to cuprous hydrate occurs on heating and an orange-red precipitate appears.

It is more convenient to employ freshly mixed Fehling's solution for the test. The Fehling's solution and the urine should be heated in separate test-tubes and mixed when hot. The mixture may be further heated if necessary.

If sugar be present in considerable quantity the reduction occurs before the liquid is raised to the boiling-point, and when this is so the presumption that it is due to the presence of sugar is very strong. When, on the other hand, sugar is only present in traces it may be necessary to boil the liquid before a scanty reduction occurs, often with the production of only a pale green opacity. Under such circumstances the presence of sugar requires to be confirmed by other tests, as even normal urine contains reducing substances, among which kreatinine and uric acid are the chief. Reducing substances also appear after the administration of certain drugs, such as salicylic acid, chloral, chloroform, and glycerine, and among other reducing agents lactose, glycuronic acid, and pyrocatechin call for mention. Homogentisic acid is an energetic reducing agent, and alkapton urines, in which

alone it is present, react with copper solutions as readily as glycosuric ones. However, alkaptonuria is extremely rare, and the conspicuous blackening by the alkali, which occurs at the same time, is very characteristic.

The bismuth test of Almén and Nylander is not very often employed in this country. The reagent is prepared by dissolving 4 grammes of sodium potassium tartarate in 100 c.c. of an 8 per cent solution of sodium hydroxide, and, after warming, basic nitrate of bismuth is added as long as it is dissolved. One part of the test solution is added to ten parts of the suspected urine, and the mixture is boiled for a few minutes. If sugar be present the liquid blackens. This test is not applicable to albuminous urines, as in them the black sulphide of bismuth is apt to form, and other reducing substances met with in urine yield a positive reaction, as also does chrysophanic acid derived from rhubarb or senna. On the other hand alkapton urines do not respond to this test.

The phenyl-hydrazin test, introduced by von Jaksch, is thus performed:—Two parts (von Jaksch describes as a part as much as can be taken on the point of a knife) of chemically pure phenyl-hydrazin hydrochloride and three parts of sodium acetate, are added to 6-8 c.c. of the urine in a test-tube. The test-tube is warmed, and if solution is not complete some water is added. The test-tube is then allowed to stand in boiling water for half an hour, or better for an hour, and is then placed in a beaker of cold water. On cooling a yellow crystalline precipitate of phenyl-glucosazone falls, and this precipitate consists of yellow needles arranged in rosettes, which are larger if the liquor is allowed to cool more slowly.

The test is an extremely delicate one, almost too delicate indeed, seeing that crystals of phenyl-glucosazone may even be obtained from normal urines. Other sugars and glycuronic acid yield similar crystalline compounds, which may be distinguished by their crystalline forms, or much better by their melting-points, which may be determined after the crystals have been removed, washed, and dried. The melting-point of pure phenyl-glucosazone is 204°-205° C., but impurities may, of course, cause a considerable lowering of the melting-point.

The fermentation which it undergoes with yeast offers one of the surest ways of detecting sugar in urine, and also supplies a simple and fairly accurate means of estimating its amount. This is the principle of Roberts's method, which is so extensively employed in our hospitals. Specimens of the urine are placed in two bottles provided with grooved corks to allow of the escape of gases. Into one of the bottles a small piece of German yeast is introduced, and both are allowed to stand in a warm place for twenty-four hours. After the bottles have cooled the specific gravity of both specimens is taken, and the loss of specific gravity of the fermented as against that of the control specimen affords a measure of the amount of sugar which has been destroyed. A difference of 1° corresponds roughly to one grain of sugar in the ounce of urine. Care must be taken that the bottles do not become overheated, with the result that the activity of the yeast is inhibited. Estimation by measurement of the carbon dioxide formed is less satisfactory, but the collection of the gas forms a ready method of demonstrating the occurrence of fermentation. It must be remembered, however, that a small quantity of gas is formed by the auto-fermentation of yeast.

More accurate results are obtained by the titration of urine with Fehling's solution.

The solution in question is made up in two portions, one of which is prepared by dissolving 34·64 grammes of cupric sulphate in 500 c.c. of water, the other by the addition of 173 grammes of sodium potassium tartarate to 100 c.c. of sodium hydroxide solution (sp. gr. 1·34) and dilution to 500 c.c. The two solutions should be mixed in equal parts before use. The fresh admixture is of importance, because when the mixed Fehling solution is kept for some time reduction is apt to occur on boiling, owing to a partial conversion of tartaric into racemic acid. Such reduction may give rise to serious diagnostic errors.

The urine is placed in a burette and, if rich in sugar, should be diluted with water to a known extent. 10 c.c. of Fehling's solution, diluted with some 40 c.c. of water, are placed in a porcelain dish and are kept boiling during the process of titration. When the urine is run into it gradually from the burette the familiar orange precipitate of cuprous oxide is thrown down and the blue colour of the solution becomes paler. Eventually, when reduction is complete, the liquid above the precipitate is rendered colourless, and the amount discharged from the burette is then read off. A preliminary rough determination of the reducing power should first be made, after which the urine may be further diluted to a known extent if it appear desirable. Owing to the presence of the orange precipitate the exact point at which the blue tint disappears is rather difficult to estimate, and the following means may be resorted to:—Upon a porcelain tile a number of drops of a solution of potassium ferrocyanide are placed by means of a glass rod, and to each a drop of acetic acid is added by the same means. When reduction approaches completion a drop of the clear liquid above the precipitate is removed by the stirring rod and added to one of the drops upon the tile. If a brown colour results the copper is not completely reduced, and after the further addition of urine from the burette the test may be repeated until no further brown colour is obtained.

Dilution of diabetic urine to five or ten times its volume is usually desirable, and the amount

of diluted urine required to reduce the 10 c.c. of Fehling's solution used will contain 0·05 gramme of glucose. From this the amount present in a given quantity of the original urine is readily determined.

Pavy's solution, which is a modification of Fehling's with the addition of ammonia, has the advantage that no precipitate is formed with it, the cuprous oxide being held in solution by the ammonia and the change of tint being readily observed. It has, however, the disadvantage that ammonia fumes are abundantly given off by the boiling liquid. This can be overcome, at the cost of some complication of the apparatus, by carrying out the reduction in a corked flask provided with an escape tube for the fumes.

The rotation of the polarised ray may also be used for the estimation of sugar in urine by means of the saccharimeter, which will be found described in works on urinary analysis. It is advisable to decolorise the urine, as a preliminary step, by means of alumina paste and subsequent filtration, and the results obtained by this method are impaired by the presence of other reducing substances, such as glycuronates. In diabetes the presence of considerable quantities of the lævorotatory β oxybutyric acid may greatly counteract the dextro-rotatory influence of the glucose and introduce important errors.

For other volumetric methods, such as those of Knapp, in which a standard alkaline solution of mercuric cyanide is employed, the reader is referred to the text-books.

Lactose or milk sugar is often met with in small quantities in the urine of nursing women, and more abundantly when suckling is abruptly discontinued, as is not infrequently the case on admission to a hospital. The recognition of the occurrence of lactosuria under such conditions is important for the avoidance of errors of diagnosis. When the conditions which lead to lactosuria are present it may be recognised with sufficient certainty by the facts that the urine reduces Fehling's solution, although not very readily, and yields a positive reaction with the bismuth test, that with phenyl-hydrazin a comparatively soluble osazone is formed, and that no marked fermentation is produced by yeast. Absolute certainty is only to be obtained by the isolation of lactose. Lactose, like glucose, is dextro-rotatory.

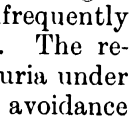
Other sugars of the hexose class, such as lævulose, laiose, and isomaltose, have been detected in urine, but are not of any special clinical interest, nor is it necessary to speak here of inosite, which has been found in the urine in cases of diabetes insipidus.

Pentose (optically active or inactive arabinose), which was first detected by Salkowski in the urine of patients addicted to the morphia habit, may even occur in normal urine, a fact which von Jaksch is inclined to attribute to the presence of pentose in beer. It has also been found in the urine of diabetics. Urines containing pentose reduce Fehling's solution and yield osazones with phenyl-hydrazin, but do not give the fermentation test.

The most satisfactory test for pentose is the following (Bial's test):—3-5 c.c. of urine is heated with twice its volume of orcin-reagent, which consists of 500 c.c. concentrated hydrochloric acid, 1 grm. orcin, 20-30 drops of a 10 per cent solution of ferric chloride. As soon as the liquid begins to boil a greenish-blue colour appears, which becomes intensified on standing. The pigment may be extracted with amyl-alcohol, and the amylic extract shows an absorption band between the C and D lines. Glycuronic acid yields a violet amylic extract, which, however, shows a similar absorption band.

IX. Acetone, Aceto-Acetic Acid (Diacetic Acid), and β Oxybutyric Acid

The three substances are closely allied in their origin in the body, and are all apt to be present in considerable quantities in the urine of patients suffering from the graver forms of *diabetes mellitus*. Clinically their abundant presence is found to be a frequent precursor of diabetic coma, and there is good reason to believe that the accumulation of β oxybutyric acid in the blood is the actual cause of diabetic coma. It is further probable that it is simply in virtue of the acid properties of this substance that this effect is produced. It is a fact which has important practical bearings that their production is favoured by the elimination of carbohydrates from the diet of the patients.



Acetone, the lowest member of the ketone series, has been shown by von Jaksch and others to be present in small amount (up to 0·01 grm. in the twenty-four hours) in normal urine. It has usually been held that it is a product of the breaking down of the proteids, both of the food and of the tissues, for it appears in the urine in markedly increased amount during fasting and when carbohydrates are withdrawn from the diet, only to reappear when they are again taken in sufficient quantities. Hirschfield maintains, indeed, that except in grave cases of diabetes the increased excretion of acetone observed under normal conditions is dependent upon dietary causes. Von Jaksch, to whose labours we owe much of the knowledge which we possess of acetonuria, distinguishes the following varieties of pathological acetonuria:—A febrile acetonuria, that occurring in diabetes; acetonuria in certain forms of carcinoma which had not yet produced cachexia; acetonuria due to inanition, and to autointoxication.

The excretion of acetone in severe cases of

diabetes may reach as high a figure as 5 grammes in the twenty-four hours, and is not dependent upon the amount of sugar; nor does it run parallel with the nitrogenous excretion. When present in such large amounts its characteristic odour is noticed both in the urine and in the breath of the patient.

In fevers some degree of acetonuria is an almost constant phenomenon, and it would appear that acetone is more apt to be formed by children than by adults. In pneumonia it is usually present, but it may be stated generally that febrile acetonuria is dependent rather upon the persistent high temperature than upon the nature of the disease to which the febrile disturbance is due. Hence this form has no important diagnostic significance. The presence of acetone may sometimes be detected in the urine as such, but it is usually necessary to obtain a more concentrated solution by distillation, and to test the distillate. A considerable proportion of the acetone present comes over in the first portion of the distillate. It is important to note that aceto-acetic acid becomes decomposed in the process of distillation with the formation of acetone, and that when this acid is present a considerably larger amount of acetone may be present in the distillate than in the original urine.

The test most usually employed is that of Lieben, which consists in the addition to several c.c. of the urine or distillate of a few drops of liquor potassæ and of a solution of iodine with iodide of potassium. If acetone be present a yellow crystalline precipitate of iodoform is produced, which is seen under the microscope to consist of yellow six-sided tables or stellate crystals. This test is a very delicate one, but it is also yielded, although less readily, by alcohol.

Legal's test depends upon the fact that acetone colours a freshly prepared solution of sodium nitro-prusside ruby-red in the presence of sodium-hydroxide, the colour quickly changing to yellow. On the addition of excess of acetic acid a carmine tint appears which, in the course of time, changes to violet and finally to blue.

Aceto-acetic acid, or di-acetic acid, $CH_3.CO.CH_2.COOH$, is not a normal urinary constituent, but it is met with in various conditions in which an increased excretion of acetone is apt to occur. Thus it appears when healthy persons are put upon an exclusively meat diet, and in fasting. It is also met with in severe cases of febrile disorders of various kinds, especially in children. Its chief clinical importance is in connection with grave cases of diabetes.

The presence of aceto-acetic acid may be detected by means of the well-known iron reaction of diabetic urine, viz. the appearance of a deep red-brown colour when ferric chloride solution is allowed to fall into the urine drop by drop. If the reaction is obscured by a precipitate of phosphates this may be filtered off, and more ferric chloride may be added to the filtrate, which will acquire a claret-red colour.

Aceto-acetic acid is decomposed when the urine is kept or boiled, and is also decomposed by distillation with the formation of acetone.

β Oxybutyric acid, $CH_3.CH(OH).CH_2.COOH$, is met with in large amounts in the urine in some severe cases of diabetes mellitus, and its production is favoured by a change from a mixed to a strictly carbohydrate-free diet. In some cases the quantities found in the urine have been very large, amounting in two cases examined by Külz to as much as 100 and 223 grammes in the twenty-four hours. More often such smaller quantities as 15-20 grammes are met with.

It is to the formation of this acid that the greatly increased excretion of ammonia by some diabetic patients is ascribed, and it is believed to play the chief part in the causation of diabetic coma. When it is present in the urine aceto-acetic acid is also found.

In addition to cases of diabetes, β oxybutyric acid has been found in smaller amounts in the urine of patients suffering from scarlatina and measles, and by Klemperer in some cases of coma associated with malignant growths.

There is no easy clinical test for the presence of β oxybutyric acid, but its presence in diabetic urine is rendered highly probable when, after destruction of the contained sugar by fermentation, the urine exerts a marked lævorotatory action upon the polarised ray.

X. Aromatic Substances in Urine

Ethereal Sulphates.—A number of aromatic substances appear in the urine in combination with sulphuric acid in the form of ethereal sulphates. Among these may be mentioned phenol and kresol, pyrocatechin and hydroquinone, which last appears in cases of carbolic-acid poisoning, and it is the presence of hydroquinone which gives to carboluric urine the property of darkening when it is exposed to the air. Indoxyl and skatoxyl are also excreted as ethereal sulphates.

The amount of the ethereal sulphates may be increased by the administration of aromatic substances, and it is the fact that in carbolic-acid poisoning the phenol present in the urine is combined with sulphuric acid that prevents the obtaining of the colour reaction of phenol with ferric chloride.

The above phenol derivatives are also produced within the body by the bacterial decomposition of albuminous substances in the intestine and elsewhere, and hence the amount of ethereal sulphates excreted affords a valuable indication of the extent of the decomposition processes at work.

An increase of aromatic sulphates is natur-

ally associated with a diminution of the simple sulphates, and accordingly marked fluctuations are observed in the ratio between them, which under normal conditions is roughly 1 : 10, but which in consequence of excessive intestinal decomposition may be greatly reduced. The actual amounts of simple and ethereal sulphates excreted affords an even more valuable criterion, but it must be remembered that even in health both the relative and actual quantities are subject to somewhat wide variation.

As might be expected, the quantity of the aromatic sulphates is markedly diminished by a diet poor in proteids, and also after the administration of certain drugs which act as intestinal antiseptics, and diminish the activity of the intestinal bacteria.

In estimating the quantities of simple and ethereal sulphates respectively, advantage is taken of the fact that the latter are not precipitated by barium chloride, but that after they have been split up by boiling with hydrochloric acid their sulphuric acid is so precipitated. It is best to take two measured specimens from the urine of twenty-four hours and to heat one with hydrochloric acid, and afterwards to precipitate the sulphuric acid from both specimens by means of barium. In order to prevent the precipitation of phosphate the untreated urine should be acidified with acetic acid before the addition of the barium salt. The precipitate is collected, washed, dried, and combusted, and in this way an estimate is obtained of the total sulphuric acid in one specimen, and of the simple sulphuric acid in the other. By subtraction of the latter from the former the amount of sulphuric acid in combination with aromatic substances is obtained.

Conjugated Glycuronates. — Aromatic substances, as well as others, such as chloral and morphine, are also excreted as conjugated glycuronates. Glycuronic acid is closely related to glucose, from which it is formed by the oxidation of its alcoholic group, the aldehyde group remaining intact. The acid itself is dextrorotatory, but its conjugated derivatives rotate the polarised ray to the left, and reduce Fehling's solution with varying degrees of intensity. The appearance of more than traces of glycuronic acid in urine is usually associated with the administration of substances which form with it conjugated glycuronates, but some indoxyl is also excreted in such a form. It is by many regarded as an intermediate product of carbohydrate metabolism. Its presence may be suspected in urines which reduce Fehling's solution, are lævorotatory, and do not ferment, and it may be detected by means of Bial's orcin test (for pentose), provided that the mixture of reagent and urine is boiled for a short time.

Indicanuria.—The parent substance of the indoxyl compounds in urine is indol, which is a product of the bacterial putrefaction of albuminous substances. Thus indol is formed in the human intestine in small quantities even in health, and the quantity is larger on a diet rich in proteids. The indol absorbed from the intestine is oxidised to indoxyl in the tissues, and is excreted in the urine for the most part in conjugation with sulphuric acid as indoxyl-sulphate of potassium, and also in part as indoxyl-glycuronic acid. The so-called "indican" of urine is an indoxyl-sulphate, and must be distinguished from the true indican, which is a glucoside met with in indigo-yielding plants.

Urines rich in indican often have a dark brown colour which is not due to the indoxyl-sulphate, which is colourless, but, according to Baumann and Brieger, to higher oxidation products of indol. This cause of a dark brown colour must be borne in mind when one is called upon to investigate urine possessing such a tint, and is not so familiar as it should be.

The detection of indican by several methods is easy. Thus a specimen of urine may be boiled with an equal quantity of hydrochloric acid and a few drops of a solution of bleaching powder (Jaffe's test), when indigo-blue and its isomer indigo-red are formed. The liquid may then be shaken with chloroform, as suggested by Stokvis, and as a further evidence, the spectrum of the blue or purple chloroform extract may be examined by the spectroscope as recommended by MacMunn, when the band in red of indigo-blue is seen and a second band near the D line.

A drop or two of nitric acid may be substituted for the bleaching powder (Weber's test), but excess must be carefully avoided lest the indigo formed should be further oxidised to isatin.

Bile-pigments if present should be precipitated by basic lead acetate. When the urine contains iodides iodine will be set free which gives a purple colour to chloroform, and which may also interfere with the test by oxidising the indigo pigments.

It should also be mentioned that after the administration of thymol the urine gives, under such treatment, a bluish-green colour reaction.

The residue obtained on evaporating the chloroform extract may be treated with alcohol which dissolves indigo-red but not indigo-blue. The washed indigo-blue may then be redissolved in chloroform, and yields a beautiful blue solution.

Various methods for the estimation of indican have been devised, but these do not call for description in this place.

The precipitation of amorphous or crystalline indigo pigments from urine, and the occasional formation of indigo calculi, will be referred to later.

The clinical significance of the presence of large amounts of indigo in urine is due to the

evidence which it affords of excessive proteid decomposition under the influence of bacteria, either in the intestinal canal or in some of the body cavities. The most frequent cause is excessive albuminous decomposition in the alimentary canal, which is the seat of origin of the indoxyl-sulphuric acid present in normal urine. A quantitative estimate of such changes is best obtained by the determination of the relation between the aromatic and ordinary sulphates of the urine.

Thus an increased output of indigo is often observed in cases of intestinal obstruction and in not a few intestinal disorders. Simon lays much stress upon a diminished secretion of hydrochloric acid by the stomach as a cause of indicanuria. Thus it occurs in many cases of carcinoma of that organ, and also in connection with gastritis.

Examples of excessive formation of indol elsewhere than in the alimentary tract are afforded by cases of putrid empyema, fœtid bronchitis, and pulmonary gangrene, and von Jaksch attaches much importance to such varieties of indicanuria. He also includes peritonitis in this group, but in this condition the associated paralysis of the intestinal walls may also contribute to the result.

The precipitation of free indigo from the urine has no special clinical significance, but a large precipitation is indicative of an excessive output of the indigo-yielding materials, especially of indoxyl-glycuronic acid. The separation is usually determined by putrefactive changes in the urine itself.

Skatoxyl compounds also yield red colouring matters under the influence of acids with an oxidising agent, and it is probable that such skatoxyl derivatives are often produced together with the indigo pigments when the urine is subjected to Jaffe's test.

Alkaptonuria is the name assigned by Boedeker to a condition of great interest, and at the same time of extreme rarity. The urine, which has a natural appearance when passed, darkens on exposure to air, passing through various shades of brown to black. This change is much more rapid when the urine is rendered alkaline. The urine also stains linen or woollen fabrics deeply, and readily reduces Fehling's solution with the aid of heat. However, it does not give the bismuth test for sugar, and the reducing substance contained in it does not ferment with yeast and does not rotate the polarised ray in either direction.

By these means, and by the rapid darkening with alkalies, the condition is recognised, and its nature may be proved by the isolation from the urine of homogentisic acid in the form of the lead salt.

Alkaptonuria is in most instances congenital, life-long, and harmless, but has been temporary, or has developed some time after birth, in a few cases. Of the congenital cases a considerable number have occurred in brothers and sisters of whom a large proportion have been the children of first cousins. It has been much more frequently met with in males than in females.

The anomaly usually comes under notice on account of the manner in which the napkins of infants are stained by their urine, or in adult life on account of the refusal of the patients by insurance offices because of the reducing power of their urine.

In some of the earlier cases the peculiarity was attributed to pyrocatechin or to protocatechuic acid, but there is every reason to believe that homogentisic acid (hydroquinone-acetic acid, $C_6H_3(OH)_2.CH_2COOH$) is the abnormal constituent in all true alkapton urines. Since the discovery of this acid by Wolkow and Baumann it has been found in every case examined. A second allied acid, uroleucic, which was detected in association with homogentisic acid in the urine of two brothers studied by Kirk, has not since been met with.

As Wolkow and Baumann first showed, the output of homogentisic acid in such cases is conspicuously increased by a diet rich in proteid, and still more by the administration of tyrosin by the mouth, and there is little doubt that tyrosin, formed in the intestine, is its parent substance. On the other hand, when tyrosin is given to normal individuals no alkaptonuria results.

The evidence available suggests that alkaptonuria is a "sport" of metabolism, a chemical analogue of structural malformations, but wherein the metabolism of alkaptonuric persons differs from that of normal individuals, and why they excrete intact the benzene ring of their tyrosin, which, under ordinary circumstances, is broken up in the tissues, is not yet apparent.

The problem is rendered more difficult by the fact that the change from tyrosin to homogentisic acid involves chemical changes of a quite unfamiliar kind. Wolkow and Baumann suggested that this change might be brought about by a special micro-organism in the intestine, but further researches have not lent support to such an infective theory, which can hardly be reconciled with the known facts.

The recognition of alkaptonuria as a harmless condition is of some importance from a clinical point of view and in connection with life insurance, but there can be no doubt that its rarity is real and not apparent. The total of cases recorded up to the present time is only forty, and of these no less than eighteen have occurred in seven families.

XI. The Neutral Sulphur of Urine, Hydrothionuria, Cystinuria

Some 15 per cent of the total sulphur in human urine has escaped oxidation to sulphuric acid, and for this fraction Salkowsky suggested

the name of neutral sulphur. The forms of combination in which the neutral sulphur exists is not fully known, but, as Lépine has shown, some of it is much more readily oxidisable than the rest. Some is certainly in the form of sulphocyanides and some of cystein, which, as Baumann has shown, is present even in normal urine. Some is almost certainly derived from the taurin of bile, for in cases of obstruction of the bile-ducts the total neutral sulphur is increased, whereas in dogs with biliary fistulæ it is diminished. In the rare condition known as cystinuria the excretion of neutral sulphur is naturally above the normal.

Hydrothionuria.—The occurrence of sulphuretted hydrogen in the urine is usually due to bacterial decomposition, either within or without the body, and the amount produced by such decomposition will naturally be greater when the neutral sulphur is increased, as in cases of cystinuria. More than one species of bacterion appear to have the power of bringing about such decomposition in urine. Another cause of hydrothionuria is the existence of a fistulous communication between the colon and the urinary apparatus, and according to Betz the gas may find its way by transfusion without the existence of any fistulous opening.

For the detection of sulphuretted hydrogen the urine is placed in a flask, and a paper moistened with a solution of lead acetate and a drop of sodium hydrate solution is held in the neck of the flask by means of the cork. If sulphuretted hydrogen is being evolved the paper becomes blackened. Considerable quantities of the gas may be recognised by its characteristic odour.

Cystinuria.—It has already been mentioned that a substance closely allied to cystin, viz. cystein, is present in traces in normal urine; but in an extremely rare condition, of which, however, more than a hundred cases are on record, considerable quantities of cystin are passed in the urine, in which it may form a sediment of colourless hexagonal crystals, or may constitute calculi of greater or less size.

Cystin $\left\{\begin{array}{l} CH_2 \\ | \\ C\begin{array}{l}NH_2\\ S-\end{array} \\ | \\ COOH \end{array}\right\}_2$ is in all probability an intermediate product of proteid metabolism, which, under normal conditions, undergoes further oxidation, its sulphur appearing in the urine in the form of sulphates. In cystinuria the neutral sulphur is increased both in absolute amount and relatively to the sulphates. Quite recently it has been shown by Mörner and Ebden that cystin may be readily obtained by the hydrolysis of proteid substances.

Cystinuria, although an anomaly of peculiar pathological interest, acquires its only clinical importance from the tendency of the cystin precipitated from the urine to form calculi; but the urine may deposit crystals of cystin, at least over long periods, without any calculi being formed. On the other hand, small calculi may be passed or may be formed in rapid succession.

Of great interest is the occurrence, in association with cystinuria, of the diamines, putrescine and cadaverine, in the urine and fæces of the patients, a fact which led Baumann and Udranszky to suggest that as these are well-known bacterial products, cystinuria itself might be indirectly of bacterial origin.

Against this we have the great tendency for cystinuria to occur in several members of a family, and the fact that it may be inherited from either parent. Moreover, the early age at which cystin calculi have occurred in not a few instances suggests that it is probably a congenital anomaly. The analogies here presented with alkaptonuria, although the chemical errors in the two conditions are entirely different in character, strongly suggest that the two conditions have something in common, and that in cystinuria we have a second example of a chemical malformation.

XII. Lipuria and Chyluria. Cholesterin in Urine

Lipuria.—Fat may present various appearances when present in the urine. When abundant it may form oily drops which float upon the surface of the liquid, and which when sucked up by a piece of filter paper produce greasy marks upon it. In other cases it appears as a cloud consisting of small droplets suspended in the urine, as in some cases of fracture of bones; or again as an evenly diffused emulsion, which gives to the liquid a milky appearance, as in chyluria. Most frequently its presence is only revealed by the microscope in the form of minute highly-refractive globules, which are usually adherent to renal casts or enclosed in degenerated epithelial cells. Lastly, the very rare urinary calculi known as urostealiths contain a large proportion of fat.

When fat is present in minute amount in a sediment it may be recognised by staining with osmic acid, but larger quantities may be removed by shaking the urine with ether and submitting the residue left after evaporation of the ether to the ordinary tests for fats.

Cases of lipuria or apparent lipuria admit of classification into four main groups, viz.:—

(1) Those in which the fat has been accidentally or intentionally added; (2) those in which the blood contains an undue amount of fat which is excreted by the kidneys; (3) those in which the fat is derived from degenerative changes in the kidneys themselves, or in some lower portion of the urinary tract; and (4) cases of chyluria, which condition calls for special discussion.

It is of great importance to exclude accidental contamination caused by the use of an oiled catheter, or the addition of milk or oil to the urine after it has been passed. Cases in which the fat is abundantly present will only repay further investigation after the exclusion of such causes.

Normal human urine contains no fat, but it has been found under conditions which may fairly be regarded as physiological, as, for example, during pregnancy, and also after the administration of oils, such as castor-, cod-liver- or olive-oil, for medicinal purposes.

In *diabetes mellitus* the abundant presence of fat in the blood, which is a common phenomenon, may be accompanied by lipuria, and a like result may follow from a like cause in other diseases, attended by conspicuous wasting, such as *phthisis pulmonalis*. In acute yellow atrophy of the liver also, and in phosphorus-poisoning, fat may find its way into the urine.

Of special interest is the lipuria which sometimes follows fractures, especially of long bones, and which is attributed to the entrance of medullary fat into the circulation. It may be met with after the operation of osteotomy, as well as after accidental fractures, and such lipuria is temporary and may be intermittent.

In chronic parenchymatous nephritis fattily degenerated epithelial cells and fatty casts are often found, and their presence indicates a marked degree of degeneration of the renal epithelium. Simple fatty degeneration of the kidneys may also be attended by fat in the urine, and among other diseases of the urinary tract which may give rise to lipuria pyelitis, abscesses rupturing into the urinary passages, and malignant growths may be mentioned.

Chyluria.—In chyluria the urine has the appearance of milk, but may have a pink tint from admixture of blood, or may deposit a scanty red sediment of blood corpuscles. The opacity is due to the presence of fat, which may be removed by shaking with ether, when the urine is left clear. The suspended fat is in the finest possible state of division, and the microscopic appearances are best imitated by adding condensed rather than fresh milk to the urine. Albumin is present as well as fat, albumoses have also been found in chylous urine, and in some cases sufficient fibrin is present to form coagula, or to cause the solidification of the whole into a mass like *blanc-mange*, which, after a time, again liquefies. Occasionally such coagulation occurs in the bladder with distressing results.

The microscope shows red corpuscles and leucocytes, but the absence of renal elements points to the absence of any serious implication of the kidneys. The essential cause of chyluria appears to be the direct opening of a lymphatic channel into some part of the urinary passages, an explanation which best accords with the very intermittent character of the phenomenon, for, in the course of a single day chylous urine may be passed at one time and normal at another.

In the great majority of instances the cause of chyluria is the *filaria sanguinis hominis*, the mechanism being the obstruction of a lymph channel by the parent worm or by the ova, the distension of the vessel behind the obstruction and its rupture into the urinary passages, with a resultant intermittent flow of chyle into the urine. However, there appear to be some cases which are definitely not of this nature, and in which the admixture of chyle is due to some other cause than the filaria.

Cholesterin in Urine.—This will be the most convenient place to consider the occurrence of cholesterin in the urine. Its presence indicates disease of the urinary apparatus, and there is no satisfactory evidence that it is excreted by the kidneys as a product of metabolism. The traces found in cases of fatty kidney may be attributed to the fatty casts and débris which the urine contains, and in which the cholesterin is probably held in solution in the fat.

In the rare cases in which it forms a crystalline urinary sediment, it is derived from degenerated urinary epithelium, or the rupture into the urinary passages of cysts containing cholesterin. Thus it may appear in cases of hydronephrosis, of congenital cystic disease of the kidneys, and in connection with chronic cystitis and fatty degeneration in the bladder walls.

Calculi containing cholesterin have been met with. In some instances they have obviously been faceted gall-stones, which have found their way along fistulous channels, but such an explanation will not apply in all instances (*see* section "Urinary Calculi," p. 304).

XIII. Pigments

The yellow colour of normal urine is entirely or almost entirely due to urochrome, but although there is good reason to believe that, apart from the effects of dilution and concentration, the excretion of this pigment is subject to marked variations, such variations do not as yet come within the scope of clinical medicine. This is largely due to the facts that urochrome yields no characteristic spectrum, and that we have not as yet any means of estimating its amount. It will here suffice to say that this pigment is in all probability derived from hæmoglobin, and that its formation is apparently independent of any processes which go on in the alimentary canal.

Urobilin, unlike urochrome, yields a very conspicuous absorption band which allows of its ready detection in urine, and it is not difficult to estimate, at least roughly, the amount which is present in any given specimen. It is largely excreted in the form of an unstable chromogen

which is readily converted, by oxidation, into the formed pigment.

Urines rich in urobilin, and in which the colour of this substance is not obscured by other pigments, exhibit a peculiarly rich orange tint, and when examined in a conical glass show a tinge of pink near the apex of the cone. When examined with the spectroscope they show a broad and intense absorption band near the F line, which becomes broader and darker as deeper layers of urine are examined. Urobilin is precipitated by saturating the urine with ammonium sulphate, and solutions of the pigment in alkalies yield a brilliant green fluorescence, and a shifting of the band towards the red, on the addition of zinc chloride. This is best seen when alcoholic solutions are employed. For clinical purposes the direct spectroscopic examination of thin layers of urine will usually suffice for its detection, but when the violet end of the spectrum is much obscured by other pigments it may be necessary to precipitate it in the manner referred to, or to extract it with amylic alcohol, after the addition of a small quantity of acetic acid to the urine. The amylic extract may show the bands of hæmatoporphyrin and of uroerythrin in addition to that of urobilin.

The evidence forthcoming points to the formation of urobilin in the alimentary canal by the action of micro-organisms upon the bile-pigment, for it can be shown that when bile ceases to enter the alimentary canal urobilin is no longer formed, and that it disappears from the urine as well as from the fæces, in which latter it is normally present in far larger amounts, mostly in the form of chromogen. There are, on the other hand, some observations recorded which make one hesitate to assert too positively that urobilin never has a more direct origin from the blood-pigment.

If the above view be correct, it is obvious that an increase of urobilin in the urine may be indirectly due to increased hæmolysis, the abnormal destruction of hæmoglobin being followed by an increased output of bilirubin, the material from which urobilin is formed. Variations in the excretion may also be due to increased activity of the bacterial processes in the intestine, to the rate of passage of the intestinal contents along the canal, and to such conditions as altered reaction of the contents, and the relative amounts of bilirubin and biliverdin in the bile.

Thus the conditions which determine an increased or diminished output of urobilin are complex, and often difficult to unravel, and it is easy to understand why, in spite of the large amount of research which has been expended upon this subject, the results afford so little that is of real clinical value. This much is certain, that when a urine shows a conspicuous urobilin band there is some distinct deviation from the normal. Nor is the occurrence of urobilinuria in connection with active hæmolysis open to doubt, as witness its occurrence in connection with pernicious anæmia, and with the absorption of large quantities of extravasated blood.

In diseases of the liver there is usually persistent urobilinuria, a fact which raises the chief difficulty in connection with the theory of its intestinal origin. It has been suggested that the diseased liver loses a power which it otherwise possesses of destroying a large proportion of the urobilin absorbed from the intestine, or, on the other hand, that the hepatic disease affects the quality of the bile and the readiness with which the contained pigment is converted into urobilin.

In many febrile conditions also there is a conspicuously increased excretion of urobilin and its chromogen.

When the common bile-duct is completely obstructed urobilin and its chromogen disappear from the urine, and the practically complete absence of this pigment from the fæces in such cases affords very valuable evidence of such occlusion. Thus in two cases of equally intense jaundice the fact that the duct is completely occluded in the one and not in the other may be readily ascertained by this means. However, a trace of urobilin usually persists in the fæces, which trace is probably formed from bilirubin which makes its way through the intestinal walls.

Diseases of the kidneys often lead to diminution or absence of urobilin from the urine, probably as the result of diminished permeability of these organs.

Uroerythrin.—The presence of this pigment is easily recognised when urate sediments are thrown down, and many of the palest of such sediments show the characteristic pink tint when filtered off. The most intensely pink sediments are met with in connection with disorders of the liver, such as cirrhosis, and in the passive congestion secondary to heart disease. Uroerythrin is also excreted in comparatively large quantities in certain febrile disorders such as acute rheumatism and pneumonia, and also in gout. In typhoid fever, on the other hand, this is not usually the case.

If, as seems highly probable, the excretion of uroerythrin is always indicative of hepatic disorder, it is clear that not only actual disease of the liver but even very slight functional disturbances, such as may result from indiscretions of diet, or may attend upon attacks of dyspepsia, are sufficient to cause it to appear even in the urine of apparently healthy persons. It may be mentioned that in cases of cirrhosis Riva has observed a marked diminution in the output of uroerythrin when the patients were put upon a milk diet.

The pink colour of urate sediments when present, and the dark absorption band near the

D line which such sediments show when examined with the spectroscope by reflected light, suffice for the recognition of its presence. When in solution in large amount it imparts to the urine a fiery orange colour, and on the addition of caustic potash the colour becomes much paler and shows a greenish tint. From such urine, slightly acidified with acetic acid, the pigment is readily taken up by amylic alcohol, to which it gives a similar colour, and before the spectroscope the feeble complex band in the green, characteristic of this pigment, may usually be recognised, together with the absorption bands of hæmatoporphyrin and of urobilin. Indeed this method, suggested by Riva and Zoja, affords the readiest means of detecting the presence of all three pigments when they are present in more than minute traces.

Hæmatoporphyrinuria forms the subject of a special article, and it is here only necessary to repeat that hæmatoporphyrin occasionally appears in greatly increased quantity, together with other little known abnormal pigments, in the urine of patients who have been treated with sulphonal or allied drugs, and much more rarely apart from this cause. Such urines have a deep port-wine colour.

In morbid urines there is often much more than the normal trace of this pigment, but not enough to appreciably affect the colour. Such increase can in most instances be traced to disorders of the liver, functional or organic, and even in rheumatic fever it is not improbably due to a secondary affection of that organ.

The evidence forthcoming all tends to show that an increased excretion of hæmatoporphyrin affords no evidence of excessive hæmolysis, but rather of a perverted metabolism of the blood pigment. Hæmatoporphyrin is also found in traces in normal bile and fæces and in larger amount in the meconium of new-born infants.

Melanuria.—Patients who are the subjects of melanotic growths occasionally pass urine which is of natural colour when freshly passed, but which soon darkens on exposure to air and in time becomes quite black. More rarely the urine is very dark in colour even when passed. This condition is readily distinguished from others in which the urine turns brown or black by certain characteristic tests which are of easy clinical application. Thus the blackness is promptly developed when nitric acid is added to the urine, and an intense blackening is also observed on the addition of ferric chloride. Lastly, bromine water yields a copious precipitate which quickly takes a chocolate hue, but this reaction is less delicate than those above mentioned, and may fail when the melanogen is scanty in amount.

Such urines also usually yield Thörmalen's reaction, namely, a deep blue colour, with sodium nitroprusside, potassium hydrate, and acetic acid, but this is apparently due to some other constituent than the melanogen present. The chromogen, melanogen, has not yet been isolated, but the formed pigment, which has been studied by Mörner, appears to be of the same nature as that of melanotic growths.

It is often stated that melanuria is not confined to cases of melanotic growths, but may also occur in connection with other forms of malignant tumour, and in wasting diseases. When, however, the accounts of the cases on which this statement is based are referred to, the evidence of true melanuria is found to be by no means conclusive, and some of them at least appear to have been examples of the very dark brown urine which accompanies a large excretion of indican. There is, indeed, good reason to believe that true melanuria is only met with in cases of melanotic sarcoma, and even in such cases it is not always present. I have never met with it in cases in which the growth was still limited to its primary seat, but only when the liver and other organs have become secondarily involved. Even in such cases the amount of melanogen present may be small, and the darkening of the urine may fail to attract attention.

The diagnostic value of this symptom is considerably impaired by the fact that in most cases before melanuria develops the patient is well known to be suffering from a melanotic growth.

XIV. Bile-Pigment and Acids

The bile-pigment may find its way into the urine in two distinct ways. In almost every instance its presence is one of the phenomena of jaundice, and is associated with coloration of the skin and conjunctivæ. Very rarely it results from the existence of a fistulous opening connecting the biliary and urinary passages, and in some such cases the presence of bile in the urine has been associated with the passage of gall-stones per urethram. Seeing that such choluria is as a rule unattended by jaundice, it affords valuable evidence of the existence of a fistulous communication.

The colour of the urine suggests the presence of bile-pigment, which in freshly-passed urine usually has the form of bilirubin, but biliverdin and other derivations are apt to be formed when the urine stands in contact with air.

Of the tests employed for the detection of bile-pigment three only need be mentioned here. Maréchal's test is carried out by allowing a dilute tincture of iodine to run on to the surface of the urine in a test-tube. When bilirubin is present a green ring appears at the junction of the liquids owing to its oxidation to biliverdin.

Gmelin's test is best performed by allowing the urine to flow on to the surface of some nitric acid in a test-tube. A coloured ring forms at the junction, and when the tube is gently shaken, so as to bring about some admixture of the liquids, the characteristic play of colours is

observed. Seeing that the chromogens of urine tend to yield pink and purple colours with nitric acid, it is necessary that the green tint of biliverdin should be clearly seen, in order that the presence of bile-pigment may be rendered certain.

When the quantity of bilirubin present is scanty, and especially when the indoxyl compounds are at the same time abundant, Gmelin's test may yield equivocal results. In such cases, Huppert's test, although not quite so simple, affords an excellent means of detecting bile-pigment. The pigment is precipitated by the addition of milk of lime, and the filtered precipitate, which will be yellow if bile be present, is treated with alcohol to which a few drops of dilute sulphuric acid have been added. On heating a rich green colour is obtained.

The biliary acids, like the pigments, are met with in icteric urines, but their detection hardly comes within the scope of clinical medicine, and, seeing that the more recent investigations have tended to abolish the distinction formerly drawn between what was regarded as hepatogenous and hæmatogenous jaundice, their presence in varying amounts has lost much of the diagnostic significance formerly attached to it. Their presence in traces in normal urine has been asserted and also denied.

It is here only necessary to emphasise the fact that Pettenkofer's test is not directly applicable to urine, as other constituents of that liquid yield colour reactions not to be definitely distinguished from that yielded by the bile acids. Hence their satisfactory detection becomes a matter of difficulty and involves their isolation.

Cholesterin is not met with in the urine as a biliary constituent in cases of jaundice; when present it is derived from the kidneys or urinary passages, except in the very rare instances in which gall-stones enter the urinary passages by a fistulous opening.

XV. Pneumaturia

This is the name given to a rare condition in which free undissolved gas is present in the bladder and is passed in bubbles with the urine, occasionally with an audible sound.

Cases of pneumaturia fall into three classes, viz.:—

1. Those in which air has been introduced from without, either in catheterisation or for the purpose of distending the bladder. This variety is clearly of no clinical importance.

2. In some cases a fistulous opening exists between the bowel and the urinary bladder, most commonly in association with malignant growths of the rectum or sigmoid flexure. In such cases the escaping gas has a fæcal odour, and fæcal particles may at times be found mixed with the urine.

3. In the cases of the third class the gas is formed in the bladder itself as a result of bacterial processes. Such formation of free gas is usually met with in cases of diabetes in which the bladder has become infected by one of several kinds of bacteria or yeasts, and the sugar undergoes fermentation with the development of carbon dioxide (*see* "Bacteriology of Urine").

As the absorption of gases from the bladder takes place somewhat rapidly, a considerable development of gas is necessary in order that the symptom of pneumaturia may be produced.

XVI. Urinary Sediments

The microscopical and chemical investigation of sediments constitutes an important part of the clinical examination of urine. When the sediments are abundant they may be taken up by means of a pipette from the bottom of the urine as it stands in a conical glass, or may be drawn off from a burette in which some of the urine has been placed. When, on the other hand, it is necessary to institute a careful search for structures or casts which may only be present in very small numbers, it is necessary to centrifuge a portion drawn from the lower layers of the liquid after it has stood for some time.

Urinary sediments may be grouped in three main classes:—

1. Unorganised sediments consisting of inorganic salts or organic compounds which have been deposited either in an amorphous or crystalline state.

2. Organised sediments, including cells derived from the blood, from the kidneys, or from the walls of the urogenital tract, and with these it is convenient to include the important structures known as casts, which frequently enclose cellular structures of various kinds.

3. Accidental contaminations which are apt to find their way into the urine, and which, although themselves of no clinical importance, require to be recognised lest they should be mistaken for true urinary deposits.

1. Unorganised Sediments.—Of the sediments belonging to the first of these groups some are extremely common, and are met with every day, others are seldom seen, and others again are so extremely rare that from a practical point of view they amount to little more than clinical curiosities. Of most of the sediments in question it will only be necessary to give a brief description in this place, seeing that their significance has been dealt with in the more chemical portions of the present article.

Amorphous Urates.—These are the commonest of all the sediments met with in urine. They consist of a mixture of uric acid salts, including those of sodium, potassium, calcium, and magnesium, and especially those of the two first-named metals. It has been usually held that they consist of the so-called quadriurates,

formed by the combination of a molecule of biurate with a molecule of free uric acid, and the ease with which their aqueous solutions throw down crystals of free acid is ascribed to the disintegration of the compound molecule. However, much doubt has recently been thrown upon the accepted views on this matter. Under the microscope the sediment is seen to consist of minute amorphous granules mostly grouped into threads and clusters.

Such sediments are deposited from acid urines on cooling, and their nature is easily recognised by the readiness with which they dissolve when the urine is again warmed. Their colour, which varies from a pale yellow, through brick-red, to a brilliant pink, is due to urinary pigments which they carry down with them. The chief of these are urochrome, which is always present in them, and which imparts a yellow tint even to the palest sediments, and uroerythrin, which has a special affinity for uric acid and the urates, and which probably enters into some sort of combination with them, for the colour of the pink sediments, and their spectrum as seen by reflected light, differs from those of the isolated pigment.

The frequency of the pink sediments is due to the fact that the same morbid conditions are wont to determine the formation of uratic deposits and the excretion of large quantities of uroerythrin.

As might be expected, urate sediments are commoner in winter than in summer, and they are met with in the urine of healthy persons after active muscular exertion or after slight indiscretions of diet. The chief factors which determine their occurrence are concentration of the particular specimen of urine and the degree of its acidity. They do not necessarily afford an indication of excessive output of uric acid, although such increase may contribute to their formation. They are apt to occur in the course of most febrile disorders, and deep pink sediments are specially frequent in connection with organic and functional disorders of the liver, as also in connection with gouty troubles, and in cases of acute rheumatism.

The disappearance of such sediments when the urine is warmed, and their coloration, usually leave no doubt as to their nature, but if further confirmation is needed this may be readily obtained by filtering off the sediment and applying the murexide test, or by dissolving it in water and observing the separation of crystals of uric acid after the addition of a small quantity of hydrochloric acid.

Ammonium biurate is occasionally deposited from ammoniacal alkaline urines in the form of minute yellow or brownish spherules which are beset with needle-like excrescences, the so-called hedgehog crystals. If the crystals are dissolved in an acid, uric acid crystals will be deposited from the solution. This deposit is usually met with in association with sediments of ammonio-magnesium and other phosphates.

Uric Acid.—Crystals of free uric acid form a very common urinary sediment. They may form an abundant deposit of deep red "cayenne pepper" sand, or may be scattered as golden crystalline points through a cloud of mucus at the bottom of the urine glass. The crystals vary very greatly in size and in form, but are usually readily recognised by their colours. These colours are due to included urinary pigments, especially urochrome and uroerythrin, but other pigments share in their coloration. When deposited from urine containing bile they are tinted by bilirubin or biliverdin or a mixture of the two, and those deposited from carboluric urine are deep brown in hue and appear black in bulk. Urobilin takes no part in the coloration of these crystals.

In scantily pigmented urines, and especially in that of young children, they may be almost colourless, or actually devoid of colour. The form of the crystals is also profoundly modified by the included colloids, and they always deviate somewhat widely from the form of the rectangular plates of pure uric acid. The predominant shape of the urinary crystals is the canoe or whetstone form, and many of the less usual forms are produced by the fusion of such crystals. Sheaves and rosettes of prismatic crystals are occasionally seen, especially in urines containing biliverdin, and hour-glass and dumb-bell crystals are also described. A double-comb form may also be referred to, which is apt to occur when the precipitation has been brought about by the addition of an acid to the urine, and it should be borne in mind that very faintly tinted hexagonal crystals of uric acid have occasionally been mistaken for those of cystin.

Any urine, if kept acid, will, in course of time, deposit crystals of uric acid, and although an excessive excretion of the acid is one of the factors which favour the formation of such sediments, they are apt to occur apart from this cause in any concentrated and highly acid urine. In early childhood they are very common, and in the urine of infants mats formed by the interlacing of innumerable minute whetstone crystals are often seen, sometimes in association with hæmaturia.

As a rule, uric acid sediments have no great clinical significance, but the frequent appearance of cayenne pepper sand is a common accompaniment of calculous formation.

When the nature of the crystalline deposit is in doubt, owing to scanty pigmentation or for other reasons, the murexide test affords conclusive evidence.

Calcium Oxalate. — Crystalline deposits of calcium oxalate are of frequent occurrence. They are met with both in acid and alkaline urines, especially in the former. The crystals,

which are minute, may be deposited upon grease streaks on the sides of the containing vessels, or may form a snow-white cloud upon the surface of a collection of mucus at the bottom of the urine glass. The crystals, which are highly refractive, are as a rule colourless, none of the ordinary urinary pigments being included by them, but when deposited from icteric urines they acquire a feeble yellow or green tint.

The crystals assume several different shapes. The most usual and characteristic of these is an octohedron with a short principal axis, which under the microscope has the appearance of a square envelope, whence they are usually spoken of as envelope crystals. Twin octohedra and other more complex interpenetrating forms are also met with.

Less commonly short, square prisms with pyramidal ends are seen, and in some specimens crystals of this type preponderate.

The so-called dumb-bell crystals are more or less flattened oval plates with deep grooves upon their oval surfaces which render them biconcave. Such crystals sometimes exhibit faint striation, and when seen edge on present the appearance of dumb-bells. They are usually mixed with the ordinary octohedral forms, and in some specimens they are exceedingly abundant.

Cystin.—Sediments of cystin are extremely rarely encountered in acid or faintly alkaline urines, but when present they usually form abundant greyish-white deposits. Cystinuric patients often suffer from cystitis. The crystals of which the sediments are composed are colourless hexagonal plates, the sides of which are often of unequal length. Superposed hexagons are also met with. They can hardly be mistaken for any other urinary crystals, with the exception of the pale hexagons of uric acid already referred to.

The crystals are insoluble in water, alcohol, and ether, but readily dissolve in ammonia, from which they are redeposited as the ammonia evaporates. The following beautiful test serves for the recognition of cystin. Some of the sediment is dried and placed upon a glass slide, some strong hydrochloric acid is allowed to run under the cover-glass, and, as this comes in contact with the deposit, rapidly growing prismatic crystals grouped in rosettes replace the original hexagonal plates, and these crystals, which consist of a compound of cystin with hydrochloric acid, melt away as rapidly as they were formed when the acid is diluted by running in some water.

Xanthin.—A crystalline sediment of xanthin was met with in one single instance in the urine of a child who had previously suffered from symptoms of renal colic. The sediments consisted of small whetstone-shaped crystals which dissolved readily when the urine was heated. The sediment was soluble in water and in hydrochloric acid, and from solution in the latter was deposited in six-sided tables and prisms. It also dissolved in nitric acid without effervescence, and on evaporating the acid a yellow stain was left. It is possible that such crystals have sometimes been mistaken for colourless or faintly tinted crystals of uric acid.

Phosphatic Sediments.—When any urine is rendered alkaline with a fixed alkali, the earthy phosphates and the normal or mono-hydrogen phosphates of calcium and magnesium are precipitated, and a turbidity due to the same substances is observed in urine which is alkaline when passed. The sediments from such urines are colourless and amorphous, and their nature is easily recognised by the ease with which they dissolve on the addition of acetic acid.

In the urines which are the seat of putrefactive changes, and owe their alkaline reaction to ammonia, there are found, in addition to amorphous phosphates, abundant crystals of ammonio-magnesium phosphate (triple-phosphate), $MgNH_4PO_4.6H_2O$. These crystals, which are amongst the largest met with in urinary sediments, are rhombic prisms, of the so-called coffin-lid form. Similar crystals are also deposited from amphoteric urines apart from any amorphous sediment.

Acid calcium phosphate, $CaHPO_4.2H_2O$, is also thrown down in crystalline form from some amphoteric or faintly acid urines. These crystals, which are usually spoken of as "stellar phosphates," are grouped into stars or rosettes, or appear as isolated crystals, or again in groups of two or three. The individual crystals are long narrow prisms.

Normal magnesium phosphate, $Mg_3(PO_4)_2.22H_2O$, has occasionally been found as a crystalline sediment in the urine of patients with gastric dilatation to whom magnesium carbonate has been freely administered, in the form of oblong, rhombic, highly refractive tables, and Bradshaw has under similar circumstances met with an abundant deposit of long needles which he identified as consisting of acid magnesium phosphate, $MgHPO_4.14H_2O$.

Like amorphous phosphatic sediments, the above crystalline deposits may be readily distinguished from other crystals deposited from urine by the readiness with which they dissolve in acetic acid.

Calcium sulphate has been hitherto found as a crystalline sediment in only three cases: in two instances in urines strongly acid in reaction, in a third in association with triple phosphate and calcium carbonate. Such deposits are insoluble in acids and in ammonia alike. The crystals have the forms of long needles, or of long tables with sloping ends.

Calcium carbonate is seldom deposited from human urine, either in amorphous or crystalline form. It may be met with in association with amorphous phosphates. Occasionally it appears as dumb-bell crystals, which may be mistaken

for those of calcium oxalate, or in spherules which show a concentric striation. They may be recognised by the fact that they dissolve in acetic acid with the evolution of carbon dioxide.

Tyrosin and leucin are occasionally met with as sediments in the urine of patients suffering from acute yellow atrophy of the liver or phosphorus-poisoning. Tyrosin appears as fine needles arranged in sheaves or rosettes, which may be either colourless or pigmented. Leucin, which usually occurs in urine in association with tyrosin, is rarely if ever deposited from the urine direct, but on evaporation it sometimes appears in the form of greenish yellow spheroids which exhibit radial and concentric striation.

Hippuric acid has very seldom been seen as a sediment, consisting of rhombic prisms. Its appearance has usually followed the administration of benzoic acid or the eating of certain fruits.

Indigo.—Urines which are undergoing alkaline fermentation frequently deposit indigo-red and blue, either in the form of amorphous particles or as acicular crystals arranged in rosettes. Such urines often exhibit a blue scum upon the surface. The masses or crystals of indigo-blue have a dark blue colour, whereas those of indigo-red are of a violet-red hue. The free pigment is in such cases formed by the decomposition of indoxyl-glycuronic acid, the less abundant of the indoxyl compounds met with in urine.

Bilirubin is occasionally met with as a crystalline or amorphous deposit in jaundiced urine. The crystals are yellow or brown rhombic tables or needles arranged in tufts or rosettes, indistinguishable from those of *hæmatoidin*, which are sometimes met with in connection with diseases of the kidneys and urinary passages, and perhaps with special frequency in cases of malignant growths. It is highly probable that bilirubin and hæmatoidin are chemically identical.

Cholesterin, when present as a crystalline sediment, appears to be always derived from the kidneys or urinary passages, as in cases of hydronephrosis and chronic cystitis, or from cysts communicating with the passages. The crystals which may form an abundant deposit have the well-known characteristic form.

Soaps.—Von Jaksch has met with crystals which might easily be mistaken for those of tyrosin, but which are shown by their solubilities and reactions to be of some other nature. From their behaviour with solvents he is inclined to regard them as consisting of the calcium and magnesium soaps of higher fatty acids.

(2) Organised Sediments.—*Red blood corpuscles* are present in the urine in every case of hæmaturia. In many instances they have lost their biconcave form, and have been to a great extent deprived of their hæmoglobin. Not infrequently they have become crenate. Their presence is of great importance in distinguishing between hæmoglobinuria and hæmaturia, and affords evidence of morbid conditions either in or below the kidneys.

Leucocytes are very frequently present in small numbers in the urine under a variety of morbid conditions, and in females their occurrence in larger numbers may be due to admixture of vaginal discharges. When pus has become mixed with the urine the number of leucocytes is often very large indeed. Their appearance is to a great extent dependent upon the reaction of the liquid in which they are suspended, and in alkaline urines their recognition may present some difficulty from swelling and opacity of the cells.

The clinical significance of the presence of leucocytes is largely dependent upon their numbers, and upon the association with them of other cells or renal casts.

Spermatozoa may be found in the urine of men after seminal emissions, and they may be readily recognised by their long, thread-like processes and by the characteristic form of their heads. Except in very fresh specimens their mobility is lost. Some expression of semen may occur in cases of obstinate constipation as the result of pressure upon the *vesiculæ seminales*, and it is also found in urine passed after an epileptic fit.

Bacteria of various kinds may often be found in the sediments, but the question of the bacteriology of the urine will be considered in a separate article (p. 307).

Epithelial Cells.—The gradual shedding of the epithelium of the urinary tract, which occurs to some extent even under normal conditions, leads to the appearance of small numbers of epithelial cells in the urine of even quite healthy persons, and such cells form the organised constituents of the nubecula. In pathological states such cells may be present in much larger numbers and acquire considerable clinical significance. The epithelium lining the several portions of the urinary tract is composed of cells which differ somewhat widely in their forms and sizes, and it is possible for the trained eye to form a fairly definite notion of the source from which particular varieties of such cells met with in the urine are derived, but such recognition is apt to be rendered difficult by the changes produced by the liquid in which they are suspended, and especially by those due to the reaction of the urine.

It is usually an easy matter to recognise the groups of large flat polygonal cells, with sharply defined nuclei, which so frequently find their way into the urine of female patients and are of a vaginal origin, but it must not be forgotten that somewhat similar cells may be derived from the bladder, ureters, and urethra.

Again, the small round cells of the renal

tubules, with their sharply defined nuclei, are liable to be confused with leucocytes, and further evidence of their origin must be afforded by the simultaneous presence of renal casts.

Other forms which admit of recognition are the conical or tailed cells of the pelvis of the kidney, but it must not be forgotten that cells of a similar character are also found in the neck of the bladder and in the prostatic portion of the urethra.

Fragments of malignant growths are occasionally met with, especially when the growth is situated in the bladder and is of the villous variety. As a rule a diagnosis can only be made when a definite mass derived from the tumour is passed, as is not infrequently the case, but Délépine considers that the repeated appearance of cells of great size, with several nuclei or a single very large nucleus, should excite strong suspicion, especially if other symptoms of malignant growth, such as vesical pain and hæmaturia, are also present.

Casts.—The forms and sizes of the objects known as urinary casts, as well as the characters of the epithelial cells which frequently adhere to them or of which they may almost entirely be made up, indicate their origin in the tubules of the kidneys. If further proof is needed it is afforded by the fact that structures apparently identical are frequently seen occupying the tubules in microscopical sections of diseased kidneys.

The casts are of several different kinds which call for individual description, and it will be well to consider first those of the so-called hyaline variety.

Hyaline casts are extremely transparent objects, the refractive power of which differs so little from that of the urine in which they lie that their outlines are often ill-defined, and they may escape detection unless carefully looked for. In searching for them it is best to employ an oblique sub-stage illumination or a very small diaphragm, and in case of doubt staining agents, such as a solution of iodine with potassium iodide, may be added. Their chemical nature and mode of origin are still in doubt, as their reactions with chemical agents do not agree with those of any other known proteid material. This is true of fibrin, of which they were once supposed to consist, as well as of other proteids, but it is possible that the material of which they are formed has undergone some change under the influence of the urine. It has been suggested by some that they have their origin in a secretion of the renal epithelium, whereas others hold that they consist of coagulated albumin which has undergone some hyaline change. The latter view gains support from the fact that their presence is in the great majority of cases associated with that of albumin in the urine.

Hyaline casts are roughly cylindrical in form, of various lengths, and sometimes convoluted. Their ends may be rounded or as if broken across.

Adherent to the surfaces of the casts may be small epithelial cells, such as line the renal tubules, a few red blood corpuscles or leucocytes, or crystals of calcium oxalate or amorphous urates.

Such casts are met with in all forms of albuminuria which are associated with disorders, whether primary or secondary, of the kidneys. They have occasionally been found in small numbers in cases in which the urine is albumin-free, and are usually present in the urine of jaundiced patients. The presence of adherent renal cells or blood corpuscles adds to the importance attaching to their occurrence, indicating a grosser lesion of the kidneys. In acute nephritis such casts are often abundantly present, together with casts of other varieties.

Amyloid casts are usually broader than the hyaline, they have rounded or broken ends, and sometimes show signs of segmentation. Although homogeneous in structure they are much less transparent than hyaline casts and often have a yellowish tint. With iodine they acquire a brown colour and they are stained pink by methyl violet, but not infrequently these "amyloid" reactions are very imperfectly obtained. The presence of casts of this character affords no evidence of lardaceous disease of the kidneys; they are frequently met with in cases of chronic parenchymatous nephritis, very seldom in cases of granular kidney.

The most probable view of their origin is that they are derived from epithelial casts, next to be described, and that the cells of which they were composed have become converted into a homogeneous waxy material, under the influence of the urine, during a somewhat prolonged sojourn in the renal tubules.

Epithelial casts consist almost entirely of epithelial cells such as are met with in the renal tubules, and often present the appearance of a complete cast of the lining of the tubule. In all probability they usually contain a core of hyaline material, and this view receives support from the fact that one occasionally meets with examples of which one portion is covered with epithelial cells, whereas the remainder looks like a bare hyaline core. In the early stages of acute nephritis the individual epithelial cells are often very clearly defined, but in other instances they show signs of fusion which, when it reaches a more advanced degree, probably results in their conversion into casts of the amyloid variety. In chronic parenchymatous nephritis similar casts are met with, and as offering samples of the renal epithelium they afford valuable evidence of its condition, for the cells in the more advanced cases show signs of granular or fatty degeneration.

Blood casts, composed of red blood corpuscles,

are also commonly seen in the acute stage of parenchymatous nephritis. Their presence is of some importance, as affording evidence that in cases in which they occur the blood present in the urine is derived from the kidneys.

Leucocyte casts are much less common, although a few leucocytes are not rarely seen attached to hyaline casts.

Granular casts appear to be made up of finer or coarser granules, often with an admixture of small, highly refractive fat globules. Such casts, which usually have a yellow or brownish tint, probably consist of the products of degeneration of epithelial cells and of red blood corpuscles. They vary greatly in size, and may be as narrow as those of the hyaline, or as broad as those of the amyloid varieties.

Fatty casts are of various kinds, the fat globules being still included in epithelial cells, or free and adherent to a hyaline matrix or intermixed with granular débris. Von Jaksch describes the occurrence of fat crystals as well as of the usual highly-refractive globules. The presence of any considerable amount of fat in casts is of grave significance, indicating as it does an advanced stage of degeneration of the renal epithelium. Fatty casts are specially common in the later stages of severe cases of chronic parenchymatous nephritis. In case of doubt the presence of fat may be rendered certain by staining with osmic acid.

Hæmoglobin Casts.—In cases of hæmoglobinuria the granular débris contains numerous casts composed of dark brown granules, which have apparently assumed this form in the renal tubules.

Uratic Casts.—Casts composed of crystals of ammonium urate are often met with in the urine of young infants, but it must not be forgotten that the particles of which amorphous urate or ammonium urate sediments are composed are apt to assume a grouping which recalls the shape of renal casts.

Animal Parasites.—The most important of the animal parasites which are met with in urine are the ova of *Bilharzia hæmatobia*, as their presence is diagnostic of the nature of the hæmaturia by which they are accompanied. They are oval in shape and are characterised by the spine-like process which either occupies a terminal position or emerges from one side of the ovum. They are readily seen with a moderate microscopic power, their average dimensions being 0·12 by 0·04 mm. Within the shell of the ovum the ciliated embryo may often be clearly seen.

Hydatid hooklets and the remnants of hydatid cysts are occasionally met with in urine, their presence being almost always due to the rupture into the urinary tract of hydatid cysts situated in some adjacent part, but the occasional development of such cysts in the urinary passages themselves has been described. The hooklets are readily recognised by their characteristic shape, and occasionally small scolices are passed entire.

The *Filaria sanguinis hominis* has also been occasionally found in the urine.

Some parasites occur in the urine as mere accidental contaminations. Thus *Pediculus pubis* is occasionally met with, and threadworms may make their way into the urine after it leaves the bladder, or through a fistulous communication between the intestinal and urinary tracts. Certain infusoria, such as *Trichomonas vaginalis*, and acari derived from starch used as a dusting powder, have also been found.

Accidental Solid Contaminations.—Of these by far the most important is starch, which is so largely used in dusting powders, especially in hospitals, and which is very frequently mistaken by beginners for a crystalline sediment. The form of the granules, which may form an abundant sediment, differs according to the source from which the starch is derived, and they may be oval or polygonal in shape. They are highly refractive, and for the most part show a dark central star by which their nature is easily recognised. When any doubt remains, advantage may be taken of the deep blue colour imparted to them by iodine.

Fibres derived from clothing, either coloured or otherwise, are frequently seen, and it is possible to distinguish fibres of cotton, linen, wool, and silk.

Hairs also call for mention. They are usually pubic in their origin, but it should not be forgotten that hairs present in the urine may be derived from dermoid cysts communicating with the urinary passages.

XVII. Urinary Calculi

Of the substances which are apt to be deposited from the urine in the form of sediments not a few are liable to form actual concretions in the kidney itself, which may increase in size in the renal pelvis or in the bladder, and so give rise to the various symptoms and to the group of serious morbid changes which are apt to result from their presence.

The great majority of such calculi are composed of uric acid, calcium oxalate or earthy phosphates, and many of them are of mixed composition. Thus a nucleus composed of uric acid or calcium oxalate may, by its presence in the pelvis of the kidney or in the bladder, set up changes which result in the urine undergoing alkaline decomposition and the deposition of earthy phosphates upon the surface of the stone.

The conditions which determine the commencement of calculous formation are not yet fully known, but calculi contain a certain amount of organic material, which may perhaps play a not unimportant part in the formation of compact concretions instead of a mere discrete sediment.

When a nucleus is once formed further deposition upon it readily occurs, as is well seen in those cases in which a foreign body lying in a bladder in which ammoniacal decomposition is going on becomes encrusted with earthy phosphates. In the same way even a calculus of uniform composition usually bears evidence of the deposition upon it of successive layers of the ground material, and a nucleus of somewhat different structure from the rest may usually be distinguished.

Metamorphotic changes may also occur, as for example when a uric acid calculus lies for a long time in a bladder containing alkaline urine. Under such conditions some of the material of the calculus may undergo solution, and may be replaced by a deposit of earthy phosphates.

Uric acid calculi are the commonest of all. They vary in size from small spherules no larger than pin-heads, which are usually multiple and are probably formed in the renal tubules, to vesical stones as large as hens' eggs. The small particles of gravel are seen on section to have a radiating crystalline structure and the larger stones are composed of concentric layers which are apt to separate in flakes when the calculus is broken up.

Like crystalline sediments of uric acid such calculi are always coloured by included urinary pigments, and exhibit a yellow or reddish-brown tint. Their surface is usually smooth, but may be slightly granular. Calculi formed in the renal pelvis are apt to assume peculiar forms corresponding to that of the irregular cavity in which they lie, but uric acid stones from the bladder are usually oval in form and somewhat flattened.

On section distinct interposed layers of calcium oxalate are sometimes seen in stones of this character, and phosphatic stones are often found to have one or more small uric acid concretions as their nucleus.

The nature of uric acid calculi may be recognised by the fact that when burnt upon platinum foil they leave no considerable ash, showing the absence of fixed alkalies or alkaline earths, and when heated with caustic alkali they give off little or no ammonia. On the other hand, they yield the murexide test, *i.e.* when a portion of the material of the stone is heated with nitric acid upon a piece of white porcelain and the acid is evaporated to dryness a reddish colour appears which changes to purple on the addition of ammonia.

In the case of mixed stones a portion of one of the broken laminæ, believed to be composed of uric acid, may be subjected to the above tests.

Uratic Stones.—A secondary deposit of urates is not uncommon upon a nucleus of uric acid, and in young infants small urate calculi, soft and easily friable when dried, and tinted like those of uric acid, are not uncommonly met with. The nature of such layers or calculi may be recognised by the fact that they yield the murexide test, but also give off ammonia freely when heated with a caustic alkali.

Calcium Oxalate Calculi.—These are the hardest of all urinary stones, and, like those of uric acid, vary in size from a fine gravel to calculi of considerable dimensions. The smaller concretions may be smooth or may exhibit spinous processes, or again may have surfaces composed of almost colourless crystals of calcium oxalate. The most characteristic are the familiar mulberry calculi, with nodular surfaces and black or dark brown in colour.

On section they are seen to be composed of narrow concentric crystalline layers with intermediate deeply-pigmented zones, the included pigment being in all probability a derivative of hæmoglobin. There is usually a definite nucleus which is more coarsely crystalline. The structure of these stones has been minutely studied and described by Ord and Shattock.

Calcium oxalate dissolves in hydrochloric acid without effervescence. When moderately heated the oxalate becomes converted into carbonate, which effervesces with acids, and by heating in the blowpipe flame this becomes further decomposed, leaving a residue of calcium oxide.

Phosphatic calculi are usually formed around a nucleus of some other material, and occasionally around a foreign body, such as the broken end of a catheter. They are chiefly formed in the bladder, and consist, as a rule, of a mixture of the phosphates of calcium and magnesium, with ammonio-magnesium or triple phosphate. The last-named salt is formed as the result of ammoniacal decomposition of the urine.

Rarely small calculi composed of calcium phosphate alone are met with, as in some cases of disease, such as osteomalacia, in which the bones are largely deprived of their inorganic constituent. Such stones are usually small; large well-formed crystals may project from their surfaces, and on section they show a distinct crystalline structure.

Calculi composed of a mixture of calcium and ammonio-magnesium phosphates are known as fusible calculi, because they have the property of melting when heated over the blowpipe flame.

Phosphatic stones are colourless or only feebly tinted, are as a rule soft, and may attain to a large size. They dissolve in hydrochloric acid, without effervescence, both before and after incineration, and the solution yields the phosphoric acid reaction with ammonium molybdate.

The presence of triple phosphate may be detected by the free evolution of ammonia on heating with caustic potash or soda.

Calcium Carbonate.—Calculi composed wholly or in large part of calcium carbonate are not common in man, although not infrequently met with in vegetable-feeding animals. They require an alkaline medium rich in carbonic acid,

such as decomposing urine, for their formation. They are usually amorphous, soft, crumbly, and almost colourless. They are readily recognised by the fact that they dissolve readily in acids with effervescence, whereas those composed of calcium oxalate only dissolve with effervescence after incineration.

Cystin calculi are decidedly rare, because cystinuria is a very rare condition, but on the other hand a large proportion of cystinuric individuals form calculi at some period. Not infrequently a succession of small calculi is passed by such patients, giving rise to a series of more or less serious troubles, but in other instances such stones attain to a large size.

The nature of such calculi is usually easy of recognition. They are pale in colour, usually have a granular surface, and on section appear crystalline and very slightly yellow-tinted. When kept for long periods they tend to acquire a green tint.

Cystin stones leave practically no residue on combustion, and burn with a bluish flame. A fragment of the calculus is readily dissolved by ammonia, and on standing the ammoniacal solution deposits the characteristic hexagonal colourless plates similar to those of which cystin sediments are composed. To obtain good crystals it is advisable to use *dilute* ammonia as a solvent.

Xanthin calculi are very much rarer still, and only six or seven examples have been recorded. The first of these was described by Marcet in the early years of the last century.

They have for the most part occurred in children. They are usually smooth, of a brownish-cinnamon tint, are moderately hard, and a polish may be imparted to the surface by rubbing. Such calculi leave practically no residue on combustion. When heated with nitric acid upon a porcelain dish the material leaves a *yellow* residue, which when caustic soda is added assumes a red colour and on warming a purple tint. This is the characteristic xanthin reaction which must be distinguished from the murexide reaction of uric acid.

Cholesterin Calculi.—It would seem probable that calculi rich in cholesterin removed or passed from the urinary bladder have usually been gall-stones which have found their way thither through a fistulous passage. Some have undoubtedly been of this nature, being faceted and rich in bile-pigment, and associated with the passage of bile in the urine of patients who were not jaundiced.

However, no such origin can be ascribed to a remarkable stone examined by Horbaczewski, which contained 95·87 per cent of cholesterin. It had been removed by supra-pubic lithotomy from the bladder of a little girl aged six, and had been regarded as a cystin stone. The calculus weighed no less than 25·4 grammes.

Indigo Calculi.—Small renal calculi consisting of indigo have been described by two or three observers, the earliest example by W. M. Ord.

Urostealith.—Under this name are classed certain very rare concretions found in the bladder, which are soft and elastic in the moist state, but friable when dried. They burn with a luminous flame, and in burning emit an aromatic odour. In some specimens analysed by Horbaczewski a large proportion of the organic material was soluble in ether, and the residue from the ethereal extract consisted of fats and fatty acids, with traces of cholesterin.

The origin and mode of formation of such calculi are quite unknown.

XVIII. Drugs, Poisons, Accidental Constituents of Urine

In this final section it will be necessary to speak of certain substances met with in the urine which in themselves are of no clinical importance, but which it is necessary to recognise if only for this reason.

Various drugs and also poisonous substances appear in the urine either unaltered, or after undergoing changes in the body, and influence its properties.

Some of these may be recognised by the odour which they impart, such as copaiba and turpentine, which latter when ingested causes the urine to smell of violets.

Others again influence the colour, as for instance santonin, which imparts a peculiar bright yellow tint which changes to a beautiful pink with excess of an alkali. Chrysophanic acid, too, which is contained in rhubarb and senna and appears in the urine when they are taken, yields a pink tint with alkalies. The urine of patients taking salicylates sometimes has a brown colour, and may have a slight reducing action upon Fehling's solution. The presence of salicylates is easily detected by the purple colour which appears in the path of a falling drop of ferric chloride solution. This test is best performed with diluted urine, as otherwise the purple colour is apt to be masked by precipitated phosphates.

The peculiar greenish-brown colour of carboluria is ascribed by Baumann and others to the presence of hydroquinone. Phenol cannot be detected in such urines by its colour test with ferric chloride, as it exists in them in the form of an aromatic sulphate. The presence of an excessive amount of ethereal sulphate serves as a useful test in such cases, for the urine gives little or even no precipitate with a barium salt, whereas, after boiling with hydrochloric acid a copious precipitation of barium sulphate will be obtained.

Carboluria occurs after the administration of phenol by the mouth, or after the application of carbolic compresses, especially to children. It is also often seen after the administration of salol.

In cases of picric-acid poisoning the urine has a red colour from a partial conversion of the picric into picramic acid. Picric acid may be extracted from such urine by shaking with ether, to which it imparts a yellow tint.

Urine containing copaiba not only has a highly characteristic odour, but it yields with cold nitric acid an opaque white ring which may be mistaken for albumin, and with hydrochloric acid a purplish colour which shows a characteristic spectrum of three absorption bands.

Rosaniline gives a pink colour to the urine of patients who are taking it, and methylene-blue imparts a brilliant green tint, or a blue colour if large doses have been taken.

The pink pigment with a bright green fluorescence which results from the eating of certain kinds of sweetmeats may also be again referred to.

Bromides and iodides may be recognised by the addition of chlorine water, which sets free their halogen components. On shaking with chloroform the latter is coloured brown by bromine and purple by iodine. It should be remembered that urine containing iodide yields a blue colour with guaiacum and ozonic ether, but rather more slowly than blood-stained urine.

Hæmatoporphyrinuria may result from the administration of sulphonal and its allies.

Some poisons produce conspicuous changes in the urine which are due to injury done in their passage. Thus cantharides or turpentine may produce hæmaturia by irritating the kidneys, and potassium chlorate in poisonous doses may set free hæmoglobin in the blood, and so cause a copious excretion of methæmoglobin.

Many other drugs and tonic substances may be recognised in urine, but the processes for their detection do not fall within the scope of this article.

Urine, Bacteria in.

Modes of Infection	307
Cystitis	309
Nephritis	310
Infective Diseases	310
Typhoid Fever	310
Tuberculosis	311
Gonorrhœa	312
Actinomycosis	312
Pneumaturia	212
Hydrothionuria	312

The urine as contained in the healthy bladder is normally free from bacteria.

The exact significance to be attached to the presence of bacteria in the urine depends in no small degree on the recognition of the channel through which the infection has taken place.

Modes of Infection. — A. *Infection from without.*— Urine passed in the everyday manner, received in a sterile vessel and preserved from contact with the air, does not always remain sterile, for the normal urethra, male or female, nearly always contains bacteria.

Lustgarten and Mannaberg who were the first to investigate the flora of the healthy urethra, found in stained films, or in cultures, the smegma bacillus, the pseudo-gonococcus, and the staphylococcus pyogenes aureus, besides other organisms of less importance.

Rovsing was able to recover nearly all the organisms which he had already described as occurring in cystitis.

Melchior found the streptococcus pyogenes and the bacillus coli communis, as well as other organisms.

Petit and Wassermann, on the other hand, found neither pathogenic organisms nor the ordinary organisms of cystitis.

Schenk and Austerlitz investigated the bacteriology of the urethra in healthy women. After cleansing not only the meatus, *but also the anterior part of the urethra*, they found the urethra sterile in 30 out of 60 cases. In only 2 cases out of the others were pathogenic organisms found. In the remaining 28 cases they identified the organisms present with those commonly found in the vestibule, and concluded that they had migrated thence into the urethra. It is obvious that an infection in this way is more likely to occur in women than in men.

Melchior found the bacilli coli communis in the vagina or on the vulva of women in half the cases which he examined, and not infrequently in the preputial secretion of men.

It is seen, therefore, that in a large proportion of cases the healthy urethra and external genital organs abound in organisms which may be carried to the bladder, and give rise to an infection of the urine or of the bladder wall if the conditions are favourable, even when the greatest care is taken in the sterilisation of instruments. The passage of instruments is by far the commonest cause of infection, although, fortunately, the other conditions do not favour the growth of bacteria in the urine, except in a very small proportion of cases.

Where there exists any microbic infection of the urethra the risk of infection attending catheterisation is, of course, greatly increased.

In women it is probably owing to the shortness of the urethra, combined with the liability of the parts to injury during parturition, and the more frequent occurrence of disease in the neighbouring parts, that bacteria may make their way into the bladder by the urethra without the aid of a catheter.

In men they are not likely to do so except by the direct extension backwards of an infective process in the urethra.

B. *By direct extension of disease from neighbouring organs.*

Abscesses arising in various parts may burst into the bladder, or fistulous communications

may be established with the bowel. An inflammation of any of the surrounding structures may implicate the bladder wall directly, and sometimes the bladder may become infected indirectly, by the blood or lymph stream, from disease of a neighbouring organ.

C. *From the kidney and ureter.*

In infection of the kidney, with gross lesions, it is universally admitted that urinary infection frequently occurs. It is still disputed, however, whether a healthy kidney will allow the passage of bacteria from the blood-stream into the urine.

It is known that in certain diseases, *e.g.* typhoid, pneumonia, and erysipelas, the specific micro-organisms sometimes make their appearance in the urine. In some of these cases the urine remains free from albumin, and at the same time there may be no symptoms of any affection of the kidney. Moreover, the kidney may be found healthy and free from infection on post-mortem examination. It has been argued, however, that morbid changes actually occur in these cases, but are only microscopical in degree and insufficient to give rise to any appreciable symptoms. It is manifestly almost impossible to disprove this theory as regards man, for it would entail, among other things, a complete histological examination of the kidneys by means of serial sections.

Much of the experimental evidence is vitiated by the fact that the number of organisms injected into the circulation was larger than is within the fair limits of experiment.

Wyssokowitsch showed that microbes injected into the circulation of animals rapidly diminish in number, and that saprophytes soon disappear altogether, whereas organisms which are pathogenic for the species experimented on show a subsequent increase. He denied that physiological excretion ever took place. He failed to find injected bacteria in the urine before such time as he considered necessary for gross lesions of the kidney to be produced, and was always able to demonstrate naked-eye extravasation or small foci of cellular infiltration post-mortem when he found organisms in the urine. On the other hand, Biedl and Kraus were able to recover from the urine, in some cases at the end of twelve minutes, organisms which they had injected into the blood-stream, and they assumed that no change could have taken place in the kidney in so short a time. Von Klecki, using much smaller quantities, confirmed their results, in one experiment finding the organisms in the urine as early as three minutes after injection. He also found that they ceased to appear in the urine before they had disappeared from the circulation. On microscopical examination, after killing the animal, the organisms could be seen lying in the glomeruli without any associated histological change.

Although objections can be and have been taken to the conclusion drawn from these experiments, namely, that organisms can pass through the "intact" kidney, the discussion is rather of academic interest, for it is clear that if some structural alteration is necessary before this can occur, it must be infinitesimal in degree and certainly less than can be appreciated clinically. In almost any severe infection where organisms may at one time or other be found circulating in the blood, the conditions in the kidney are such as would favour an escape of bacteria from it into the urine.

Kraus, working at the urinary infections in infective diseases, recovered organisms from the urine in 36 out of 70 cases, but he only found those of the specific process in cases of typhoid fever and puerperal infections. The results of Kraus are of great interest because they tend to show that, with a lowered resistance, arising from many and varied infections, any organisms may penetrate into the blood-stream from the intestine, the pharynx, or other mucous surface which is not itself the seat of the morbid process. It follows from this that organisms found in the circulating blood are not necessarily those of the primary disease.

A large number of recent observations point to the same conclusion.

Posner and Lewin have shown that by simultaneous mechanical occlusion of the anus and urethra a general infection can be caused, and that organisms will then be found in the urine. That the urinary infection is not due to direct extension from the rectum to the bladder but takes place indirectly through the blood-stream, is proved by the fact that if one renal artery is also ligatured, infection of the corresponding kidney does not occur.

Of late years a good deal of attention has been directed to cases in man, of cystitis or bacteriuria, where this mode of origin has seemed probable. Hutinel, Hogge, and others have pointed out the frequent association of cystitis and intestinal affections, the urinary infections in some cases probably taking place indirectly by way of the circulation.

D. *Infection of the bladder by the blood-stream.*

Although there is no theoretical objection to the *bladder* becoming infected and secondarily infecting the urine in this way, there is no evidence that this often happens. The best example is to be found in tubercular disease of the bladder, which sometimes occurs in the absence of any affection of any other part of the genito-urinary tract.

The precautions which must be taken in collecting samples of urine for bacteriological examination vary considerably with the nature of the investigation required.

For all ordinary purposes in the male, at any rate, it is sufficient after cleansing the meatus to allow the patient to pass some of his urine into a sterilised vessel; the first portion, which

is more likely to contain organisms from the urethra, being rejected. In the female it is always necessary to withdraw a specimen with a sterilised catheter, care being taken to avoid contamination of the instrument by the external genitals. Into the procedure necessary in those cases where it is of vital importance to exclude every possibility of contamination from the urethra, or where it is desirable to remove urine from one or other ureter, there is no need to enter here. In any case all cultural examinations should be made immediately. A very short delay in so doing is sufficient to vitiate any result.

Cystitis.—The exact bacteriological examination of the urine in cases of cystitis is of quite recent growth.

The researches of Bumm, Clado, Albarran and Hallé, Rovsing, Krogius, and more especially of Melchior, following rapidly on one another, served to prove that cystitis might be caused by a number of different organisms. A comparison of their work, however, shows that certain of the organisms in question, notably bacillus coli, had been described under several names, so that the total number of the different varieties commonly found is much smaller than appeared at one time probable. It is true also that, even allowing for this, the results obtained varied considerably with the different observers. The reason for this latter circumstance is at once seen if the widely differing clinical fields from which their material was drawn are considered. Their investigations have been repeated by a very large number of other workers without much being added to our stock of knowledge on this point. The sum of all this work shows that, including all cases of cystitis in both sexes, the bacillus coli is the commonest organism found, and after that in turn the staphylococcus aureus and albus, proteus vulgaris, streptococcus pyogenes, and the gonococcus. Less frequently the bacillus typhosus, the pneumococcus, and Friedländer's bacillus may be present. Finally, a large number of other organisms, not otherwise pathogenic for man, have been described as occurring occasionally.

Any of these bacteria may be present in pure culture, but two or three or even more varieties may be found in the same urine. It is important to bear this in mind, lest, for example, the presence of the bacillus coli or one of the pyogenic cocci in a specimen of urine should lead to the gonococcus, or the tubercle bacillus, which may be associated with them, being overlooked.

The attempts which have been made to associate special organisms with different forms of cystitis have not stood the test of time. It is possible, of course, to classify the causes of cystitis according to the organism present, but the classification would give no clue to the character or intensity of the inflammation in any given case. The reaction of the urine as freshly passed does vary, however, with the organism. In the majority of cases it is acid in correspondence with the preponderance of cases caused by the bacillus coli. In cystitis, due to the gonococcus or the tubercle bacillus, the urine is also acid. When the pyogenic cocci are present the urine presents usually a diminished acidity or may be faintly alkaline. In the presence of proteus vulgaris the reaction is strongly alkaline (ammoniacal), and the same is true in the case of some of the less frequently found organisms already mentioned. Very little is known of strictly anaërobic organisms as a cause of cystitis, but Albarran and Collet have described them as occurring in periurethral abscesses and extravasations of urine. Their presence is usually marked by a foul smell.

All attempts to produce cystitis by the mere injection of organisms into the healthy bladder have failed except in those cases in which the proteus vulgaris was the one employed. The bacteria are gradually expelled, and after three or four days, as a rule, no trace of them can be found in the urine. It should be noted that not even bacteriuria can be produced in this way. If, however, retention of urine be produced, or some injury be done to the bladder, or a small stone be introduced, then the injection into the bladder of any of the usually found organisms will set up a cystitis, though retention or a like injury by themselves do not. This coincides with what is found in man under natural conditions. As we have seen, organisms must often reach the bladder from the kidney or through the urethra, but if the bladder be healthy and capable of emptying itself no multiplication takes place.

Between bacteriuria, in which bacteria alone are present as an abnormal constituent of the urine, and cystitis the writer does not think it is possible to draw any hard-and-fast line either clinically or pathologically. On centrifugalising the urine, leucocytes and epithelial cells are always to be found in every bacteriuria, and a continuous series can be traced from such cases through all the stages up to the most severe purulent cases of cystitis. The healthy bladder with normal urine is a barrier against bacteriuria, even as it is against cystitis. If this were not so bacteriuria would follow the passage of a catheter in a very large proportion of cases. The difference between cystitis and bacteriuria is merely one of degree, and depends partly on the virulence of the organism, partly on the resistance of the patient, modified as it may be by so many causes, local or general.

In certain rare cases cystitis may be due to chemical irritants such as cantharides, and under such circumstances the urine may remain free from micro-organisms.

In pyelitis, pyelo-nephritis, and ascending nephritis, the organisms found are those commonly present in cystitis.

Nephritis.—In infective processes associated nephritis may be due to the circulating toxines, and organisms may be absent from the kidney and the urine. In other cases the organisms of the primary disease or of some secondary infection may be present in the kidney and pass into the urine. In so-called primary nephritis, Mannaberg found streptococci, Letzerich bacilli, but their etiological connection with the disease is doubtful.

Infective Diseases. — Examination of the urine in infective diseases gives varying results. It is gradually becoming more and more evident that, even in diseases, *e.g.* typhoid, in which the morbid processes are more or less restricted to certain situations, the organisms must at some time or other circulate in the blood-stream. In certain pyogenic infections, *e.g.* acute osteomyelitis, the condition is often complicated by a true septicæmia, and the organisms are constantly in the blood in greater or less numbers. On the other hand, in tetanus the organisms have never been found except locally. The oftener organisms find their way into the blood-stream the greater will be the frequency of their appearance in the urine. Unfortunately their presence there is no indication of a specific nephritis, for, as we have seen, the organisms may be absent from the kidney after death when they have been discovered in the urine during life; nor does it necessarily imply a severe blood infection, in other words, a true septicæmia, for a few stray organisms may escape by the kidney.

It is, however, safe to say that in any given disease, for obvions reasons, an infection of the urine is more likely to occur in severe than in mild cases.

In addition it must be remembered that any lowered resistance of the body favours the penetration into the blood-stream of organisms commonly living as saprophytes on the mucous surfaces of the body. Organisms found circulating in the blood are therefore not necessarily those of the specific disease. This is true also as regards the urine. If, therefore, the organisms found in the urine belong to the class which commonly lives on the mucous or even skin surfaces of the body saprophytically, the conclusions drawn from their presence must be very guarded. This objection does not weigh against those organisms which are only found in the body in diseased conditions.

The specific organisms of the disease have been found frequently in the urine in pneumonia and other pneumococcal infections, in malignant endocarditis, erysipelas, puerperal septicæmia, and many other pyogenic infections in plague, glanders, and typhoid fever. The significance to be attached to their discovery may be gathered from the immediately preceding sentences.

The identification of these organisms can only be arrived at by cultural experiment. Their presence in the urine indicates the need for its disinfection. This is more especially so in the case of typhoid fever.

Typhoid Fever.—Of all general diseases of microbic origin typhoid fever is the one in which the specific organisms are most often present in the urine.

Gaffky, who may be called one of the discoverers of the typhoid bacillus, found it in the kidney in 3 cases out of 7. It has been since found by many observers in many cases. It is not, however, so frequently recovered culturally from that organ as from the spleen and abdominal lymphatic glands, partly, no doubt, owing to its being present in smaller numbers, but partly also owing to the kidney being less succulent, and so presenting less favourable material for cultivations as commonly carried out.

Seitz was the first to describe its occurrence in the urine, and his results were soon confirmed by Konjajeff, Neumann, Karlinski, and others. These earlier cases are, of course, open to the objection that affects all the early attempts at the identification of the typhoid bacillus, viz. the absence of sufficient data for distinguishing it from the bacillus coli.

Horton Smith, Petruschky, and Richardson were the first to apply the serum agglutination test to the organisms they obtained, and so to prove as absolutely as we are able their specific nature.

Typhoid bacilli have been found in the urine by different observers in a varying proportion of the cases examined. Neumann found them in 11 cases out of 48 cases examined—8 times in countless numbers. In 2 other cases he found the streptococcus pyogenes in pure culture. Horton Smith found them in 17 cases out of 45; Richardson in 23 out of 103. Other observers have found them in varying proportion, from under 10 up to 100 per cent of the cases examined.

It is now generally held that in any large series taken consecutively without selection, they are to be found in about 25 per cent of the cases. In a large proportion of such positive cases, as pointed out first by Neumann, they are present in very large numbers, making the urine as freshly passed quite turbid, and on shaking the urine, if it be in a glass vessel, it presents a peculiar shimmering appearance which is very suggestive. As a good deal of stress has been laid on this point by many writers, it may be pointed out that the same appearances can be seen in bacteriuria caused by other organisms, so that its significance is very limited. In other cases the bacteria are relatively scanty in number—a fact which suggests that in some of the "negative" cases they may have escaped detection owing to this circumstance. Wright and Semple, who in-

cubated large quantities of the urine directly, found the organism in 6 out of 7 cases, and as early as the tenth day. Their identification of the bacillus was not as complete as with our present knowledge it might have been, but their large proportion of positive results may have been due, not to error, but to the large quantity of material taken. In addition to the bacteria the urine commonly contains albumin, often in small amount, but it may entirely be absent; a few pus cells and epithelial cells can, however, be always found.

In the condition already described there is an infection of the urine, the bacilli multiply in it, but the bladder wall is not obviously affected, and the signs and symptoms of cystitis are practically absent. On the other hand, in a small proportion of cases pus and bladder epithelium may be present in large amount and the symptoms of cystitis appear. Between these two extremes there are cases of every degree of severity.

In addition to the cases where the infection seems limited to the bladder, or its contained urine, there are others in which the chief urinary symptoms point to the kidney. Albumin, casts, and perhaps blood appear; hæmaturia, indeed, may in rare cases be the first sign of serious illness. It must not be supposed that all cases of nephritis occurring in typhoid fever are due to the typhoid bacilli, for in some of these cases they may be absent from the urine and even from the kidney. There is, indeed, no good evidence to show that the nephritis is ever due to the actual presence of the typhoid bacillus itself apart from its toxin. The changes in the kidney are not restricted to the area of the small lymphoid nodules in which the typhoid bacilli usually lie when present in the organ.

Date of Appearance.—With some doubtful exceptions there are no records of the appearance of bacilli in the urine before the end of the second week; on the other hand, they may be noticed for the first time three or four weeks, or even more, after the temperature has become normal.

The condition, whether of cystitis or merely bacteriuria, has a tendency to disappear spontaneously, as a rule, in two or three weeks, but this period may be greatly exceeded. Gwynn has recorded a case where the patient suffered from cystitis for four years after an attack of typhoid fever, the typhoid bacillus being recovered at the end of that time, and Young a case lasting seven years. Houston found this organism in a case of cystitis which had lasted three years and which dated from the time the patient had nursed a child with "diarrhœa." There was no evidence that the patient herself had suffered from typhoid fever.

Neumann has tried to prove that the urinary infection coincides with the eruption of the roseola, and now that it is known that the rose-spots contain the specific organism, there can be no difficulty in accepting the view that an overflow of typhoid bacilli into the blood-stream may infect the kidney and the skin simultaneously. In this case it might be expected that cases would be recorded as occurring earlier than at present is admitted, in a correspondence with the date of appearance of the rash, but it is probable that in the earlier stages of the disease, at the time of the first appearance of the eruption, the bladder is in a condition to resist infection. On the other hand, Osler records 12 cases of typhoid fever in which rose-spots made their appearance during an apyrexial period. These may be taken as analogous to the late appearance of the bacteriuria.

In the prevention of the spread of typhoid fever the disinfection of the urine of patients suffering from this disease now plays a large part. The late appearance of the organisms ought to be borne in mind, and an eye kept on the condition of the urine for some weeks after the temperature has become normal.

Cystitis and bacteriuria occurring in the course of typhoid fever are not always caused by the typhoid bacillus. The bacillus coli, the streptococcus pyogenes, and the staphylococcus pyogenes aureus are the more likely foreign organisms to be found, and they may be present in pure culture or associated with the specific organism.

The organism can only be positively identified by careful and thorough bacteriological examination.

Tubercle Bacillus.—Tubercle bacilli are present in the urine only when there exists some tubercular affection of the genito-urinary tract.

Phillippowicz, quoted by Sherrington, it is true, claimed to have demonstrated them in the urine of a case of general tuberculosis in which the kidneys after death showed no tubercular lesions, but this is a solitary exception. In the early stages before ulceration has taken place, of course, they are absent from the urine. Before a negative report can be justified the urine should be repeatedly examined, for when tubercle bacilli are present it may be only in very scanty numbers. They are often found lying together in small clumps of three or four to twenty or more, the intervening parts of the film showing none at all. A large quantity of the urine should be allowed to sediment, or better still, should be centrifugalised. By this latter means tubercle bacilli can sometimes be detected in cases where there is only a very small amount of ulceration, and consequently also of discharge.

The smegma bacillus, which is a common saprophyte in the vulvar and preputial secretion, and which is occasionally present in the urethra, adds considerably to the difficulty of making a diagnosis by microscopical evidence alone, since

this organism in some cases will show a resistance to the action of acid *and* alcohol equal to that of the tubercle bacillus. A carefully obtained catheter specimen will often avoid this difficulty, but in cases where any doubt still remains, and indeed in every case, inoculation into a susceptible animal will decide the matter with absolute certainty.

Gonococcus.—The urine in the great majority of cases becomes first contaminated with gonococci in its passage down the urethra. True gonorrhœal cystitis does, however, occur. Wertheim was able to demonstrate culturally and microscopically the presence of gonococci in a portion of the mucous membrane removed from the bladder of a young girl. When the urine contains an appreciable amount of pus, films may be made from it, if necessary, after centrifugalising. In the absence of much pus the best material to take for examination lies in the gonorrhœal "threads," which consist of shreds of mucus with adherent leucocytes. If the specimens are stained by Gram's method and counterstained by some contrasting dye, the gonococci will be found stained by the counterstain (they lose the stain in Gram's method), and any of the ordinary pyogenic cocci, if present, will be stained by the gentian violet. The typical shape and arrangement of their intracellular situation, taken with this staining reaction, will in a majority of the cases suffice to establish the diagnosis. When the gonococci are only present in very scanty numbers repeated examination may be necessary.

Sometimes admixture with foreign organisms renders the diagnosis by microscopical examination difficult or even impossible. In such cases of difficulty, and also where the cocci are few in number, cultural experiments, which require special media, may enable a diagnosis to be arrived at.

Actinomyces.—A large number of cases are now on record (Tuffier) of actinomycosis affecting the urinary tract. The presence of the characteristic granules in the urine with a microscopical examination of fresh and stained specimens is sufficient to establish a diagnosis.

Pneumaturia.—When gas is voided with the urine there exists in most cases a fistulous communication between the bladder and the intestine. In certain rare cases gas is actually formed in the bladder without any such lesion being present, and is then due to action of micro-organisms.

In 1883 Guiard published four cases of spontaneous development of gas in the bladder. The gas was odourless, but no chemical analysis was made of it nor any proper bacteriological examination. The presence of large numbers of "vibriones" was noted in two cases. All the patients were diabetics and suffered from cystitis, and the author suggested that fermentation by some micro-organism was the cause of the gas formation. In the same year Dumenil published a case of a similar nature.

In 1889 Fr. Muller described a case also in a diabetic.

A bacillus was present in large numbers, but no pure culture was obtained. An elaborate analysis of the gas found in the bladder gave CO_2 19·8 per cent, N 35 per cent (probably by interchange with the gases of the blood), H 44 per cent, 0·23 per cent, CH_4 ·086 per cent.

Senator demonstrated the presence of T. cerevisiæ in one case, the gas being CO_2 only, and Frisch the parasite of thrush (oidium albicans), and Péré an organism of the coli group. The urine of all these cases contained sugar. Montt Saavedro recovered Friedländer's bacillus from two cases of cystitis, with free gas in the urine. In one of these cases the urine contained no sugar. Heyse and Schnitzler have also published cases of pneumaturia, the former's due to the B. lactis aërogenes, the latter to B. coli, in which the urine was free from sugar. It is well to remember, as pointed out by Senator, at least as to cases where the fermentation is due to yeast, that though the urine may be free from sugar when voided, it may have contained it as secreted.

Gas-forming, strictly anaërobic, organisms have been found post-mortem by Williams, Lindenthal, Dunham, and others, as the cause of emphysema of some part of the genito-urinary tract, free gas being also present in the urinary passages, and it is not improbable that anaërobes may be present more often than is suspected. Their presence would enable us to understand more readily those cases of gas formation in sugar-free urines which have been recorded.

Hydrothionuria.—The presence of sulphuretted hydrogen in the urine may result from the action of micro-organisms in the bladder, as shown by the cases of Rosenheim, Karplus, and Savor. The organism found has always been some member of the coli group. Apart from the existence of vesico-intestinal fistulæ, it may be doubted whether cases of hydrothionuria can occur in any other way.

The earlier explanations of diffusion from the intestine into the bladder or excretion from the blood by the kidney of gas absorbed from the bowel may now be reasonably doubted.

Urinettes.—Public urinals for the use of women; large cities are slowly being provided with them.

Uriniferous.—Carrying or transmitting urine, *e.g.* the tubules of the kidney, the ureters, and the urethra. *See* Kidneys, Physiology of.

Urino-.—Relating to the urine or to the urinary apparatus; e.g. *urinogenital tract* (the various urinary and genital organs), *urinocryoscopy* (the cryoscopic examination of the

urine), *urinometer* (an instrument for determining the specific gravity of the urine), etc.

Urnings.—Individuals (male or female) with sexual perversion, preferring persons of their own sex to those of the opposite sex. *See* URANISM.

Uro-.—In compound words *uro-* (Gr. οὖρον, urine; οὐρά, the tail) means either relating to the urine or relating to the tail or the caudal region; e.g. *uromelus* (a monstrosity in which the lower limbs are fused into a projection resembling a tail, sympodia), *urocele* (swelling of the scrotum from extravasation of urine), *urodynia* (painful micturition), *uroschisis* (suppression of urine), etc.

Uroanthropos. *See* HOMO-CAUDATUS.

Urobilin. *See* INTESTINES, DISEASES OF (*Anatomical and Physiological Consideration*) (*Result of Removal of Large Intestine*); LIVER, DISEASES OF (*Venous Congestion, Physical Signs, Urine*); PHYSIOLOGY, EXCRETION (*Urine, Physical Characters, Pigments*); PIGMENTS OF THE BODY AND EXCRETA (*Bile-Pigments*); SPECTROSCOPE IN MEDICINE (Fig. 10); URINE, PATHOLOGICAL CHANGES IN (*Pigments*). The presence of an excess of urobilin in the urine constitutes *urobilinuria*.

Urochrome. *See* PHYSIOLOGY, EXCRETION (*Urine, Physical Characters, Pigments*); PIGMENTS OF THE BODY AND EXCRETA (*Bile-Pigments*); SPECTROSCOPE IN MEDICINE (*Spectrum of Urochrome*); URINE, PATHOLOGICAL CHANGES IN (*Pigments*).

Urocyanogen.—A blue pigment found sometimes in urine.

Urocyanosis.—A blue discoloration of the urine due to indican.

Uroerythrin. *See* PHYSIOLOGY, EXCRETION (*Urine, Characters, Pigments*); SPECTROSCOPE IN MEDICINE (*Various Spectra*); URINE, PATHOLOGICAL CHANGES IN (*Colour, Pigments, Uroerythrin*).

Urogenital.—Relating to the urinary and the genital organs; *e.g.* the *urogenital sinus* in the embryo (*see* EMBRYOLOGY, *Human*); *urogenital tuberculosis* (*see* TUBERCULOSIS, *Lesions of Various Organs*).

Urogosan.—A preparation consisting of gonosan and urotropin, recommended (in capsules) in cystitis and urethritis.

Urohæmatoporphyrin.—A urinary pigment found in certain diseased conditions, such as Addison's disease, acute rheumatism, etc. *See* HÆMATOPORPHYRINURIA.

Urolith.—A urinary stone or calculus; the presence of such calculi in the bladder or kidneys is called *urolithiasis*. *See* BLADDER, INJURIES AND DISEASES OF (*Stone*).

Urophæin.—A urinary pigment, perhaps the cause of the odour of the urine.

Uropoietic.—Secreting or excreting urine, thus the kidneys are described as uropoietic organs and their function is named uropoiesis.

Urorosein.—A rose-colour urinary pigment, probably of intestinal origin; it is obtained by the action of hydrochloric acid upon urine from which the nearly colourless chromogen has been isolated in crystalline form. *See* PIGMENTS OF THE BODY AND EXCRETA (*Melanins, etc.*).

Urostealiths. *See* URINE, PATHOLOGICAL CHANGES IN (*Lipuria, Calculi, Urostealiths*).

Urotoxic.—Belonging to poisonous substances present in the urine or simply to the urine itself as a cause of poisoning, *e.g.* the urotoxic coefficient in uræmia. *See* URÆMIA (*Theories of Causation*). The urotoxic coefficient depends on the number of units or urotoxies formed in twenty-four hours by one kilogramme of an individual, and each urotoxy (unit of toxicity) is the amount necessary to kill a kilogramme of living substance.

Urotropin.—Hexamethylenetetramine. Consists of colourless crystals obtained from the action of ammonia upon formaldehyde. It is soluble in water. *Dose*, 5-10 grs. It is used as a urinary antiseptic, and was supposed to act by the liberation of formaldehyde in the urine. This is incorrect, but its antiseptic value is undoubted nevertheless. It is especially useful when the urine is alkaline, purulent, and loaded with phosphates. It is employed widely in cystitis of various kinds, and also as a prophylactic during the course of prolonged catheterisation of the bladder.

Uroxanthin.—A yellow pigment found in urine, and yielding indican blue when oxidised.

Urtica. *See* STINGING PLANTS; DERMATITIS TRAUMATICA ET VENENATA (*Vegetable Causal Agents*).

Urticaria.

SYN.:—*Nettlerash, cnidosis* (κνίδη, *a nettle*).

DEFINITION	314
SYMPTOMS	314
VARIETIES	314
ETIOLOGY	315
PATHOLOGY	316
TREATMENT	317

See also CONJUNCTIVA, DISEASES OF (*Exanthematous Conjunctivitis*); DERMATITIS TRAUMATICA ET VENENATA; DERMATITIS HERPETIFORMIS (*Urti-*

carial Forms), DRUG ERUPTIONS (*Urticarial*); GOUT (*Irregular, Cutaneous System*); HYDATID DISEASE (*Diagnosis, Hydatid Rash*); INTESTINES, DISEASES OF (*Enteritis, Symptoms of Ptomaine-Poisoning*); LEUCOCYTOSIS (*Eosinophile Variety*); MUSCLES, DISEASES OF (*Polymyositis accompanying Urticarial Affections*); PEDICULOSIS (*Differential Diagnosis*); PRURITUS (*Causation*); RHEUMATISM, ACUTE (*Cutaneous Manifestations*); SNAKE BITES AND POISONOUS FISHES (*Mackerel-Poisoning*); STOMACH AND DUODENUM, DISEASES OF (*Symptomatology, Cutaneous*); STINGING INSECTS; STINGING PLANTS; THERAPEUTICS, SERUM THERAPY (*Toxic Effects of Antidiphtheritic Serum*); TUMOURS OF THE SKIN (*Urticaria Pigmentosa*).

DEFINITION.—Urticaria is an eruption characterised by the formation of wheals. Wheals are raised, flatly convex, circumscribed lesions, white or pinkish in colour, firm and elastic in consistence, which form rapidly, are more or less ephemeral, are associated with itching, tingling, or stinging sensations, and are similar to the lesions produced by the sting of the common nettle (*Urtica urens*), from which the eruption derives its name.

SYMPTOMS. — The eruption may come out suddenly without warning, or it may be preceded for a day or two by various prodromal symptoms, such as fever, malaise, lassitude, nausea, vomiting, diarrhœa, and epigastric pain, or by burning and tingling of the skin locally. Wheals vary greatly in size and shape. They are usually about the size of a split-pea or a small bean and discrete, but they may coalesce to form irregular, pad-like patches sometimes as large as the palm of the hand. As a rule they are circular in outline, but they may be irregular, and occasionally take the form of streaks, crescents, or gyrate figures.

They are solid and more or less elastic to the touch, and are generally elevated about 2 mm. above the level of the skin, though at times they may be scarcely raised, and may be difficult to feel. When they first appear they are pinkish in tinge, but in most cases this colour is quickly replaced by a whitish tint which has been compared to that of porcelain. Surrounding the wheal there is a hyperæmic halo which fades gradually into the neighbouring skin. Wheals vary in number in different cases from a few isolated lesions to hundreds of them.

They do not show any tendency to be arranged systematically, or to be grouped in any special manner. They may occur in any situation in the skin, but are most liable to form in the parts covered by clothing. They may be present also in the mucous membranes, and are most common in those of the mouth, pharynx, gastro-intestinal and respiratory tracts. In the latter situation they cause sensations of suffocation and dyspnœa of the asthmatic type, and occasionally asphyxia, while in the gastro-intestinal tract they are associated with nausea, vomiting, and diarrhœa. The subjective symptoms of urticaria are not infrequently worse immediately before the wheals form, and they tend to diminish when the lesions are fully evolved. Occasionally wheals may go on to vesicular or bullous formation, and they may even become hæmorrhagic. The life of a wheal is, as a rule, brief; it generally lasts only a few minutes, but at times it may persist for several hours, and sometimes for days.

On disappearing wheals seldom leave any trace behind them, such as staining or desquamation, but on the other hand they may be replaced by a hemp-seed sized inflammatory papule, or in rare instances be followed by pigmentation. The latter sequela must on no account be confused with the disease known as urticaria pigmentosum, which belongs to a different category from true urticaria.

On account of the distressing subjective symptoms associated with the efflorescence of wheals, the patient is induced to rub the skin, and by so doing to cause more wheals to form, or he may scratch and excoriate it, and by inoculating pyogenic organisms produce pustular lesions of the type of ecthyma.

VARIETIES.—Several varieties of urticarial eruptions are distinguished, and a brief reference to these will serve to indicate the leading clinical types of the disease. There are four chief varieties of urticaria, namely: *Urticaria acuta, U. chronica, U. factitia*, and *U. papulosa*.

1. *Urticaria Acuta.*—The eruption in acute urticaria is, as a rule, associated with a rise in temperature of several degrees. Prodromal symptoms are generally present, and the condition is almost invariably the result of the circulation in the blood of food-toxins or drugs. The eruption is profuse, the wheals remaining discrete on the face, but tending to run together to form patches on the trunk and limbs. It readily disappears when the offending toxins have been eliminated and leaves no trace of its occurrence.

2. *Urticaria Chronica.*—Chronic urticaria is so named, not because the individual wheals persist much longer than those in the acute variety, but because the dermatitis is kept up for a prolonged period by successive crops of wheals, which appear at regular or irregular intervals, and sometimes exhibit a periodicity which suggests malaria. The wheals are discrete and comparatively few in number. It is often difficult to trace the cause of this condition, and it is far more intractable to treatment than the acute type.

3. *Urticaria Factitia.*—Factitious urticaria is the name given to wheals which are readily excited by local irritation. It occurs in a peculiar condition of the skin in which there is an excessive state of irritability of the nerve-

mechanism which regulates the cutaneous blood-vessels. In such cases the slightest irritation results in the formation of wheals. The stroke of the finger-nail down the back produces a linear wheal, and in a marked case raised white letters with erythematous borders may be written across the back in wheals by any pointed instrument ("dermographia"). The artificial wheals may only persist for a few minutes, or they may last for several hours. In patients suffering from this condition of the skin the wheal-formation may be demonstrated even when the patient is asleep or under a general anæsthetic.

4. *Urticaria Papulosa* (*Lichen Urticatus of Bateman*).—The wheals in this type of urticaria are usually small. They are rarely white in colour, but present a pinkish tinge, and they are situated, as a rule, in the centre of an erythematous blotch. It is a disease of infancy and early childhood. In a severe case the child may be covered with the blotches in the evening. In the morning the majority of the wheals and blotches have disappeared, and have been replaced by small papules about the size of hemp-seed. These papules are intensely itchy and are generally excoriated from scratching, and capped by a tiny crust. Scratch-marks, excoriations, and pustular lesions may all be present to mask the real nature of the eruption. Occasionally vesicles or bullæ develop, these being chiefly found on the hands and feet. This singularly intractable type of urticaria is most common in infants, and is occasionally designated as "infantile urticaria." It forms one of the commonest diseases of infancy and early childhood. It is generally the result of the assimilation of some toxin from the alimentary canal. In most cases it is not associated with any general disturbance in the child's health, but there may be more or less chronic intestinal catarrh. It is rare to find it in children over ten years of age. Certain observers maintain that it may develop into the prurigo of Hebra, but this assertion has not been satisfactorily proved, and the chief argument against it is the fact that prurigo is an exceptionally rare disease in this country, while lichen urticarius is one of the commonest of all the skin-diseases met with in any of the children's hospitals. But besides these four main types of urticaria several sub-varieties have been specially named on account of differences in the size, shape, and character of the wheals, and other purely secondary considerations. These sub-varieties do not require more than a passing reference. When the wheals form variously shaped figures the names of *Urticaria figurata*, *U. annularis*, and *U. gyrata* have been applied to them. When they are unusually large, forming deep-seated, brawny yellow or reddish tubercles, the condition has been termed *U. tuberosa* and *U. gigans*. When they occur in the lax tissue about the eyelids or genitals, and form œdematous swellings, they constitute *U. œdematosa*, a variety of which is the "acute circumscribed œdema" of Quincke. When the presence of wheals is associated with a definite rise in temperature the condition is known as *U. febrilis*. If the wheal is short-lived it has been christened *U. evanida*, in contra-distinction to wheals which persist for hours or days, *U. perstans*. When they recur in crops it is known as *U. recurrens* or *U. intermittens*; and when vesicles, bullæ, or hæmorrhages form in the course of the eruption, it is known respectively as *U. vesiculosa*, *bullosa*, and *hæmorrhagica*.

Etiology.—Urticaria in all its forms should be regarded rather as a symptom than as a disease *per se*. The wheal is the result of a peculiar reaction on the part of the vaso-motor nerves of the skin to some irritant or toxin. This may act from without like the sting of a nettle, or from within by circulating in the blood and so reaching the skin, or reflexly through the higher nerve-centres, as the urticaria which occurs in certain individuals from intense emotional excitement. But in addition to the actual wheals which are formed, the skin in the neighbourhood of them is also rendered vulnerable, and slight rubbing brings out new lesions, showing that the irritant is either widely diffused, or that a reflex instability of the vaso-motor mechanism in the neighbourhood of the wheal is established.

Idiosyncrasy also plays an important part in the etiology of urticaria. In no disease is the time-honoured axiom of "one man's food is another man's poison" more applicable, for certain articles of food which are usually harmless will, in the smallest quantities, produce a profuse urticaria in susceptible individuals. Heredity and the neurotic temperament are also predisposing causes.

These general observations serve to indicate the great diversity of causes which may be responsible for an attack of urticaria. In some cases it is difficult, if not impossible, to discover any definite cause, for the habit may have been set up at some remote period, and the most trivial causes may be capable of occasioning a fresh outbreak.

It is customary and convenient to divide the causes of urticaria into two groups, namely, the direct or local causes and the indirect or reflex ones.

I. *Direct or Local Causes.*—(1) *Vegetable Irritants.*—The common nettle (*Urtica urens*) and the variety known as *Urtica dioica* are capable of causing wheals in the most resistant skins. The handling of certain plants which, as a rule, set up an erythemato-vesicular dermatitis, may produce an urticaria; for example, the *Primula obconica*, the poison ivy, and the cowhage.

(2) *Animal Irritants.*—The most common instance of animal irritants causing wheals are

the bites and stings of insects; for example, those of fleas, bed-bugs, wasps, gnats, and mosquitoes, and certain caterpillars, such as the common "woolly bear"; the bites of leeches, and the stings of several varieties of jelly-fishes.

(3) *Mechanical and other Irritants.*—Scratching and rubbing readily induce urticaria in a susceptible skin, or when some pruritic affection is present, such as scabies or prurigo; the application of the galvanic current, as in performing electrolysis for the removal of hairs, is often followed by a wheal; and a lash from a whip, if sharp enough, may cause one.

In some individuals the application of a lint-seed poultice will produce wheals, in others dusting with iodoform, or even sponging with boric lotion, will be sufficient to do so, and in highly susceptible patients even the smell of iodoform can cause urticaria. Again, in rare instances, cold air, cold winds, cold water, salt baths and Turkish baths, have all been known to produce it.

II. *Indirect or Reflex Causes.*—(1) *Age and Sex.*—Urticaria may affect either sex, though it is somewhat more common in females. It may occur at any age, but the papular variety may be said to be a disease of early life, while true urticarial wheals are rare in infancy.

(2) *Toxins.*—The most common cause of urticaria is the action of a toxin circulating in the blood, and acting directly or reflexly on the vaso-motor mechanism of the skin.

The poison may be in the nature of some drug, such as copaiba, turpentine, quinine, salicin, santonin, iodide of potassium, chloral or morphia, any of which in the smallest dose may cause a profuse urticaria in an individual with the requisite idiosyncrasy towards the drug.

Errors in diet are, however, more frequent causes of urticaria than drugs. The list of articles of diet which may set up urticaria is, of necessity, a long one, since the factor of idiosyncrasy plays such an important part. Among the most common offenders are shell-fish, and especially mussels, crabs, and oysters; oily fish or fish fried in oil, such as sardines, herrings, and eels; certain meats, as pork, veal, and sausages, especially when highly seasoned; fruits, as strawberries, raspberries, dates, and nuts; miscellaneous articles such as cheese, eggs, porridge, and pickles; the ordinary beverages, tea, cocoa, and coffee; and sweet wines such as port, moselle, and champagne.

Urticaria may also occur in association with gout, rheumatism, and malaria, and is due to the circulation in the blood of the respective toxins of these diseases. It not infrequently alternates with attacks of gout and chronic rheumatism, and has been known to replace the cold stage of a malarial paroxysm.

The serums and antitoxins which are employed therapeutically in various diseases may cause urticaria.

(3) *Disorders of other Organs.*—Digestive disturbances, chronic dyspepsia, and the like, may render the patient prone to attacks of urticaria. In infants and children a slight chronic intestinal catarrh may be associated with urticaria, and an outburst of wheals is in such cases liable to take place from such causes as a change of air, injudicious dieting, or the cutting of a tooth. A common cause of urticaria in children is the presence of worms.

Functional and organic disorders of the nervous system may also cause urticaria. Severe emotion, grief, anger, shame, and fright have all been known to do so. It may also occur in connection with neuralgia and locomotor ataxia.

Disorders of the genital system, such as diseases of the ovaries, may be associated with it, and it may occur during menstruation, pregnancy, and lactation, and has been known to follow the passing of a uterine sound. It has also been reported after an enema. Diseases of the liver with jaundice, and diseases of the kidneys with glycosuria and albuminuria, may be complicated by urticaria, and paroxysmal disorders, such as asthma and biliary colic, may alternate or be associated with it. In several instances a reflex urticaria has been reported after the tapping of a pleuritic effusion or a hydatid cyst.

PATHOLOGY.—It is customary to classify urticaria as an angioneurosis, and to define it pathologically as an acute spastic œdema, the result of a vaso-motor disturbance directly or reflexly induced. To a certain extent this definition is adequate, but it is insufficient, as it loses sight of the inflammatory changes which are part of the pathological process.

A wheal, such as that produced by a nettle, if it be excised a few minutes after it appears, presents the following histological architecture: —The capillaries of the skin are dilated, the lymph-spaces, especially in the deeper layers of the corium, are widened, and there is a consequent stretching, attenuation, and sometimes rupture of the fibrous elements of the skin, especially the elastic framework. Around the dilated vessels and lymphatics there is a deposition of polynuclear leucocytes, and sometimes fibrin. The epidermis is not implicated to any extent, and is simply raised up and stretched by the œdematous fluid beneath. But the cellular infiltration does not consist only of leucocytes, there is also a large number of mast-cells, and sometimes a few small connective tissue cells. In other words, the cellular infiltration is similar to that of an acute inflammatory process.

There is at present considerable diversity of opinion with regard to how these histological changes are brought about. It is generally believed that the irritating toxin causes directly or reflexly a spasmodic contraction of the small

vessels of the hypoderm—not of the capillaries of the corium, for they do not possess any muscular wall—and that this spasm is quickly replaced by a paralytic dilatation, or by a dilatation the result of a stimulation of the vaso-dilator nerves. This vaso-dilatation is responsible for the initial hyperæmia of the wheal, but associated with it there is an exudation of serum from the dilated vessel, which, by constricting the vessels through pressure, causes the anæmic appearance of the wheal to supervene. The lymph in the skin returns chiefly through the veins, and as these are constricted, it is dammed back, collects, and dilates the spaces in the deeper layers of the corium. When the collection of œdematous fluid becomes excessive the epidermis is also affected, and a vesicle is formed by the fluid collecting either immediately below the epidermis or between the epidermal cells.

Prognosis.—Urticaria is never in itself dangerous to life unless where the mucous membranes are seriously involved, but it may sometimes be an indication, especially in elderly people, of serious underlying organic trouble, such as cancer. In acute urticaria the prognosis is, as a rule, excellent, for when the determining cause has been eliminated the urticaria quickly disappears.

In the chronic type, and in the papular urticaria of infants, the prognosis should be more guarded, as it may take several months, or even years, before the disease is got rid of. The prognosis in these chronic cases largely depends on the recognition of the underlying cause, and in those cases which seem to baffle treatment the difficulty has generally been to discover the cause. In infantile urticaria the ultimate prognosis is generally good, though for several years fresh outbreaks may occur from trivial causes. The disease is frequently worse in hot summer weather, and may disappear entirely during the winter.

Diagnosis.—There is, as a rule, no difficulty in the diagnosis of urticaria, the characters of the wheals being so peculiar as to prevent mistakes. In the giant-type and in the œdematous form affecting the loose skin about the eyelids and genitals, some difficulty may be experienced in distinguishing it from erythema nodosum or gummata. Where it is present in the mucosæ, and occasions symptoms of suffocation or gastric disturbance, it may be difficult to recognise, but the diagnosis can generally be made by the discovery of ordinary urticarial wheals on the skin. The diagnosis of papular urticaria in infants is sometimes far from easy on account of the scratch lesions, excoriations, and pyogenic infections which complicate the eruption. It has to be differentiated from pediculosis, scabies, and, where vesicles are present, from varicella. From scabies it is distinguished by the absence of the acarus and its characteristic burrows, and from pediculosis by the absence of the small hæmorrhagic puncta surrounded by an erythematous halo which are peculiar to pediculosis vestimentorum. In varicella, feverishness or other general symptoms are usually present for twenty-four hours before the eruption comes out; these are rare in urticaria papulosa; and in varicella the lesions generally come out first on the trunk, forehead, or face; while in urticaria vesiculosa they are most common on the hands and feet, and ordinary urticarial lesions, or those peculiar to lichen urticatus can, as a rule, be detected elsewhere on the skin.

Treatment.—The first essential for the successful treatment of any of the forms of urticaria is to discover and remove the cause of it. Mere empirical formulæ for internal or external remedies are only palliative so long as the causal factor remains. The treatment of urticaria thus varies so greatly in individual cases that it will only be possible to indicate a few of the general lines which should guide the physician in attacking this troublesome condition. For convenience of description the treatment of urticaria may be divided into the internal treatment and the external or local treatment.

Internal Treatment.—In acute urticaria, due to the taking of some drug, such as copaiba or iodide of potassium, the offending drug must be completely stopped, and not simply reduced in dosage. If it should result from a toxin derived from some article of diet, the latter should be eliminated as rapidly as possible. If the patient be seen soon after the eruption has made its appearance an emetic of mustard, zinc sulphate, copper sulphate, or apomorphine may be ordered; if seen later, saline purgatives, such as sulphate of magnesium or sodium, and diuretics are indicated. When the attack of urticaria occurs in association with malaria, quinine should be employed. In the more chronic types of urticaria which are so often connected with gastro-intestinal derangements, it is the latter which most urgently call for treatment. Where chronic gastric catarrh and dyspepsia are present the indigestion should be treated on general principles. The diet must be most carefully regulated. The patient is usually the best judge of what he is capable of digesting. Rich food, pastry, shell-fish, potatoes, and strong tea should all be avoided. Beer and sweet wines must be eschewed, and if alcohol be taken at all it had best be in the form of well-diluted spirits or good claret. Hot water may be drunk with advantage the first thing in the morning and before going to bed. The bowels must also be carefully looked after, as the slightest constipation and irregularity may determine a fresh efflorescence of wheals. A sedentary life should be as far as possible avoided, and exercise strongly advocated. The

usual gastric sedatives, antiseptics, carminatives, and acid and alkaline tonics, such as bismuth, salol, bicarbonate of soda, dilute nitro-hydrochloric acid, strychnine, and pepsin, should be used where specially indicated.

In cases where the kidneys are acting sluggishly diuretics may prove to be of the greatest value, such as acetate of potash, citrate of potash, and the various alkaline mineral waters such as Vichy, Ems, and Bilin.

Where urticaria complicates chronic rheumatism or gout, salicylate of soda or colchicum will generally give relief, and where it occurs in association with biliary colic or jaundice, the cholagogues, such as podophyllin, rhubarb, and calomel may be resorted to.

Neurotic patients seem to be specially prone to urticaria, and in some cases it is the patient rather than the disease which requires treatment. Nerve tonics, such as strychnine and quinine, may be valuable, but, as a rule, more benefit is obtained from nerve sedatives. The latter diminish the irritation and itching, and, what is of most importance in many such cases, ensure a night's rest; of these bromide of potassium, phenacetin, and antipyrine have all been recommended. Atropine, either in the form of the tincture of belladonna in pill, or in small hypodermic injections ($\frac{1}{150}$ grain) at night is frequently serviceable, and in the worst cases a hypodermic of morphia may be given. Ichthyol has also been extensively used. It may be prescribed in capsules, 5 ♏ three times a day, or with equal parts of glycerine. Where these measures prove of no avail, the patient is often benefited by a change of air and scenery, rest from some irksome occupation, and the daily regime and amusements of some suitable health resort.

In infantile urticaria the diet must be regulated with the utmost care. It is generally advisable to begin the treatment by administering a saline mixture, and if there be any signs of chronic intestinal catarrh these must be treated on general principles. Intestinal antiseptics, such as salol or small doses of dilute lactic acid, are recommended, and where diarrhœa exists "grey powder" may be prescribed. In children the possibility of the presence of worms should never be lost sight of, and if detected the usual anthelmintics should be ordered. The subjective symptoms are frequently so distressing in infants and children that sleep is interfered with, and the child tosses about, becomes feverish, and by rubbing and scratching aggravates the condition of the skin. In such cases a few grains of chloral at night insures sleep, is easily tolerated, and greatly benefits the patient.

External Treatment.—In all cases of urticaria local treatment is demanded to alleviate the distressing subjective symptoms. In urticaria directly produced it is all that is necessary, but in reflex or indirect urticaria local treatment alone is incapable of curing the affection.

The treatment may be in the form of sponging with various antipruritic lotions, or of sedative baths. The most serviceable lotions are those containing carbolic acid, dilute hydrocyanic acid, subacetate of lead, salicylic acid, tar, turpentine, or camphor, and evaporating lotions of chloroform or rectified spirit. In mild cases the time-honoured calamine lotion, with the addition of 1 per cent of carbolic acid, gives great relief (Calaminæ preparatæ ♏xxx., acidi borici ♏x., zinci oxidi ♏xv., glycerini ♏xx., aquam ad. ℥j.); or the following lead lotion may be substituted, Liq. plumbi subacetatis ♏xv., glycerini ♏xx., aquam ad ℥j. Sponging with eau de Cologne or "toilet vinegar" is soothing. In severe cases 2 per cent solutions of carbolic acid in camphor water, or, still better, a 3 per cent solution of the liq. carbonis detergens in water often gives relief.

As a rule ointments and oily substances generally rather aggravate the condition than improve it, but in some chronic cases ointments containing 10 grains of menthol or camphor, or a drachm of chloral to the ounce of soft white paraffin, are beneficial.

In many cases baths seem to relieve the symptoms better than any other form of local treatment. Tepid baths generally give the best results. Alkaline baths containing six ounces of potassium or sodium bicarbonate to thirty gallons of water are particularly soothing. But acid baths with half an ounce of nitric acid, or containing six ounces of borax, or 2 lbs. of starch, bran, oatmeal, or gelatin may be substituted. A thin solution of starch about the thickness of syrup, with the addition of a small quantity of glycerine of borax, has been strongly recommended. After coming out of the bath the skin should be gently but thoroughly dried and dusted with a non-irritating dusting powder such as fuller's earth or starch.

It is necessary to have a large number of local remedies to choose from, because different cases react so differently to local treatment.

Where these remedies all fail galvanism may be tried, and it has been known to prove of benefit. The negative pole is applied at the lower end of the spine and the positive pole at the upper.

Besides these local measures the skin should be protected by light warm clothes, irritating underclothing must be dispensed with, and the bed-clothes should be light and the rooms kept cool. Scratching and rubbing should be avoided, but it is useless to tell the patient so unless it can be made possible for him to resist. This is almost impossible in the case of children, but the effects may be rendered less harmful by the wearing of woollen gloves, or by binding on small cardboard splints on the flexor aspects of the arms to keep them fully extended, and in this way to prevent the child from reaching

the face or the trunk. Strapping the child down to its crib is not a recommendable measure, as it only tends to intensify the irritation and restlessness.

Uskow's Theory.—The theory of the Russian school regarding the development and origin of leucocytes; almost all the varieties of leucocyte are developmental stages, the first being the small lymphocyte (originating in the lymph glands, bone marrow, and spleen), the second the large lymphocyte, the third the large mononuclear lymphocyte, the fourth the transitional form, the fifth the polynuclear neutrophile or mature cell, and the sixth and final form being the eosinophile leucocyte or over-ripe cell.

Ustilago Segetum.—A parasitic fungus (smut) which makes flour and meal mouldy.

Ustion.—To cauterise (Lat. *uro*, I burn).

Uteralgia.—Pain in the uterus; hysteralgia (*q.v.*).

Uterine.—Belonging or relating to the uterus; *e.g.* the *uterine appendages* or *annexa* (Fallopian tubes and ovaries), *uterine-fibroids* (myomata growing in the uterus), *uterine prolapse* (downward displacement of the uterus), etc. *See* UTERUS.

Utero-.—In compound words *utero-* (Lat. *uterus*, the womb) means relating to the uterus, e.g. *utero-abdominal* (belonging to the uterus and the abdomen), *uterofixation* (the suturing of the uterus in cases of displacement, or hysteropexy), *utero-gestation* (pregnancy inside the uterus as distinguished from tubal pregnancy), *utero-sacral* (related to the uterus and sacrum, *e.g.* the utero-sacral ligaments), *utero-vesical* (relating to the uterus and bladder, *e.g.* the utero-vesical space between the uterus and the bladder), etc.

Uterus.

I. Malformations of the Vulva, Vagina, and Uterus . . . 319
II. Displacements of the Uterus . 327
III. Inflammations of the Uterus and Cervix Uteri 340
IV. Benign Tumours of Uterus . . 348
V. Malignant Tumours of the Uterus 363
VI. Uterine and Ovarian States, Insanity associated with . . 376

See also ABDOMINAL TUMOURS, DIAGNOSIS OF (*Uterus*); ASTHMA (*Cause of Attack*); AUDITORY NERVE AND LABYRINTH (*Nerve Deafness, Toxic*); BROAD LIGAMENTS; CURETTAGE, UTERINE; ECLAMPSIA; ECTOPIC GESTATION; ELECTRICITY (*In Gynæcology*); EMBRYOLOGY; FALLOPIAN TUBES; GENERATION, FEMALE ORGANS OF (*Uterus*); GENERATION, FEMALE ORGANS, ARRESTED DEVELOPMENT OF (*Uterus*); GYNÆCOLOGY, DIAGNOSIS IN (*Uterus*); HEADACHE (*Causes, Reproductive System*); HYDROPATHY (*In Uterine Diseases*); HYPNOTISM (*Effect on Uterine Contractions*); HYSTERIA; LABOUR, PHYSIOLOGY OF; LABOUR, STAGES AND DURATION; LABOUR, DIAGNOSIS AND MECHANISM; LABOUR, MANAGEMENT OF; LABOUR IN MULTIPLE PREGNANCY; LABOUR, PRECIPITATE AND PROLONGED; LABOUR, FAULTS IN THE PASSENGER; LABOUR, ACCIDENTAL COMPLICATIONS; LABOUR, RETAINED PLACENTA; LABOUR, POST-PARTUM HÆMORRHAGE; LABOUR, INJURIES TO THE GENERATIVE ORGANS; LABOUR, OBSTETRIC OPERATIONS (*Forceps, Version, etc.*); LUNGS, VASCULAR DISORDERS (*Embolism, Causes, Puerperium*); MALINGERING (*Genito-Urinary*); MENOPAUSE; MENSTRUATION AND ITS DISORDERS; MICTURITION (*Retention due to Retroversion of the Gravid Uterus*); OVARIES, DISEASES OF (*Tumours, Diagnosis*); PELVIS, PERINEUM AND PELVIC FLOOR (*Lesions, Prolapsus Uteri*); PELVIS, DISEASES OF CELLULAR TISSUE AND PERITONEUM; PELVIS, HÆMATOCELE AND HÆMATOMA; POST-MORTEM METHODS (*Cavities, Abdomen and Pelvis*); PREGNANCY, CHANGES IN MATERNAL ORGANISM; PREGNANCY, DIAGNOSIS OF; PREGNANCY, MULTIPLE; PREGNANCY, MANAGEMENT OF; PREGNANCY, PATHOLOGY OF; PREGNANCY, OVUM AND DECIDUA; PREGNANCY, DISEASES OF PLACENTA AND CORD; PREGNANCY, AFFECTIONS AND ACCIDENTAL COMPLICATIONS; PREGNANCY, HÆMORRHAGE IN; PUBERTY (*Changes in Uterus*); PUERPERIUM, PHYSIOLOGY OF; PUERPERIUM, PATHOLOGY OF (*Puerperal Infection, Deciduoma Malignum*); PURPURA (*Symptomatic*); SPLEEN, SURGERY OF (*Wandering Spleen, Diagnosis*); STERILITY (*Causes*); URACHUS (*Fistula*); VAGINA; VENEREAL DISEASE; VISCERAL PAIN.

I. Malformations of the Vulva, Vagina, and Uterus

This subject will be considered under the following headings:—

A. DEVELOPMENT OF THE FEMALE GENITAL ORGANS 320
B. MALFORMATIONS OF THE FEMALE GENITAL ORGANS 321
I. *Malformations by Arrest of Development—*
(*a*) *Total Arrest, or Absence of Organs* 321
(*b*) *Partial Arrest, or Underdevelopment* 321
II. *Malformations by Persistence of Septa* 322
(*a*) *Persistence of Longitudinal Septa—*
Double Uterus and Vagina . 322
(*b*) *Persistence of Transverse Septa—*
Atresia of the Genital Passages 323
(*c*) *Persistence of Longitudinal and Transverse Septa—*
Unilateral Atresia . . . 324

III. *Malformations by Excess of Development*—
Pseudo-hermaphrodism . . 324
Accessory Organs or Ostia . 324
Defects of the Hymen . . 325
C. Clinical Aspects of Malformations of the Female Genital Organs—
1. *Symptomatology* 325
2. *Diagnosis* 327
3. *Treatment* 327

In order to understand the malformations to which the female generative organs are liable, some knowledge of the broad outlines of the development of these organs is necessary.

A. Development of the Genito-Urinary Organs.—Up to the end of the eighth week of fœtal life there is no differentiation of sex (Fig. 1, I.) On each side of the median line is found a Mullerian duct (*m*), a Wolffian duct (*w*), and a ureter (*u*). The allantois (*g*), destined to become the bladder, communicates with the rectum (*h*); there is no communication with the outside, but opposite the end of the hind-gut is a depression (*b*), the urogenital sinus. In front of this depression is a prominence (*a*), the genital eminence, prolonged backwards on each side of the urogenital sinus, and destined to form the external organs. In close relation to the Wolffian duct is a thickening of peritoneum, from which the genital gland (ovary or testis, as the case may be) will be formed.

From the eighth to the twelfth week (II. and III.) important changes occur. The Müllerian ducts coalesce from below upwards (*m*), forming the vagina (which opens below into the urogenital sinus) and the uterus; for some time the uterine horns remain distinct, the demarcation between each horn and its corresponding Fallopian tube being indicated by the insertion of the round ligament (*r*). The Wolffian ducts (*w*) atrophy and finally disappear, except on some vestigial traces still found in the adult (*see* Table on p. 321). The communication between the hind-gut and the outside is effected, forming a cloaca (*c*), which by the downgrowth of the perineum (*p*) becomes divided into urogenital opening and anus.

From the twelfth to the twentieth week the female characters become more marked (IV.). The uterine horns become fused to form an undivided uterus; the cervix with its arbor vitæ is indicated; the urethral and vaginal orifices become separated (*d* and *e*); the perineum (*p*) enlarges, separating the anus (*f*) farther from the vaginal orifice. The genital eminence (*a*) becomes relatively smaller, forming the clitoris; the folds which pass backwards from it, bounding the urogenital sinus, become the labia minora, and within the vestibule between them the hymen forms round the vaginal orifice. From the twentieth week to the end of fœtal life the organs already formed become better developed; the fundus of the uterus

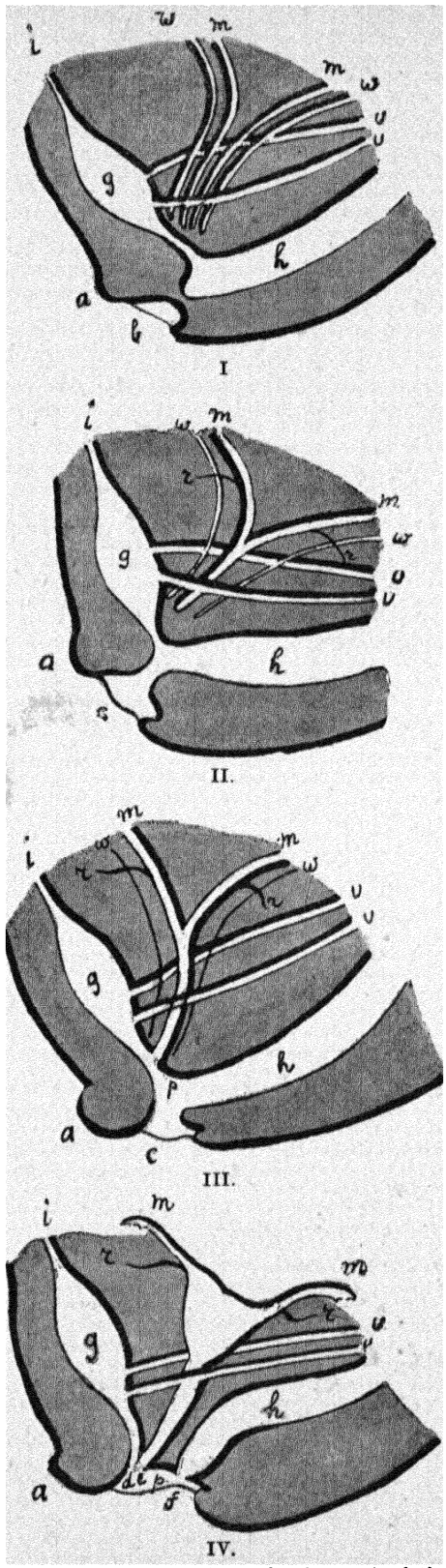


Fig. 1.—Diagram of the mode of development of the female genital passages. *a*, Genital eminence; *b*, urogenital sinus, forming, *c*, cloaca, and subsequently, *d*, urethral orifice, *e*, vaginal orifice, *f*, anus; *g*, bladder; *h*, rectum; *i*, urachus; *m*, Müllerian ducts, forming Fallopian tubes, uterus, and vagina; *p*, perineum; *r*, round ligament; *u*, ureters, *w*, Wolffian ducts.

increases in size, though it does not keep pace with the size of the cervix; folds form in the

vagina and cervix, and the labia majora become fuller and more rounded.

From birth to puberty the body of the uterus gradually enlarges till it becomes larger than the cervix, and the vulva increases in size.

It would take us beyond the scope of our subject to trace in detail the development of the genital organs in the male; but it is important to remember that they arise from the same primitive structures as are found in the female; and this fact explains the phenomena of pseudo-hermaphrodism. The relation of the adult organs in the male and female, and their common derivation from the same fœtal structures, are indicated in the following table, the parts shown in italics being rudimentary or vestigial in the adult:—

A Table of Homologous Parts in the Male and Female.

Adult Male.	*Fœtal Structures.*	*Adult Female.*
Body of testis	Genital gland	Oöphoron
Paradidymis Vasa efferentia	Wolffian body	Paroöphoron *Parovarium (epoöphoron)*
Vas deferens	Wolffian duct	*Duct of Gartner*
... ... *Sinus pocularis*	Müllerian duct	Fallopian tube Uterus Vagina
Corpora cavernosa (penis) Corpus spongiosum (penis)	Genital eminence and folds prolonged backwards from it	Corpora cavernosa (clitoridis) Glans clitoridis and vestibular bulbs.
Prostatic urethra Membranous urethra Folds at entrance to sinus pocularis Cowper's glands	Urogenital sinus	Urethra Vestibule Hymen Bartholin's glands
Scrotum	Outer portion of genital folds	Labia majora

B. Malformations.—From a consideration of the facts of development, as we have briefly outlined them, it will be readily understood that malformations may occur in three ways:—

I. Arrest of development, partial or total, of individual portions.

II. Persistence of septa, longitudinal or transverse, which should normally disappear.

III. Excess of development of individual portions.

We shall describe the malformations of the genital organs in the order indicated.

I. Arrest of Development.

(*a*) *Total Arrest, or Suppression.—Absence of the genital organs* is found only in the case of gross monstrosities, and is therefore very rare.

Absence of the vulva is also a very rare condition. The labia minora or majora may be absent; but even then some trace of the clitoris is nearly always found. The internal organs in such cases are generally under-developed.

Absence of the vagina sometimes occurs. The interval between cervix and vulva is then occupied by a fibrous thickening or cord-like band, which is all that remains of the vagina. The urethra comes to lie nearer the anterior rectal wall than usual.

Absence of the uterus is said to occur even when a vagina is present; but this is very doubtful, as some trace of the uterus, however small, is usually found on dissection. Clinically it is very difficult to determine whether the uterus is absent or not. The same remarks apply to absence of one or both of the ovaries and tubes.

Absence of the anterior or posterior wall of the urethra may occur, producing the conditions known respectively as epispadias and hypospadias.

(*b*) *Partial Arrest of Development, or Under-development.—Under-development of the whole genital organs* is not very infrequent; and all grades are found, corresponding with arrest of growth at various stages from birth to puberty.

Under-development of the Vulva.—Individual portions may be concerned, such as the clitoris, labia minora, or labia majora. In the condition known as *exstrophy of the bladder*, in which the abdominal wall from the umbilicus to the urogenital sinus (*i* to *b*, Fig. 1, I.) fails to unite, the clitoris is usually bifid and rudimentary, and the labia minora are also small. Or the whole vulva may be small and infantile.

Partial Absence of the Vagina.—The lower or middle part of the vagina is sometimes absent, whilst the upper portion is patent. The deficient part is represented by a fibrous cord. This condition is referred to more fully later in discussing atresia of the genital passages.

Under-development of the Uterus.—This may be present in one of two degrees:—

Rudimentary Uterus.—The uterus may be represented only by a little knob or button surmounting the vagina; or the cervix may be fairly well marked, whilst the body of the uterus is very narrow, and the Fallopian tubes meet at the fundus. Menstruation is absent; and the

secondary sexual characters, such as the breasts and pubic hair, are usually poorly developed. The patient may show general arrest of development, the woman of twenty-five presenting the physical and mental characteristics of a child.

Infantile Uterus.—In this case the body of the uterus preserves the characteristics of childhood; it is small, with an ill-developed fundus; and the cervix may be two or three times the length of the body. Menstruation seldom occurs; or it comes on very late and is scanty and irregular. The vulva, vagina, pubes, and breasts may share in the retardation; but on the other hand they may not differ from the normal adult condition. Sexual desire is usually absent.

Uterus Unicornis.—If one-half of the uterus develops normally whilst the other retains its fœtal condition, the condition known as uterus unicornis or one-horned uterus results (Fig. 2). The round ligament marks the limit between the rudimentary horn and the Fallopian tube. Both tubes and ovaries may be normal; but more often the tube and ovary remain rudimentary on the side of the undeveloped horn. The condition may give rise to no symptoms and remain undiscovered, the normal half of the uterus carrying on the functions of menstruation and gestation. Pregnancy may, however, occur in the rudimentary horn, giving clinically the symptoms and signs of a tubal gestation.

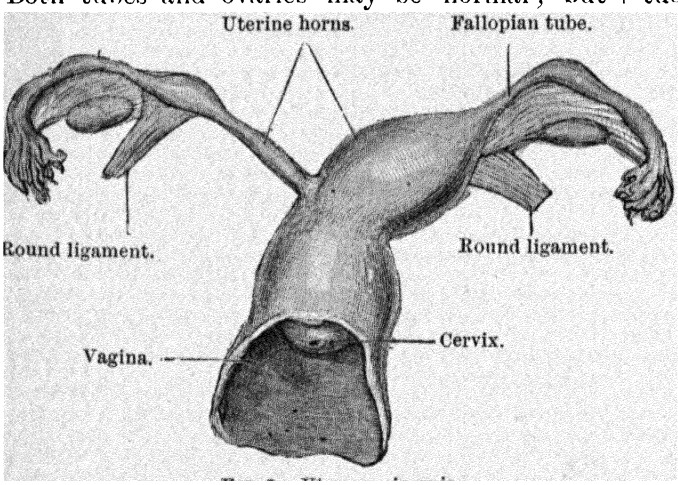


FIG. 2.—Uterus unicornis.

Rudimentary Fallopian Tubes.—One or both tubes may remain under-developed; sometimes this is the result of fœtal peritonitis. In rare cases, of which we have seen an example, the tubes become occluded along their course and divided; the fimbriated end shrivels up, and the remainder of the tube comes to resemble closely a vermiform appendix. In other cases the fimbriæ remain well developed, whilst the portion of tube nearer to the uterus atrophies to a cord-like structure.

Rudimentary ovaries usually present the long, narrow shape characteristic of fœtal life; and this form is seen not infrequently in cases of undescended ovary and of congenital hernia of the ovary. In other cases the ovary has the normal shape, but is small. Menstruation and sexual desire may be absent; and in any case sterility results.

As the result of arrest in the development of the cloaca and urogenital sinus several rare malformations occur. Thus the anus may be imperforate terminally, the rectum opening into the posterior wall of the vagina; conversely the vagina may be imperforate below, but open into the anterior rectal wall. The urethra may be absent, the bladder opening into the anterior vaginal wall. There is an example in the museum of the London Hospital of rectum and bladder both opening into the vagina, the orifice of which thus served as a general cloaca. There was apparently no suspicion of the state of things till the patient was ordered an enema in the hospital.

II. MALFORMATIONS DUE TO THE PERSISTENCE OF SEPTA.—A persistent septum may be longitudinal or transverse, or there may be a combination of both conditions; and three types of malformation are thereby produced:—

(*a*) Double uterus and vagina, from failure of coalescence of the two Mullerian ducts.

(*b*) Atresia of the genital canal, from failure on the part of the coalesced Müllerian ducts to open into the urogenital sinus.

(*c*) Doubling of the uterus and vagina with atresia in one-half, that is, unilateral atresia.

(*a*) *Double Uterus and Vagina.*—Inasmuch as the fusion of the Müllerian ducts occurs from below upwards, it is not uncommon to find a double uterus with one vagina, or a double fundus with a common cervix and vagina; but the reverse condition, that is to say, a double vagina with undivided uterus, or double cervix with undivided fundus, is never found.

Double Vagina.—This condition occurs in some cases of double uterus. The septum between the two halves of the vagina may extend to the vulva, when the appearance of the vaginal orifices somewhat resembles a double-barrelled gun. Or the orifice may be single, and the septum is discovered only on making a digital examination. The septum itself may be quite complete; or it may be perforated in parts; and in some cases it is incomplete, and represented by longitudinal ridges on the anterior and posterior vaginal walls.

Double Uterus.—Three types are found: the uterus septus, the uterus bicornis, and the uterus didelphys. In the first two the cervix may be single or double, but the uterus didelphys has necessarily two cervices.

Uterus Septus (Fig. 3, 1 *a* and 1 *b*).—Here the Mullerian ducts have fused externally, so that when seen from the outside the uterus appears normal save for some broadening of

the fundus. But on section a partition is seen extending from the fundus to the os externum or even to the vaginal orifice (Fig. 3, 1 *a*).

In the variety known as *uterus subseptus* (Fig. 3, 1 *b*) the septum reaches only to the os internum, so that the body of the uterus is double, whilst the cervix and vagina are single.

Uterus bicornis (Fig. 3).—In this case external union takes place in the lower part of the uterine body and cervix, but is wanting at the fundus, so that externally this portion of

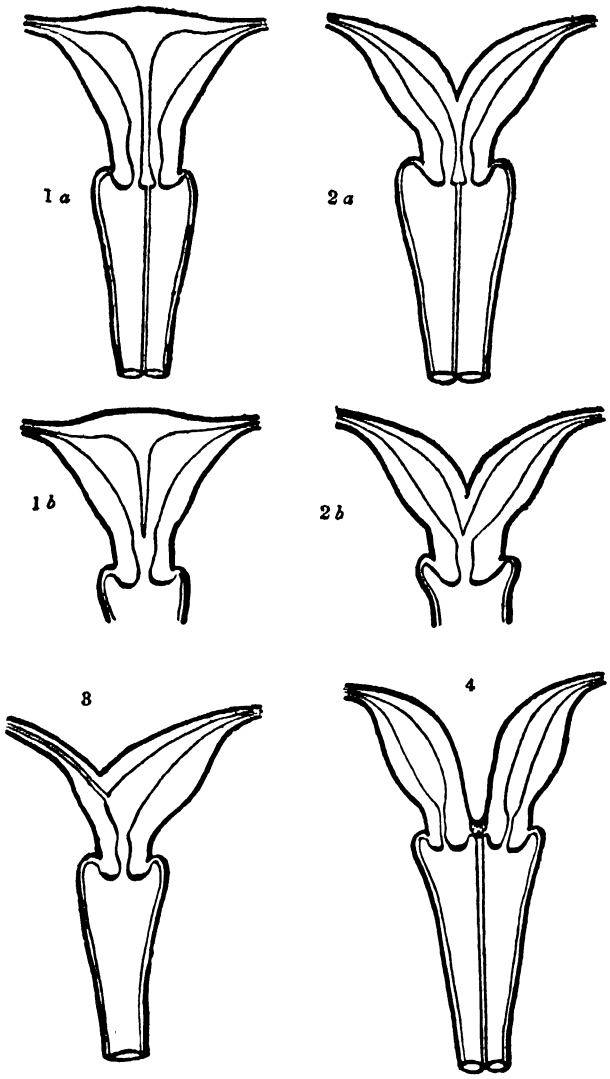


FIG. 3.—1 *a*, Uterus septus; 2 *a*, uterus bicornis; 1 *b*, uterus subseptus; 2 *b*, uterus bicornis unicollis; 3, uterus unicornis; 4, uterus didelphys.

the uterus appears as two horns. The septum extends throughout the cervix, which is also double, and may reach the vaginal orifice (Fig. 3, 2 *a*). The demarcation between uterine horn and Fallopian tube is indicated by the insertion of the round ligament.

Uterus Bicornis Unicollis.—This is a variety of the preceding type, and cannot be distinguished from it externally. The septum reaches only to the os internum, so that the cervix and vagina are normal (Fig. 3, 2 *b*). A vaginal examination will, of course, enable one to distinguish between a uterus bicornis and a uterus bicornis unicollis, or between a uterus septus and a uterus subseptus.

Uterus unicornis, already referred to, is of course also a variety of the uterus bicornis, in which one horn has remained undeveloped (Fig. 3 (3)). It is therefore a combination of the two main types of malformation which we have indicated, namely, partial arrest of development with persistence of a septum.

Uterus Didelphys (Figs. 3 (4) and 4).—In this form the two halves of the uterus remain quite distinct, and can be moved independently of one another. The vagina is always double, the two halves being united by connective tissue, and a loose bridge of connective tissue usually extends between the two cervices. A well-marked fold of peritoneum passes between the two uteri from the back of the bladder to the front of the rectum. Each half of the uterus has its own Fallopian tube and ovary, and the round ligament shows where uterus ends and tube begins. One-half of the uterus may be considerably displaced, so that it comes to lie in front of the other.

(*b*) *Atresia of the Genital Canal.*—Congenital atresia only affects the vagina. Clinically atresia of the os externum or internum is also found, but it is secondary. We shall, however, consider all kinds of atresia when discussing the clinical significance of malformations.

Vaginal atresia is of various grades. In the simplest form a thin septum is present, occluding the vaginal orifice. This was formerly called atresia of the hymen; but the septum is vaginal, and on its external surface the hymen is found adherent. For it must be remembered that the hymen is not a complete diaphragm at any stage of its development; it arises as a membranous fold from the margins of the urogenital sinus.

The vagina may altogether fail to reach the surface, so that its lower third, or half, or even the whole of it may be absent. There is nearly always, however, some portion of it unoccluded just below the cervix. When the lower two-thirds are wanting, the apex of the upper patent portion is connected with the vestibule by a fibrous cord; and this may fail to elongate as the pelvis grows, with the result that the vestibule is drawn in so as to form a *cul-de-sac* resembling the lower part of the vagina. It is very doubtful if a portion of true vagina is ever present externally when the middle or upper part is wanting; in other words, it is doubtful if the Müllerian duct persists at its lowest part and opens into the vestibule whilst a portion higher up is suppressed.

Atresia of the Fallopian tubes has been referred to; it is not a true congenital (*i.e.* developmental) atresia, since it is the result of disease in the fœtus.

In all forms of persistent septa, whether longitudinal or transverse, the patient is otherwise

well developed as a general rule. Longitudinal septa are compatible with the performance of genital functions, menstruation, conception, and pregnancy. Atresia is a bar to conception; but menstruation may occur, although its products are necessarily retained.

(c) *Unilateral Atresia.*—In cases of double uterus and vagina coalescence may fail to take place not only longitudinally, but also terminally, so that one vagina may run down by the side of the other as a blind sac. Hence, while one-half of the uterus and vagina may appear, clinically, to be normal, retention of menstrual products may occur in the other half. Similarly in cases of double uterus with a single vagina, one-half of the uterus may have no opening into the vagina; and if this half of the uterus menstruates, as usually occurs, its products will be retained.

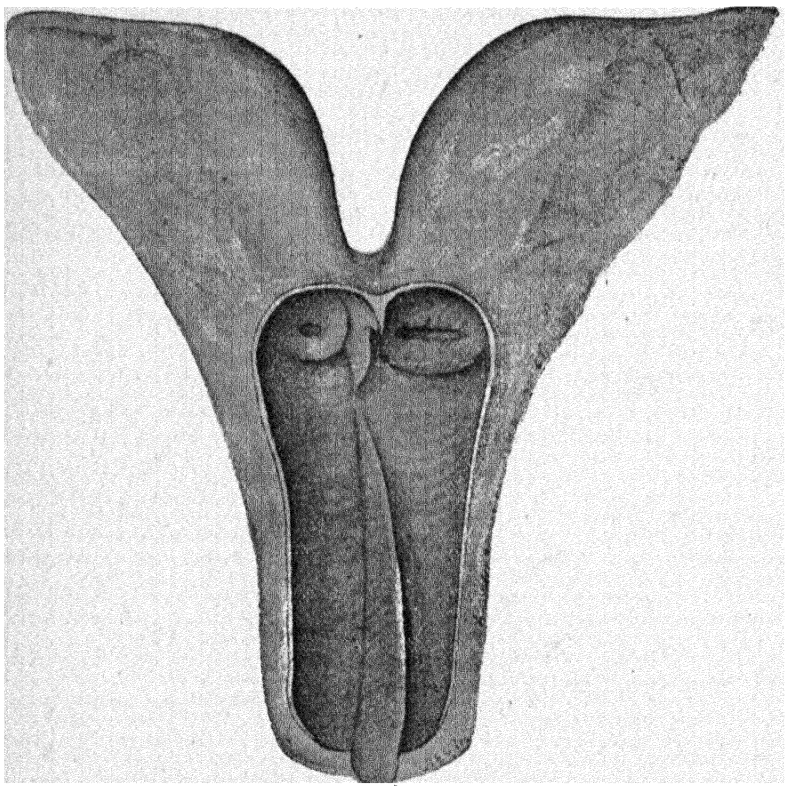


FIG. 4.—Uterus didelphys.

III. MALFORMATIONS BY EXCESS OF DEVELOPMENT.—*Hermaphrodism* means the development of both male and female generative organs in the same subject, and occurs in some of the lower animals. The presence of both ovary and testis is necessary in order that a case may be regarded as one of true hermaphrodism, and this condition has not been demonstated in the human species.

Pseudo-hermaphrodism means the development in the same subject of male and female accessory genital glands, including in this term everything except ovary and testis. Pseudo-hermaphrodism is found in the human species, and is of two principal types:—

Gynandria, in which the subject is a female and possesses ovaries, but has also accessory organs resembling the penis and scrotum of the male.

Androgyna, in which the subject is a male and possesses testicles, but has also the accessory organs of a female, such as vagina, uterus, and Fallopian tubes. The penis may be undeveloped, resembling a clitoris; and the two halves of the scrotum may fail to unite, resembling labia majora. It is often very difficult, and in some cases impossible, to say whether an individual is male or female; for nothing but a microscopical examination of the genital gland, to determine whether it is an ovary or a testis, will decide the point. As a matter of fact, most cases of pseudo-hermaphrodism are really males, and these we are not here concerned with.

In gynandra the clitoris is large, like a small penis, and the labia majora fuse in the middle line, resembling the scrotum. The resemblance is all the more striking if the ovaries are herniated into the labia majora. In such a case the vagina, which is generally small, opens into the urethra at the root of the clitoris like a hypospadiac male; the uterus may be fairly well developed. A typical case of this malformation was the now historical one of Madeline le Fort.

Hypertrophy of the clitoris may occur independently of pseudo-hermaphrodism, and it may be difficult to distinguish this condition from a benign new growth.

Hypertrophy of the Labia Minora.—These are minor degrees of enlargement which are in no sense congenital, and which are due in some cases to masturbation. Elongation may, however, be excessive, as is typically seen in the Bush-women of South Africa, giving rise to the condition known as the Hottentot apron.

Congenital elongation of the cervix affects the vaginal portion, which may attain a length of 2 to 3 inches, and project through the vulvar orifice. This condition is seldom observed till puberty, so that it is difficult to say whether it is always present from the time of birth, or whether it is incidental to the general enlargement of the pelvic organs that takes place at puberty.

Accessory Ostia Tubæ.—The Fallopian tube is sometimes found with two or even three fimbriated openings. The condition has no clinical importance.

A third or accessory ovary has been described, and some writers state that this is a common occurrence. "A careful consideration of the evidence makes it clear that small pedunculated

bodies near the ovary are very frequent, but they are not accessory ovaries. Many of them are partially detached tubes of the parovarium, stalked corpora fibrosa, or small myomata of the ovarian ligament. So far as the facts stand at present, a supernumerary ovary, so separated from the main gland as to form a distinct ovary, has yet to be described by a competent observer" (Bland-Sutton and Giles).

Defects of the Hymen.—The hymen is subject to variations in shape and in structure. The orifice is normally crescentic, concave anteriorly; instead of this the aperture may be central and circular, or it may be divided so that two apertures exist, either one in front of the other or side by side. In the latter condition the appearance closely resembles that of double vagina, from which it must be distinguished. The *cribriform hymen* presents a number of openings.

In structure the hymen may be distensible and yielding, allowing coitus to occur without rupture; or it may be dense and unyielding, requiring division before coitus can take place. In rare cases the hymen has persisted during pregnancy, and formed an obstacle to labour.

C. Clinical Aspects of Malformations of the Female Genital Organs.—1. *Symptomatology.*—It will be convenient to consider here the various malformations in relation to the general bodily and mental development, to the secondary sexual character, to menstruation, and to child-bearing.

Absence of the genital organs is only found, as we have said, in the case of monstrosities. With marked under-development of the uterus and ovaries—for instance, with rudimentary uterus or infantile ovaries—there are often found retarded physical growth and imperfect mental powers. Slight degrees of under-development are compatible with an otherwise well-formed body and a vigorous mind. The same may be said of malformations in the second and third groups, namely, doubling and atresia of the genital passages and hypertrophy of vulva or cervix. The development of the secondary sexual characters maintains a fairly close parallel with that of the pelvic organs, so that when the latter are imperfectly formed we find the breasts flat, small, and even absent, the pubic hair scanty or absent, as in the case of a child, and the vulva small.

Menstruation is established late or not at all when the uterus is under-developed. A woman with a rudimentary or infantile uterus, or with imperfect ovaries, does not menstruate. Broadly speaking, the occurrence or otherwise of menstruation gives a fairly reliable indication of the degree of development, whilst its characters, where it does occur, also have their significance. If menstruation is normal it may be presumed that there is not any considerable arrest of growth; if it is very scanty and irregular, and if it comes on late, it can generally be assumed that some degree of under-development exists.

In the case of double uterus menstruation has nearly always the normal characters, and even if one-half be under-developed, as in the uterus unicornis, the other half is generally functional. In cases of atresia the changes incidental to menstruation occur in the uterus, but the menstrual products are of course retained (*cryptomenorrhœa*). When distension of the vagina comes on symptoms arise, chiefly in the form of abdominal pain, which has at first a monthly exacerbation, but later on becomes more or less constant. In cases of unilateral atresia the symptoms may be misleading, because the retention of products in the occluded half of the vagina and uterus produces the characteristics just described, whilst menstruation is apparently taking place normally on the other side.

Effects of Retention of Menstrual Products in Cases of Atresia.—According to the situation of the obstruction and the duration of cryptomenorrhœa the following conditions may result, shown diagrammatically in Fig. 5:—

(i.) *Atresia of the Vaginal Orifice.*—As first mentioned, blood collects in the vagina, producing the condition known as *hæmatocolpos* (Fig. 5 A). In the course of time distension occurs successively in the cervix—*hæmatotrachelos* (B), in the body of the uterus—*hæmatometra* (C), and in the Fallopian tubes—*hæmatosalpinx* (D).

(ii.) *Absence of the Lower Part of the Vagina.*—The distension proceeds in the same order as above, affecting first the vagina (E), and then the uterus (F). No indication of the distension is, however, seen at the vulva, whilst in the preceding group the vaginal septum bulges down between the labia.

(iii.) *Atresia of the Os Externum.*—Here the vagina remains normal, and the cervix suffers (Fig. 5 G), and then the whole uterus.

(iv.) *Atresia of the Os Internum.*—The cervix and vagina both remain free, and a pure hæmatometra is formed (H). The last two conditions are seldom if ever congenital.

(v.) *Atresia affecting One-half of a Double Uterus or Vagina.*—When one vagina is occluded *lateral hæmatocolpos* at first results, the swelling extending by the side of the normal vagina down to the vulva (I), or only part of the way if the occluded vagina is also partly deficient (J). Hæmatometra follows (K), or it occurs alone if the atresia affects the os internum (L).

If the contents of these various retention-sacs suppurate, either spontaneously or through a temporary fistulous aperture, pyocolpos, pyometra, and pyosalpinx may occur.

Characters of retained Menstrual Blood.—This is a dark chocolate colour, sometimes black. It is viscid, owing to partial absorption of the liquid portion of the blood, and flows slowly, like treacle or honey. It is mixed with

mucus, and seldom contains coagula. Cholesterin crystals are sometimes found. Microscopical examination shows the presence of old and altered epithelial cells and blood corpuscles in various stages of decomposition.

Child-bearing in Relation to Malformations.—When the pelvic organs are under-developed sexual desire and feeling are frequently absent or only slightly present. Desire probably depends more upon the development of the ovaries than upon any other individual factor; but sexual feeling during intercourse will also vary according to the degree of development of the vagina and vulva. Child-bearing does not, however, depend upon sexual inclination or sensation, but upon the production of healthy ova, the ability of the uterus to nourish an ovum, and the possibility of coitus taking place. Under-development of the ovaries leads to complete and permanent sterility. Under-development of the uterus has the same effect, both because it is usually associated with defective ovulation, and because the uterus is unfit for pregnancy. A woman with a double uterus is very often fertile, and several pregnancies may occur without the malformation being even suspected.

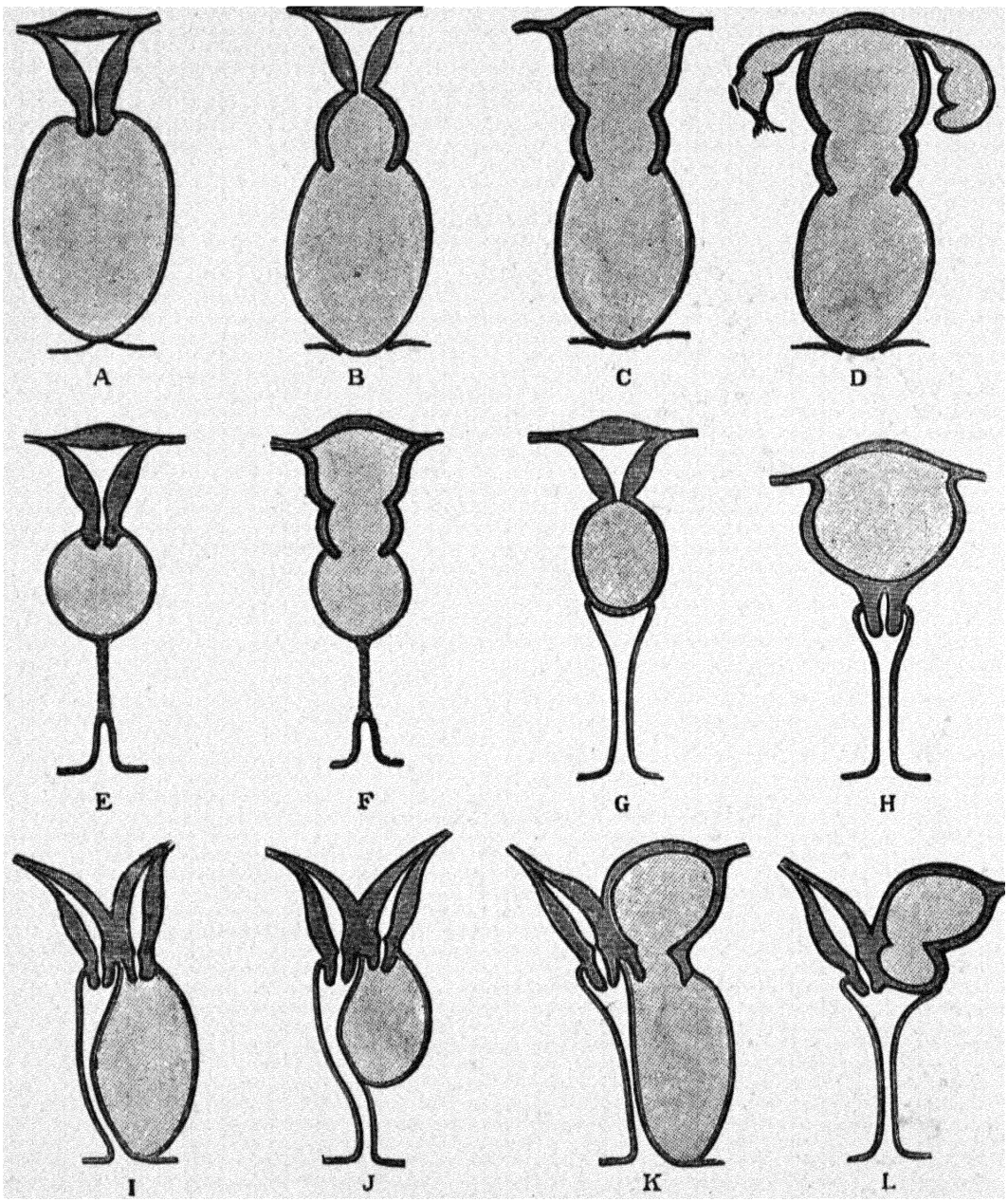


FIG. 5.—The effects of atresia on the genital passage.

Complications with labour may, however, occur. Sexual intercourse is possible as long as the vagina is well enough developed, whatever be the condition of the internal organs: the conditions which prevent coitus are absence, atresia, or stenosis of the vagina. In pseudo-hermaphrodism the vagina is usually small. If a case of atresia of the vagina be treated early coitus can take place, and, theoretically at least, may be followed by conception; but if considerable

changes have taken place owing to distension before the obstruction is removed it is doubtful whether the patient could conceive. We have not met with or heard of a case where a patient who had had atresia of the vagina became pregnant subsequently.

2. *Diagnosis.*—The diagnosis of malformation will depend in the first place on inspection, when any external abnormality will be at once discovered, and the patency or otherwise of the vaginal orifice determined. A digital vaginal examination will show whether the vagina is unduly narrow or short, or whether the cervix is normally developed. Two vaginæ may be thus discovered, each containing a cervix; or two cervices may be found in a single vagina. Bimanual examination will then be necessary to determine the degree of development of the uterus, tubes, and ovaries. This information can also be obtained, though less satisfactorily, by a recto-abdominal examination. The sound will lend assistance in ascertaining the length of the uterus, and may also enable one to demonstrate that the uterus has two cavities. The uterus may be found to be very small or rudimentary, and it may even be impossible to feel it at all bimanually. When the ovaries are small they are often difficult to feel, and it may not be possible to form any conclusion as to their development except by inference. Unilateral atresia often presents great difficulties in diagnosis; a lateral hæmatocolpos will resemble an abscess burrowing down by the side of the vagina, whilst a lateral hæmatometra will probably be mistaken for a broad ligament or ovarian cyst until the condition is ascertained by operation, or on the post-mortem table.

3. *Treatment.*—The scope of treatment for malformations of the female genital organs is necessarily limited. When there is underdevelopment of uterus and ovaries no treatment is of any avail; and in cases of doubling of the uterus and vagina no treatment is necessary, unless it be that the vaginal septum interferes with intercourse or obstructs labour; in which case it should be divided, and, if possible, either a portion or the whole length of it excised. If the clitoris or labia are so enlarged as to cause discomfort, or if the cervix is much elongated, resection of the part is indicated.

The conditions that more particularly call for treatment are different forms of atresia and of absence of the vagina. Thus hæmatocolpos will require a free incision to allow of the evacuation of the retained fluid. The treatment of hæmatometra, with a normal vagina, is equally simple; but special means must be taken to prevent reclosure, as by packing with gauze, and by the use of dilators later on if necessary. Absence or considerable deficiency of the vagina is a more difficult matter to deal with. Treatment may be required either to allow of the outflow of menses or to permit of intercourse. Dissection may have to be carried to some depth between the urethra and rectum before the blind end of the vagina or the cervix is reached; and when this has been done it is a very troublesome matter to keep the channel patent. Various plastic operations, with the turning in of flaps of skin, have been suggested and practised from time to time; in a few cases a successful result has been attained, it may be after repeated operations. In other cases the attempt has had to be abandoned, and it has been found necessary to perform abdominal hysterectomy to prevent the recurring hæmatometra. Lateral atresia requires to be treated on the same principles as the more common forms of atresia; it is important that the septum between the patent and the occluded half of the vagina or uterus should be removed as far as possible to prevent reclosure.

Occluded tubes usually require removal; but in some cases it is possible to restore the patency of a tube by a plastic operation.

Mere stenosis of the vagina or rigidity of the hymen is easily treated by dilatation of the former or incision of the latter. Some care in the after-treatment will probably be required to maintain the normal calibre.

II. Displacements of the Uterus

THE NORMAL MOBILITY 327
NORMAL MOVEMENTS AND POSITIONS . 328
ABNORMAL POSITIONS—
Displacements as a whole . . . 328
Displacements by Version . . 328
,, ,, *Flexion* . . 329
,, ,, *Inversion* . . 329
1. ASCENT 329
2. DESCENT 329
3. PROPLAPSE 329
4. ANTEPOSITION 336
5. RETROPOSITION 336
6. SINISTRO- AND DEXTROPOSITION . . 336
7. PATHOLOGICAL ANTEVERSION AND ANTEFLEXION 337
8. CONGENITAL ANTEFLEXION . . . 338
9. RETROVERSION AND RETROFLEXION . 339

THE NORMAL MOBILITY OF THE UTERUS.—The most rigid attachments of the uterus are those of the supravaginal portion of the cervix. Here the uterus is inserted in the pelvic floor, and is embraced by the pelvic fasciæ. In front it is attached for a distance of 2 cm. to the posterior wall of the bladder; by the utero-vesical ligaments it is attached to the pubes. Behind it is attached to the sacrum by the utero-sacral ligaments, which contain strong muscular bands in their folds. These ligaments converge to form the muscle of Luschka, and are inserted into the posterior aspect of the uterus at a point a little below the junction of body and cervix.

These attachments make the supravaginal

portion of the cervix the part of the uterus which is least movable. Above, the body of the uterus is comparatively free, while below, the vaginal portion is entirely unattached, consequently the upper segment of the cervix uteri becomes the axis of rotation of its antero-posterior movements. It may also be called the point of suspension of the uterus, because if any ligaments are concerned in the support of that organ these are the utero-sacral.

The attachments of the body are loose. The round ligaments are "attached to the upper angles of the uterus on either side immediately in front of the Fallopian tube. From this point each ligament proceeds upwards and forwards to gain the inguinal ring; and after having passed, like the spermatic cord in the male, through the inguinal canal, reaches the fore part of the pubic symphysis, where its fibres expand and become united with the substance of the *mons veneris*. Besides areolar tissue and vessels, the round ligaments contain plain muscular fibres like those of the uterus, from which, indeed, they are prolonged" (Quain). They thus permit of an antero-posterior range practically from pubes to sacrum.

The folds of peritoneum, which form the broad ligaments, pass outwards and backwards to the sides of the pelvis in front of the sacro-iliac joint; they permit the body of the uterus to be moved from one side of the pelvic inlet to the other.

Normal Movements and Positions of the Uterus.—The position which the uterus occupies depends chiefly on the amount of distension of the bladder and rectum for the time being. When the bladder is distended the uterus is pushed into retroposition or retroversion. When the rectum is distended and the bladder is empty the uterus becomes anteposed. When bladder and rectum are both distended, the uterus is elevated. The uterus also moves upwards and downwards with every respiration. These displacements may be considered normal, because they are temporary and due to physiological acts. Strictly speaking, the uterus has no fixed normal position, but the position which it takes up when the bladder and rectum are empty may, for gynæcological purposes, be considered the normal one, as these organs are emptied before an examination is made; then the uterus is anteverted, placed as far forward as the bladder will allow, turned a little to the right, and, when the woman stands erect, is nearly horizontal, being perpendicular to the long axis of the vagina. The fundus is in the plane of the pelvic brim. When the woman is erect and the uterus is in this position all its ligaments, with the exception of the utero-sacral, have their origins and insertions at the same level, and are therefore in no sense suspensory. "The uterus," to quote Alexander, "floats, as it were, in the midst of structures of a specific gravity not much less than itself," and the ligaments simply poise or steady it. When the body is pushed back to the physiological limit, the round ligaments restrain the movement and prevent dislocation of the fundus below the sacral promontory.

The utero-sacral ligaments differ from the others—their uterine attachment occupies a lower level in the pelvis than their insertion into the sacral vertebræ. The ligaments may therefore be called suspensory. They also keep the cervix towards the sacrum, and along with intra-abdominal pressure are the chief factors in maintaining the normal anteversion of the uterus (Fig. 1).

Abnormal Positions of the Uterus.—1st. *Displacements of the Uterus as a whole.*—We have seen that the organ may be elevated, retroposed, or anteposed as a whole by physiological causes. The mobility of the uterus also permits these changes of position to occur as

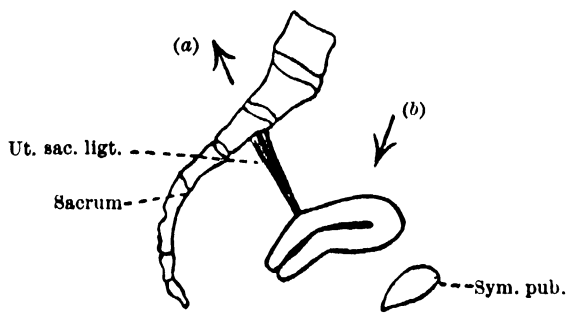


Fig. 1.—(*a*) Represents action of ut. sac. ligt.; (*b*) intra-abdominal pressure.

the result of pathological agencies which render the displacement permanent. For example, *ascent* may be caused by adhesions drawing up the uterus or by tumours pushing it up from below and fixing it permanently in its abnormal position. In like manner *retroposition* of the uterus may be brought about by adhesions posteriorly drawing it and fixing it towards the sacrum, or by tumours situated in front of the uterus pushing the organ back. *Anteposition* may be caused by adhesions in front drawing the uterus forwards, or by tumours behind pushing it against the pubes.

The mobility of the uterus is also such that by fixing a volsella in the cervix one can draw it down to the vulva. This descent occurs pathologically, as when the uterus is pushed down from above by intra-abdominal pressure or by tumours, as in prolapsus uteri. The lateral mobility of the uterus is normally less than in other directions, still it allows of that organ being drawn to one side of the pelvis by contractions or adhesions, or of being pushed there by tumours.

2nd. *Displacements of the Uterus by Version.*—In version the uterus rotates (1) Round an imaginary transverse axis with its fundus forwards and cervix backwards constituting anteversion, or with the fundus backwards and the

cervix forwards—Retroversion; (2) Round an imaginary antero-posterior axis with the fundus to one side of the pelvis and the cervix to the opposite side—Lateriversion.

3rd. *Displacements of the Uterus by Flexion*, by which the relative position of body and cervix is altered. The normal uterus is flexible. Intra-abdominal pressure acting on the upper surface of the uterus (Fig. 1) causes the body to bend forwards on the cervix—the angle found at the junction of body and cervix constitutes the normal anteflexion of the uterus. This normal anteflexion may be increased as in congenital and pathological anteflexion, or the axis of the uterus may become straight as in anteversion. The body is bent over on the posterior aspect of the cervix in retroflexion, and to one or other side in lateriflexion.

4th. *Inversion of the Uterus.*—In this displacement the form of the uterus is altered by its inner surface being turned outwards and its outer surface inwards.

1. Ascent of the Uterus.—The uterus ascends normally during pregnancy. After the fourth month it becomes an abdominal organ. Various diseased conditions of the uterus and surrounding parts cause its ascent; the body may be so much enlarged by a fibroid tumour that it thereby becomes elevated, or a fibroid may grow downwards from the uterine cavity into the vagina and from its size lift up the organ. Vaginal neoplasms and the condition known as hæmatocolpos act in a similar manner. Ovarian, parovarian, and broad ligament tumours, cystic or solid, may, so long as their size keeps them in the pelvis, act similarly. A tube enlarged by extra-uterine gestation, a collection of blood, serum, or pus in the pelvis, either encapsulated or not, may have the same effect.

When the uterus has been elevated into the abdominal cavity by some of the causes already mentioned, and an attack of peritonitis supervenes, it may become permanently fixed in that position. In this, as in every case, the treatment is that of the cause producing it.

2. Descent of the Uterus.—Descent of the uterus occurs in retroversion and retroflexion. In these affections the uterus occupies a lower position in the pelvis than normal. When the uterus descends so low that the vaginal portion protrudes through the vulva, it is said to be prolapsed.

3. Prolapse of the Uterus.—The abdominal viscera, the uterus and its appendages are, when normal, in a state of equilibrium. The abdominal cavity is always full, and intra-abdominal pressure is exerted in all directions; but in the erect posture that pressure, along with gravity, is directed anteriorly against the anterior abdominal wall and the back of the pubes. Inferiorly it is directed against the sacral segment of the pelvic floor. Anteriorly this pressure is counteracted by the resistance of the anterior abdominal wall. Inferiorly the pressure is counteracted by the resistance of the sacral or fixed segment acting at right angles to it.

If from any cause this equilibrium is disturbed, if there is increased intra-abdominal pressure, or weakening of the anterior abdominal wall or pelvic floor, we get protrusions of the nature of herniæ—herniæ of the abdominal wall or the pelvic floor. In the former the herniæ usually occur at the umbilicus, inguinal or femoral rings. In the latter it is the displaceable segment of the pelvic floor that is herniated.

This displacement of the pelvic floor is commonly but erroneously called prolapse of the uterus. Certainly the uterus becomes prolapsed, but as it has nothing to do with the causation of the displacement, taking only a passive part from the fact of its being attached to the portion of the pelvic floor that becomes displaced, it is more correct to call the affection displacement of the pelvic floor.

The affection is essentially a hernia of the displaceable segment of the pelvic floor. The size of the uterus has nothing to do with it. This displacement occurs when the uterus is smaller than normal. Enlarged uteri may become and often do become retroverted, as in subinvolution, or anteflexed as in pregnancy, but one does not get displacement of the pelvic floor on account of the increased size of the uterus.

Displacement of the displaceable segment of the pelvic floor, and the accompanying prolapse of the uterus, is caused by (*a*) increased intra-abdominal pressure; (*b*) weakening of the sacral or fixed or supporting segment of the pelvic floor; and (*c*) deficient tone of the anterior segment of the pelvic floor.

Intra-abdominal pressure is increased when the abdomen is distended by tumours or by fluid; it also results from tight-lacing and from the habit some women have of supporting heavy clothing on bands fastened round the abdomen. It is likewise caused by strong and prolonged muscular exertion, and is induced by the repeated expiratory effort of cough in chronic bronchitis.

The resistance of the supporting segment of the pelvic floor is impaired by parturition, which causes the segment to be straightened out or torn. One often meets with cases, however, where the perineum is completely divided, and yet the uterus retains its normal height in the pelvis. In such a case the remainder of the pelvic floor is sufficiently strong to maintain equilibrium.

Deficient tone of the displaceable segment of the pelvic floor is also a result of parturition. For some time after labour this portion of the pelvic floor may be said to have lost its tonicity, and if a woman rises too soon, that is, before its normal strength has been restored, intra-

abdominal pressure will force the parts down and produce herniation. This descent is favoured by the lax and toneless condition of the utero-sacral ligaments, and may occur when the sacral segment is unbroken and its power of resistance unimpaired. In old women the whole pelvic floor may be wanting in tone, and a slight increase of abdominal pressure may cause the displacement. And, conversely, the resistance of the entire pelvic floor may be normal, but the intra-abdominal pressure may be so enormously increased, as when the abdomen is occupied by large tumours, that the whole

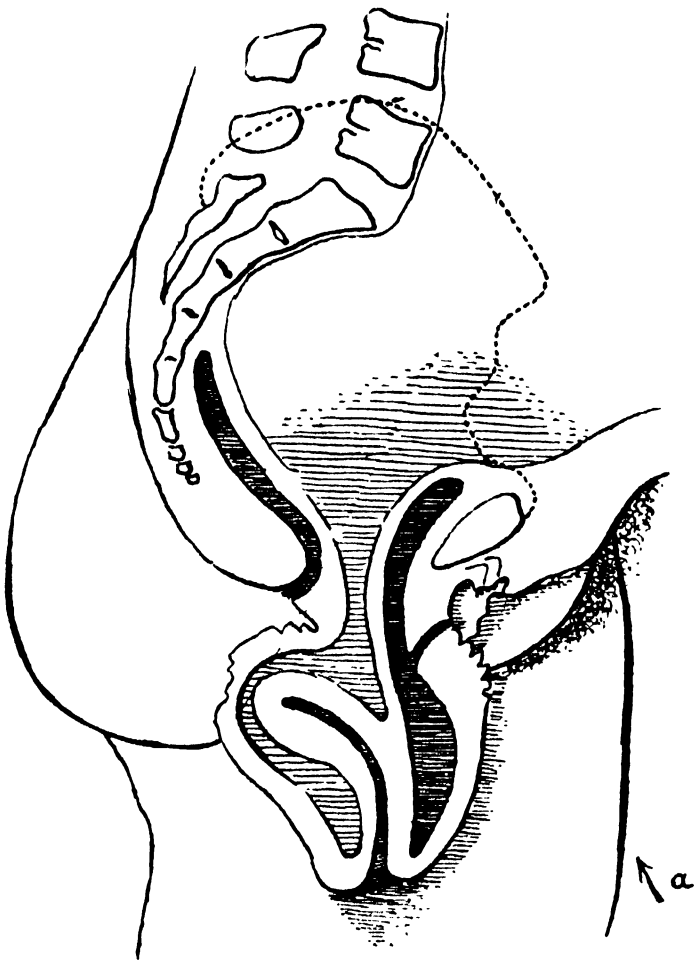


FIG. 2.—Position of the organs consequent on displacement of the pelvic floor.

displaceable portion may be forced out of the pelvis.

The order in which the placed structures appear at the vulva is as follows:—The lower end of the anterior vaginal wall appears first, and then the whole of the anterior vaginal wall from below upwards. This descent is brought about by intra-abdominal pressure separating the anterior vaginal wall from the posterior surface of the bladder. With the descent of the anterior vaginal wall the urethra and posterior wall of bladder also descend, from the fact that the urethra is incorporated with the lower part of anterior vaginal wall. As the uterus is attached to the upper part of the vagina, it necessarily now descends, and in its descent it peels from above downwards the posterior vaginal wall from the anterior rectal wall. When the external os reaches the vulva, the posterior vaginal wall is bent upon itself, and as the uterus descends farther the whole of the posterior vaginal wall becomes everted, with the exception of that portion attached to the perineal body which is fixed to the rectum. So when prolapse is complete the whole uterus, the anterior vaginal wall, the urethra, part of the bladder, the posterior vaginal wall, and the peritoneum are all outside the vulva.

Thus the whole of the anterior segment accompanied by the uterus is outside the vulva.

Let us consider these different structures in their new situation (Fig. 2).

Urethra and Bladder.—The direction of the urethra is altered from downwards and forwards to upwards and forwards, and is completely extra-pelvic. The base of bladder forming cystocele is outside the vulva, and is beneath the urethra, consequently one of the symptoms the patient complains of is dribbling away of urine.

The Anterior Vaginal Wall.—The whole extent of it is outside, and is subjected to irritation from exposure and friction. The result is that it becomes hypertrophied. The epithelium becomes thickened, resembles skin, the rugæ are undone, apt to get horny, or to ulcerate, often covered with a membrane. In long-standing cases changes take place in the bladder walls. They become hypertrophied and catarrhal, and not infrequently the catarrh extends to the ureters and pelvis of the kidney. In the prolapsed pouch of the bladder the urine becomes stagnant, and may lead to the formation of calculi.

The posterior vaginal wall assumes a similar condition to the anterior.

The Uterus.—The external os uteri is situated at the lower anterior part of the protrusion, the body of the uterus lies above it and posteriorly, and can be felt by the finger and thumb between the vaginal walls.

The external os uteri in some cases barely projects beyond the general surface of the protrusion, in other cases the cervix is greatly hypertrophied, and is frequently ulcerated. If it is lacerated the lips may be everted and eroded, and the mucosa of the cervical canal thickened and pouring out a copious secretion. While the body of the uterus will in many instances be found enlarged, with thickened walls and hypertrophied endometrium, in others, especially in women who have passed the menopause, it may be quite small.

The peritoneum is driven down. The anterior and posterior uterine reflections are undone, so

as to present when viewed from above a funnel-shaped opening, in the floor of which is seen the fundus, and at its sides and upper part, rather anteriorly, the ovaries and broad ligaments lie. The utero-sacral ligaments are much stretched, elongated, and thinned. The broad ligaments are also put on the stretch, and their attachments to the side wall of the pelvis are seen to be pulled on. The round ligaments are straightened out, but not stretched, unless the broad ligaments have given way.

The rectum remains in position, but the lower portion of it, which is attached to the perineal body, may be pulled forwards and a pouch formed. This constitutes "rectocele."

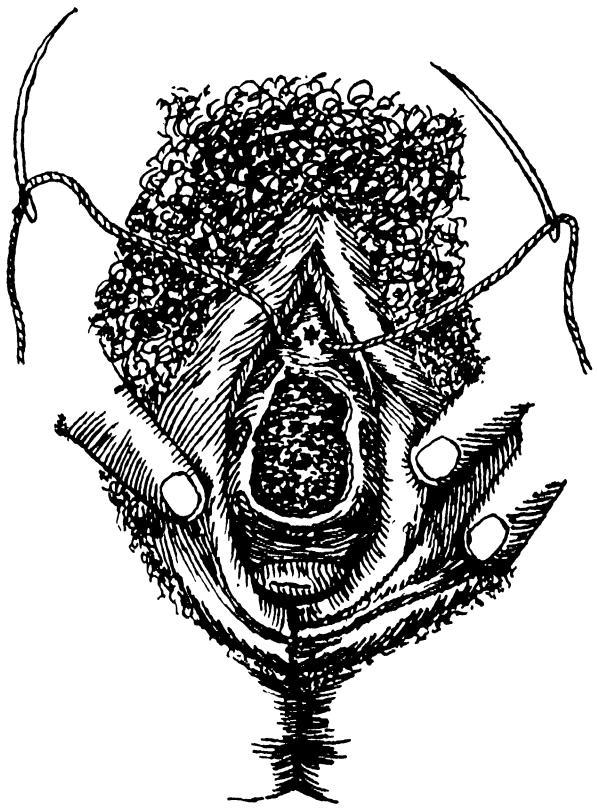


FIG. 3.—Anterior colporrhaphy (Stoltz).

This process of descent is a gradual one, and it is convenient to divide it into three stages :—

(1) The earliest stage, when the anterior vaginal wall with posterior wall of the bladder attached to it appears through the pudendal opening, constituting what is termed cystocele. The uterus in this instance has descended slightly, but is still anteverted.

(2) In descent of the second degree, the external os uteri is at the vulva and the uterus is retroverted. Cystocele and perhaps rectocele are also present.

(3) In descent of the third degree, bladder, both vaginal walls, uterus, and peritoneum are outside the vulva (Fig. 2).

Symptoms.—The patient complains of something coming down in front, or of the womb coming down. When she lies down and the examiner inspects the vulva he may find the parts apparently in a normal condition, inasmuch as no protrusion is to be seen, the explanation being that when the patient assumes the recumbent posture intra-abdominal pressure is removed, and the parts which were prolapsed when the patient was erect have sunk back into the pelvis. In such a case one has only to ask the recumbent patient to bear down, thus bringing intra-abdominal pressure into action, and the anterior vaginal wall will be seen to descend through the vulvar orifice in the shape of an egg. The patient also complains of a sense of weight and dragging in the back. When the uterus is enlarged and endometritis is present the menstrual flow may be profuse, but more often it is scanty. As a rule there is acquired sterility, but conception may take place in a prolapsed uterus and the pregnancy go to term. Abortion is apt to occur from incarceration when the enlarged uterus does not ascend above the pelvic brim. There are difficulties with micturition. In complete descent the direction of the long axis of the urethra is changed from downwards and forwards to upwards and forwards; a considerable portion of the bladder forms a pouch below the level of the urethra, consequently when the patient walks this pouch is subjected to pressure and the urine is forced out in jets *per urethram*. From the changed relations of bladder and urethra there is also difficulty in voiding urine, and some patients affirm that before they can accomplish the act they must push the protrusion back into the pelvis.

Differential diagnosis must be made from the following conditions :—

Hypertrophic elongation of the Vaginal portion of the Cervix.—In this condition the cervix is increased in length but not in thickness, the external os uteri, which is usually small, lies immediately within the vaginal orifice, while both anterior and posterior fornices are in their normal position, and the body of the uterus occupies its normal height in the pelvis. It is found in the virgin, and is probably congenital.

Hypertrophy of the Supra-vaginal portion of the Cervix.—The fundus uteri remains in its normal position, there is great increase in the length of the cervix, so that the external os comes to lie outside the vulva. Both anterior and posterior fornices are obliterated.

Inversion of the Uterus.—In this displacement a globular swelling occupies the vagina, at the upper end of which the uterus can be felt encircled by the cervix. On abdomino-rectal examination the body of the uterus is found to be absent from its normal position, and in its place the margin of a cup-shaped hollow may be palpated.

A uterine polypus which has become pediculated cannot be mistaken for prolapsus uteri if a careful examination be made. The finger introduced into the vagina can trace the pedicle

of the tumour coming through the external os, and if a sound be passed the uterus will be found in its proper place.

Vaginal and vulvar tumours may simulate prolapsus uteri, but can always be differentiated. In the latter the external os can always be discerned at the apex of the protrusion. The only other condition in which this obtains is hypertrophic elongation of the cervix.

Cystocele and rectocele without accompanying prolapse of the uterus can be diagnosed by the passage of a sound into the bladder in cystocele, a finger into the rectum in rectocele, and by ascertaining bimanually that the uterus is in its normal position.

TREATMENT. — *Reduction.* — Reduction as a rule is accompanied with no difficulty. The structures can usually be replaced with ease when the patient assumes the recumbent position and the order of ascent is the reverse of the order of descent. The posterior vaginal wall is first reduced, then the uterus, and lastly the anterior vaginal wall and bladder. In old-standing severe cases the prolapsed parts may be so congested and swollen that reduction cannot be effected without some preliminary treatment, taking the form of rest, hot applications, etc. The presence of intra-peritoneal adhesions may prevent reduction.

Retention. — Keeping in mind the factors which bring about this condition, our aim in treatment with a view to attaining permanent retention must be directed (1) to diminishing intra-abdominal pressure, and (2) to strengthening the pelvic floor so that it may successfully resist the pressure brought to bear on it.

(1) *Diminishing Intra-abdominal Pressure.*— This is best accomplished by rest. When a woman with a prolapsed uterus lies down the pressure is so diminished that the protrusion slides back or can be pushed back into the pelvis, and as long as she remains in this position there is little tendency for it to descend. Rest also enables the pelvic floor to regain its tone. Consequently a patient with this affection should rest as much as possible. Especially is it applicable in the puerperium in those who have any tendency to descent of the pelvic floor. But rest, however useful, is not sufficient to cure; even in the slightest and most favourable case we must adopt some plan of treatment whereby we strengthen the pelvic floor.

(2) *Strengthening the Pelvic Floor—Minor Measures.*—(*a*) Tampons. After the displacement has been reduced the vagina may be packed with cotton-wool soaked in the following mixture — R Boroglyceride ʒss., Aluminis ʒj., Glycerinum ad ʒxvj. The boroglyceride keeps the tampon from becoming fœtid, and along with the glycerine reduces the congestion of the parts, while the alum helps to tone up the relaxed structures. Tampons are serviceable in old women who are too feeble to undergo operative treatment, and in many cases are sufficient to keep up the protrusion. They require to be renewed at least once a week. Ichthyol may take the place of boroglyceride. Iodoform gauze or marine lint also makes an excellent pack.

(*b*) Pessaries are useful in cases in which the descent is slight and the perineum has not lost its retentive power, and in women who from age and general conditions are unsuitable for operation.

In cases of slight descent of the anterior vaginal wall the ring pessary, with perforated diaphragm, will be found most useful. The ring pessary strengthens the pelvic floor more than one after the Hodge pattern, while the diaphragm prevents the cystocele from slipping down. In most instances an efficient sacral segment is necessary for the retention of the instrument, but in exceptional cases one will find that this is not essential.

When the perineum is deficient and a pessary refuses to stay in, the addition of a perineal pad attached by strings to a waistband may secure its retention and make the patient comfortable. An instrument we have found very successful, even in cases of complete prolapse, is Simpson's disc and stem pessary. "The herniation," says Simpson, "can sometimes be prevented by making the patient wear a disc and stem pessary; the stem projecting from the lower surface of the disc lies between the labia. The disc may be circular, but is better elongated from side to side, so as to keep the walls of the vagina extended transversely. The patient learns easily to introduce such a pessary as she lies on her back, by passing in first the one side through the vaginal orifice and then the other, as a button is passed edgewise through a buttonhole. She removes it from time to time when going to bed by laying hold of the stem with the finger and thumb of one hand while the forefinger of the other hand lays hold of one edge of the disc and presses it out. She can thus secure the cleanliness of the instrument, and if need be she can douche the vaginal cavity in the interval of removal."

When the above-mentioned pessaries fail, stem pessaries made of vulcanite or soft rubber with cup-shaped extremities attached by bands to a waistbelt, may be tried. For example Cutter's, or Leith-Napier's. We have found the latter's instrument the best of this class.

OPERATIVE MEASURES. — Before describing some of the numerous operations which have been devised for prolapse it will be well to mention those we consider suitable for cases met with respectively under the first, second, or third stage of descent respectively.

1st. *In the earliest stage*, when the lower end of the anterior vaginal wall appears at the vulva, the uterus being anteverted and the supporting segment intact, the operation indi-

cated is anterior colporrhaphy, and after the patient has recovered from the operation it will be advisable as a precautionary measure that she wear a Hodge pessary for a considerable time to keep the uterus in anteversion.

2nd. *In cases of descent of the second degree*, where the uterus is retroverted and has descended so far that the external os uteri has reached the vulva, cystocele and perhaps rectocele being present and the perineum deficient. Here the operator will restore the integrity of the perineum, and will narrow the vagina by anterior and posterior colporrhaphy, or better by bilateral colporrhaphy. These operations do not affect the position of the uterus. It must be raised to its proper height, placed in anteversion, and measures taken to keep it in that position. To accomplish this we must adopt measures which will keep the cervix towards the sacrum (that is, which will perform the lost function of the utero-sacral ligaments) and the fundus to the front, so that intra-abdominal pressure may be directed on the posterior or upper surface of the uterus. The first of these indications may be fulfilled by the Hodge pessary, which, by pushing upwards and backwards the posterior fornix, keeps the cervix towards the back.

The pessary may be sufficient to keep the uterus in anteversion, but this result will be more certain of attainment if the operator in addition shortens the round ligaments extra-peritoneally. The maintenance of the uterus at its normal height in the pelvis, with its long axis directed towards the front, is all-important. If this cannot be accomplished, no matter how successful the operations which have been performed on the pelvic floor may be, a recurrence of the displacement is almost certain to occur sooner or later, for the simple reason that the uterus falls back into retroversion, and intra-abdominal pressure acting on the anterior surface of the uterus pushes down the pelvic floor. The operative measures above mentioned we have found successful in many old and severe cases of prolapse. The pessary, which is playing the function of the utero-sacral ligaments, may, however, be rightly considered a weak point. In some cases of recent prolapse the utero-sacral ligaments may only be stretched and weakened, and may after a time regain their tone and function and allow the pessary to be dispensed with. But these are exceptions. In the majority of cases these ligaments are irreparably destroyed, and in them the pessary will be a necessity for an indefinite period. For these reasons some gynæcologists feel justified in opening the abdomen and adopting measures which will fix the uterus permanently in anteversion, and at a level which is either normal or above it. For accomplishing this end the gynæcologist may adopt the operation of ventral fixation devised by Olshausen and modified by Kelly, or he may attempt to attain the same end by restoring the utero-sacral ligaments, either by the recently introduced method of Stanmore Bishop or by that of Bowreman Jessett. These operations for the restoration of the utero-sacral ligaments fulfil the indications desired, and the operator can attain his object with greater certainty if he at the same time shortens the round ligaments intra-peritoneally.

3rd. *When the uterus is completely prolapsed* both vaginal walls are everted, and the bladder and peritoneum are outside the vulva. In extreme cases of this sort the surgical procedures mentioned under class 2 are applicable, especially in women who have not passed the menopause, or who, having passed it, are unwilling to have the marital function interfered with. Those not affected by this consideration may have an operation which has been recently introduced, and which promises to be a complete and permanent cure of the condition. The operation referred to consists in removal of the entire vagina and uterus, that is, panhystero-colpectomy.

In the foregoing summary we have mentioned various operations and their indications, and we now describe these operations in detail.

When the vaginal portion of the *cervix is hypertrophied* it should be amputated. This is done by fixing a volsella in the antero-posterior lip, and with a pair of strong scissors or a knife make a circular amputation, the edges being brought together by passing stitches antero-posteriorly, or better, from side to side, taking care not to obliterate the cervical canal.

Anterior Colporrhaphy —One of the simplest methods is that of Stoltz (*see* Fig. 3), which consists in fixing the anterior vaginal wall at four points, and dissecting away all the redundant tissue between these, thereby making a raw surface which is narrowed by means of a suture introduced around the rawed surface in the form of a purse-string. Another effective plan is to fix a volsella in the cervix and draw the latter down to the vulva, thereby making the anterior vaginal wall taut. With the knife one cuts through the entire thickness of the anterior vaginal wall in the middle line from cervix to pubes. The vaginal wall on each side of the incision is reflected off the bladder for a distance of about one inch, then the rawed surface of the vaginal wall on each side of the incision is brought into apposition by means of mattress sutures of thick catgut or silkworm gut, so that the reflected vaginal portions of the anterior vaginal wall resemble the keel of a ship.

A third method may be proceeded with in the following manner :—An elongated elliptical round surface is made on the anterior vaginal wall, beginning at the cervix and finishing near the urethral orifice. In the

middle part the whole width of the vagina is rawed. The wound is brought together by a continuous catgut suture; the result is a great constriction of the lumen of the vagina. A similar procedure may be practised in the posterior vaginal wall.

Bilateral colporrhaphy is one of the best methods of narrowing the vagina. In this operation a strip of mucous membrane is removed from the sides of the anterior and the posterior vaginal walls throughout the entire length of the vagina (Fig. 4), (fig. 594, p. 460, Montgomery's *Gynæcology*). The fixation of the parts is more permanent after this than after any of the operations already mentioned. It has the advantage that the narrowing can be made to any degree desired.

Restoration of the Perineum.—When the Tear does not extend into the Rectum.—The integrity

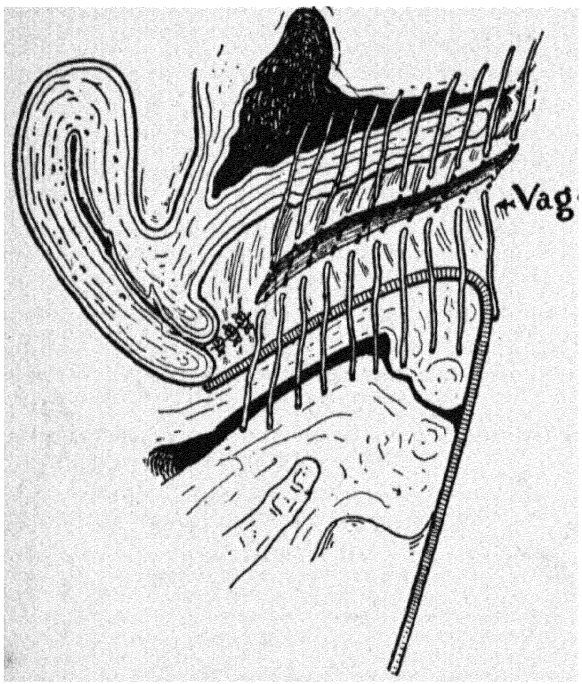


FIG. 4.—Bilateral colporrhaphy.

of the perineum can be satisfactorily restored by a simple flap-splitting operation. The lower part of the posterior vaginal wall and the lateral margins of the vulva can be raised up in the form of a flap by simply carrying the knife or scissors underneath the parts mentioned and dissecting up the flap. A large rawed surface is produced which is brought together by a continuous catgut suture in such a manner that the whole of the rawed surface is brought into apposition, and the edges of the vulva meet in the middle line. The suture is introduced in the deepest part of the wound, and is carried upwards to the upper extremity of the wound, and then downwards alternately until the skin edge is reached This method rolls the tissues up into the form of a thickened pad.

When the Tear has extended into the Rectum and the External Sphincter Muscle is divided and retracted.—One of the best operations for the repair of this condition is one devised by Kelly, and is a flap-splitting method with the addition that the ends of the divided sphincter muscle are sought out, and by stitching are restored to normal shape and position (Fig. 5) The patient is placed in the lithotomy position, and after the field of operation has been thoroughly cleansed a plug of gauze is inserted into the rectum with a string attached to prevent the escape of fæcal matter during the operation. The assistants then stretch the recto-vaginal septum by placing a finger on the buttock immediately opposite each extremity of the septum. The operator then with the knife or scissors carefully separates the posterior vaginal wall from the anterior rectal wall to the extent of about one inch, taking care to avoid button-

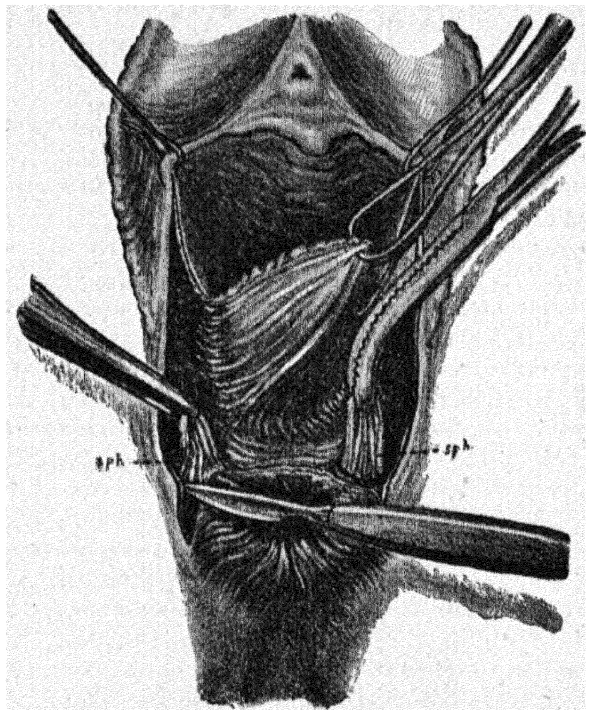


FIG. 5.—Restoration of the perineum when the rectum has been torn and the external sphincter muscle divided (Kelly)

holing. Then the knife or scissors is inserted at the extremity of the wound with which the operation was begun, and carried under the skin upwards along the margins of the vulva as far as the base of the nymphæ. Forceps are placed on the cut edges, and the flap is dissected upwards to the necessary extent. The operator then carries two incisions downward and backwards directly over the retracted ends of the sphincter, which can always be felt between the finger and thumb, and there is seen always a slight dimple on the skin at the retracted end. The ends of the sphincter are now carefully dissected out, and seized with forceps which pull them into their original position and hold them there while the operator stitches them together with catgut. It is usual to put in three stitches. It is advisable to insert at this stage a silk-worm gut suture from the cut edge

of the skin on one side through the restored sphincter, to be brought out at the edge on the opposite side with the object of supporting the parts. This suture may be cut short and may remain as a permanent buried suture. The further stages of the operation consist in bringing all the rawed surfaces together. This again is best accomplished by the continuous suture of catgut in the same way as described in the previous operation.

Ventral Fixation.—In order that this operation may be of any service in the treatment of prolapse, the uterus must be firmly fixed in the abdominal parietes. The uterus is drawn up into the abdominal wound, two stitches are inserted through it to the depth of about a quarter inch on its posterior aspect, immediately below the fundus, with the object of keeping it in anteversion, so that intra-abdominal pressure may be exerted on the posterior or upper surface of the organ. The material may be either silk, silkworm gut, or chromicised catgut. Many operators will doubtless prefer a non-absorbable material where such a firm fixation is required. The operation should be limited to those cases in which pregnancy is impossible.

Intra-peritoneal Shortening of the Round Ligaments.—This operation consists in doubling up from two to four inches of the round ligament on each side and uniting it by sutures. The shortening thus produced keeps the fundus uteri towards the front. It will be well in this operation to use a non-absorbable suture.

Operations for the restoration of the utero-sacral ligaments may be accomplished—(*a*) By the method of Jessett. He describes the operation thus; "In cases of complete procidentia, in which the peritoneum in Douglas's pouch is prolonged quite through the vulva, the sacro-uterine ligaments, which have become much elongated, should be restored by drawing up the posterior fornix of the vagina, and fixing the peritoneum covering it to the posterior wall of the uterus and to the parietal peritoneum lining the sacrum. If necessary a fold or tuck may be made in each broad ligament, and in cases of cystocele the bladder may be drawn up and stitched to the peritoneum covering the uterus". (*British Gynæcological Journal*, May 1901, pp. 29, 30).

(*b*) Another method is that of Bishop. He says: "The technique of the operation, as I have carried it out, is briefly this. The protrusion being reduced the patient is placed in the extreme Trendelenburg position, and the abdominal wall is opened by a median incision. As soon as the intestines have sunk away from the pelvis towards the diaphragm, two threads are passed through the broad ligaments, one on either side of the uterus, enclosing tube and round ligament; the ends of these threads are tied, and by them as tractors the fundus of the uterus is drawn forwards. A special sound is passed by an assistant into the vagina, and made to press up towards the posterior fornix so as to render it prominent. On either side a stout, thick thread is passed vertically through the substance of the fornix, avoiding the mucous lining, so that each protruding end is half an inch distant from the other, and the whole loop half to three-quarters of an inch from the cervix. The fornix is now applied to the sacrum, and a spot is chosen directly opposite, free from vessels and subjacent nerves and ureter, and well outside the rectum, where the needle carrying this suture is entered deeply so as to embrace the periosteum covering the bone; it is brought out again half an inch directly above its point of entrance. Before tying this suture a narrow strip of peritoneum is removed from that portion of the fornix which lies in its grip, so as to bare the connective tissue beneath. This is repeated on the opposite side; the sutures are tied, and their ends cut short. Sometimes the position of the rectum will only permit of single fixation. This should then be more central in its position as regards the uterus, and somewhat broader. The new ligament or ligaments are now formed, and the cervix hangs in its normal position from the sacrum by that portion of the vagina which lies between it and the sutures" (*Medical Press*, 24th December 1902).

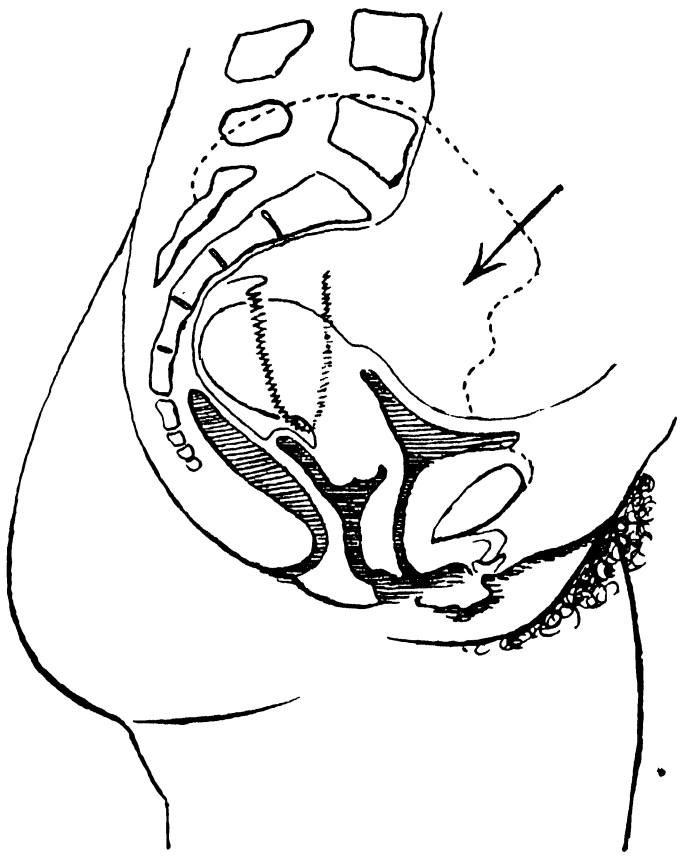


FIG. 6.—Retroversion of the uterus.

(*c*) *Panhystero-colpectomy.* — This operation has been performed by several operators with good results. The method described by C. Martin of Birmingham is excellent, and we here give his description of it:—

"(1) The patient was anæsthetised and placed in the lithotomy position, and the vulva, cervix, and vagina (which was completely turned inside out) rendered aseptic.

"(2) The cervix was seized with volsella and drawn forward, and an incision made through the vaginal mucous membrane from the posterior lip of the cervix to the edge of the perineum. From the latter point two curved incisions were carried forward, one on either side at the junction of the vaginal mucous membrane and the skin of the labia meeting in front about three-quarters of an inch behind the meatus urinarius, that is, near the posterior edge of the vestibule. It will be seen that these incisions encircled the ostium vaginæ.

"(3) The mucous membrane of the posterior vaginal wall was dissected off with scissors and turned forward; but at this stage the mucous membrane of the anterior vaginal wall was not interfered with.

"(4) The peritoneum of the pouch of Douglas was next opened by a transverse incision, and the uterine fundus exposed and drawn down. The broad ligaments were ligatured and divided from above downwards outside the ovaries and tubes.

"(5) The peritoneum between the bladder and uterus was divided transversely, and the bladder stripped off the cervix and upper part of the vaginal wall with the finger.

"(6) The mucous membrane of the anterior vaginal wall was next dissected off the bladder with scissors, and the uterus, ovaries, and tubes, together with the whole vaginal mucous membrane, removed in one piece.

"(7) All bleeding points having been seized and ligatured with fine silk, the peritoneum of the pouch of Douglas was sutured to that on the bladder with a fine silk suture.

"(8) The fascia at one side of the vagina was next sutured to that on the other side by a fine continuous suture beginning above at the level of the broad ligaments, and working gradually downwards to just above the vulva. In this way the bladder was pushed forward and the rectum backward by a firm column of connective tissue derived from the lateral fasciæ.

"(9) The vulvar wound was then closed by interrupted silkworm gut sutures approximating the posterior halves of the labia majora.

"(10) The wound was dusted with iodoform, and a pad of iodoform applied. For some days the patient's urine was drawn off through a catheter. She was kept in bed for three weeks. After leaving the hospital she continued to improve, and when I saw her again on 30th June (the operation was performed on 11th May) she was quite well. Since that date she has been to the out-patient department to report herself from time to time. I examined her in the early part of July, and found her condition most satisfactory. She was perfectly comfortable, had complete control of the bladder, and no trace of any protrusion of any kind. The vulvar scar was quite painless, and, in her own words, 'life was now a pleasure instead of a continual misery'" (*British Medical Journal*, 5th October 1901).

4. Anteposition — Retroposition — Dextro-position—Sinistro-position.—In this group of displacements the uterus has been moved bodily from the normal position like one of the above-mentioned. These abnormal positions are caused either by tumours pushing the organ out of its place, or by cicatricial contractions drawing it out of place. Tumours of the sacrum, rectum, and ovaries, tumour in Douglas's pouch, or a fibroid tumour growing from the posterior wall of the uterus, may push the organ forwards into anteposition. The same result may be caused by hæmorrhagic extravasation or inflammatory effusion in Douglas's pouch. The organ may also be drawn forward by the cicatricial contractions resulting from anterior parametritis.

5. Retroposition.—Tumours in front of the uterus push it into retroposition, such as tumours of the bladder or ovarian tumours. The influences which produce this position, acting from behind, are the adhesions which result from either a collection of serous matter or extravasation of blood.

6. Sinistro- and Dextro-position of the uterus are produced by tumours growing between the layers of the broad ligament, or are the results of cicatrisation following the inflammation of the connective tissue of these layers.

It is noteworthy that an inflammatory or a hæmorrhagic swelling which at an early stage produces one displacement, at a later period produces a displacement in the opposite direction. For example, a swelling of that nature in Douglas's pouch during the earlier stages of its existence thrusts the uterus forwards into anteposition, while at a later stage, after absorption has been well accomplished, cicatrisation follows and pulls the uterus back into retroposition. In like manner a cellulitis between the layers of the left broad ligament pushes the uterus to the right side of the pelvis, while after absorption and the cicatricial contraction which follows it have occurred, the uterus is drawn to the left side of the pelvic cavity. The left lateral position of the uterus is a very common displacement, and is explained by the frequent occurrence of left-sided cellulitis arising from the frequency of a tear on the left side of the cervix during labour, and the septic absorption which follows it.

Retroposition of the uterus frequently gives rise to troublesome rectal symptoms from stenosis, owing to the narrowing of the rectum, which is inevitable at the site of the peritonitic bands and consequent interference of its function. The diagnosis of all these positions is very important, more especially in ante- and retro-position. It may be difficult to differentiate the uterine body from the swelling which produces the displacement. The careful use of the sound and bimanual examination under an anæsthetic should clear up the matter in case of doubt. The practitioner cannot exercise too much care in recognising the presence of retro-peritoneal hæmatocele. This condition has frequently been mistaken for an enlarged retroverted uterus, and attempts at replacing such have been followed by most disastrous results. All the above displacements do not call for treatment, but the condition causing them does so.

7. Pathological Anteversion and Anteflexion.—The uterus normally is anteverted and anteflexed, but the degree of version and flexion which may be considered physiological cannot be defined, and what one examiner may consider pathological another would consider normal. Symptoms do not help us much in making the definition, because one patient may have an exaggerated anteversion, without symptoms, or the change in the inclination and the bend of the uterus may be very slight, yet the symptoms may be well marked. And again, the symptoms may be due, not to the displacement, but to the causes which produce it. There are, however, two well-defined anomalies of the normal condition which can be without doubt pronounced pathological. These are known as pathological anteflexion and pathological anteversion, both of which occur as the result of inflammation posterior to the uterus, and the chief characteristic of these forward displacements is that the uterus has lost its normal mobility. Schultze defines pathological anteversion as "that position of the uterus in which, extended in shape, it lies with the fundus forwards, and is more than normally stabile," and pathological anteflexion as "that position of the uterus in which, permanently flexed over its anterior surface, it lies with the fundus forwards, and is more than normally stabile."

Pathological Anteversion.—*Physical Signs.*—The uterus is straightened out, the relation between the body and cervix is altered, that is, the normal anteflexion is abolished. The body is enlarged, is stiff and rigid, and has lost its flexibility and much of its mobility.

On making a bimanual examination, th
vaginal portion of the cervix will be felt
placed and altered in direction from do
and backwards to backwards and
Through the anterior fornix the
of the uterus will be made out,
ence, with its mobility impaire
and cervix will be felt to be in the same straight line. If a thorough examination be made under anæsthesia inflammatory thickening may be recognised in the region of the utero-sacral ligaments. This thickening can be best felt *per rectum.*

Etiology.—Anteversion is generally a result of puerperal inflammation. The extended shape of the uterus is due to metritis, which is usually brought about by interference with the involution of the uterus. The metritis makes the uterus rigid, and increases its bulk. Posterior cellulitis accompanies the metritis, and leads to rigidity and contraction of the utero-sacral ligaments, which pull up and fix the cervix to the sacrum at a high level. Intra-abdominal pressure pushes down the rigid, straightened-out body, while the cervix is moved in an elevated position posteriorly.

Symptoms. — The symptoms are those of metritis and posterior cellulitis. In addition there may be added vesical troubles, pain whenever the bladder becomes moderately distended, and frequent desire to micturate.

Pathological Anteflexion.—*Physical Signs.*—On bimanual examination the vaginal portion of the cervix will be found high placed and nearer the sacrum than normal. Through the anterior fornix the body of the uterus is felt acutely flexed on the cervix. The uterus may be flexible, or it may have lost its flexibility. The thickened utero-sacral ligaments may be palpated through the posterior fornix, and the examiner will feel that they prevent him from drawing the uterus forward to the front.

Etiology.—The commonest cause of pathological anteflexion is shortening of the utero-sacral ligaments, the result of cellulitis. The contracted folds of Douglas pull the cervix back towards the sacrum, and fix it there. Intra-abdominal pressure acting on the posterior surface of the uterus, which is still flexible, pushes it downwards towards the anterior aspect of the cervix, thus producing an exaggerated anteflexion. The subsequent metritis destroys its flexibility. The posterior cellulitis is not always puerperal, as it is frequently met with in the unmarried and in those who have had no children.

Symptoms.—The symptoms are those of utero-sacral cellulitis, metritis, and endometritis. Pain in the back, and pain and difficulty in defæcation, are the leading symptoms which can be attributed to the posterior inflammation. The uterine conditions give rise to leucorrhœa, perhaps menorrhagia, dysmenorrhœa, and
terility.

Treatment.—Treatment is directed to the abnormal condition of the utero-sacral ligaments and of the uterus. Pain in the back, which is due to dragging of the uterus on the shortened and inflamed ligaments, should be treated in such a way as to procure rest to these struc-

tures. The chief factors producing unrest or tension of these are—(1) passage of large masses of fæces; (2) intra-abdominal pressure forcing down the uterus. The first of these is treated by aperients or by daily enemata. Intra-abdominal pressure is minimised by rest, and is resisted by a Hodge or figure-of-eight pessary, which by its upward pressure supports the uterus, and thus removes the tension from the utero-sacral ligaments, and relieves the dragging pain in the back which is the leading symptom of this condition. One may also attempt to restore the ligaments to their normal condition by first promoting absorption of the thickening. This may be accomplished by glycerine plugs, hot douches, warm sitz baths, or special treatment at watering-places such as Kissingen, Marienbad, where the methodical application of heat is practised in the form of sulphur, peat, or sand baths. Secondly, by endeavouring to lengthen the ligaments by massage under anæsthesia, or, as has been recently advocated by Reed of Chicago, one may cut through the posterior vaginal wall and resect the ligaments, thus allowing the cervix to come forward and assume a more normal position. The dysmenorrhœa may be premenstrual and menstrual. In the former case it may be attributed to the congestion which takes place in the pelvis before the flow appears, and which would affect the inflamed ligaments. The pain which is present during the period is due to the metritis, endometritis, and the acute bend between the body and cervix. Dilating the uterus and curetting the endometrium will be found most beneficial. Should any erosions be present on the vaginal portion, or if the cervix is lacerated or hypertrophied, these conditions should receive appropriate treatment.

8. Congenital Anteflexion. — Under this term is usually described a uterus having the characters of that of the child. In early life the body of the uterus is small, and acutely anteflexed on a comparatively large cervix. At puberty the body should enlarge and become more erect, but when it fails to do so the organ retains its childish shape. This anomalous form of uterus being due, therefore, to an arrest in the development of the organ, cannot be properly considered a displacement, though it is generally classed amongst them.

Physical Signs.—In a typical case they are as follows:—The vagina is short, the cervix, which lies in the long axis of the vagina, is long, slender, and pointed. The increased length of the intermediate portion is usually very marked. The external os uteri is small and round (pinhole os), the body of the uterus is also small and round, and is acutely anteflexed on the cervix. The flexibility is increased in some cases, and in some cases the corpus uteri is not deficient in size.

Associated with this condition there is occasionally thickening and shortening of the utero-sacral ligaments.

Symptoms.—The chief are dysmenorrhœa and sterility. The onset of pain usually occurs a day or two before the period, and continues severe during the first day or until the flow is established. The amount of menstrual discharge is scanty, usually pale in colour, frequently clotted, and sometimes contains pieces of membrane. Occasionally the menstrual flow is copious. Sometimes there is frequency of micturition. Specially is this the case during the periods. In severe cases the premenstrual pain may last a week, and in some there may be more or less continuous intermenstrual pain. Frequently there is dyspareunia.

Treatment.— In the unmarried, dysmenorrhœa is the most urgent symptom. In the married, sterility is often quite as important. Both symptoms may be said to be due in the majority of cases to the shape of the uterus. The obstacle to the easy outflow of menstrual fluid is the narrowness of both the external and the internal os uteri, and explains in the most rational manner the production of the dysmenorrhœa. It is also not unreasonable to consider the sterility due to the same obstruction. The relief which the patient obtains from pain, as well as the frequency with which sterility is cured by free division of the cervix and dilatation of the internal os, justify the above view. The pain is sometimes in part due to the accompanying endometritis and utero-sacral cellulitis, and in instances where this symptom is in force throughout the intermenstrual period the operator may infer that the uterine adnexa, particularly the tubes, are at fault. In a few cases of this class, in which the symptoms were severe, we found on abdominal section that the tubes were imperfectly developed with blind extremities. In extreme cases abdominal section is justifiable for the purpose of removal of the tubes and ovaries to induce the menopause, but even in such cases one would first try the effect of division of the cervix and dilatation of the internal os. The best operation for accomplishing the latter object is that of Dudley of Chicago, in which not only is the external os permanently enlarged and the internal os dilated, but the flexure is overcome by altering the direction of the cervix from downwards to downwards and backwards. The operation also removes any endometritis that may be present.

The patient is put in the lithotomy position, the speculum is introduced, the cervix is divided with scissors backward in the middle line
the utero-vaginal attachment. The uterus
dilated and curetted. For the rest of
tion we quote Dudley's own words:
urfaces thus incised are then held
y means of two tenacula in the
tant; the incision is somewhat

deepened by means of a scalpel, especially in the uterine wall next to the cervical canal, and a small angle is cut out on either side, as shown by the dotted line in Fig. 382. The cut surface on each side is now folded on itself by a single silkworm gut suture, as shown in Fig. 382. This suture is tied and fortified by interrupted sutures on either side. The lines of union thus made are shown in Fig. 383. These sutures are not introduced in such a manner as to stitch the intra-cervical to the vaginal margin of the wound, but the cut surface is folded upon itself in a direction at right angles to this. On either side, that point at the margin of the os externum where the backward incision commenced is stitched to the very angle of the incision, so that each cut surface is folded upon itself, not from within outward, but from before backward. Thereby the os externum is carried directly back to the angle of the incision. The cervix now points backward in its normal direction toward the hollow of the sacrum, instead of forward toward the vaginal outlet. . . . Conjoined examination upon completion of the operation in each of the author's cases has invariably shown the uterus either to have been straightened, or the anteflexion to have been reduced to a degree quite within physiological limits. The results have been substantially the same whether the point of flexure was at the os internum or below it. The two posterior lines of sutures have the effect of transplanting the os externum to the very angle of the posterior incision. The anterior sutures, if used, have the effect of carrying the cervix back by a distance equal to one-half the length of the anterior cut surface, which is doubled upon itself. By these means a permanent change, quite equal to overcoming the flexure, is effected in the direction of the cervix. As the result of the anterior portion of the operation, the uterus in a suitable case is lifted also to a higher plane in the pelvis, where it ceases to be a mechanical irritant to the bladder. This portion of the operation may therefore be indicated for descent when complicated with anteflexion" (Dudley, *Diseases of Women*, p. 581, etc.).

From a large experience of this operation we are well satisfied with the results. As a rule, patients are cured of the dysmenorrhœa for a considerable length of time, and very frequently pregnancy occurs after the operation. The introduction of a glass stem pessary half an inch in diameter after the posterior lip of the cervix has been divided and the internal os widely stretched is a method favoured by some operators. The stem pessary is kept in position by packing the upper part of the vagina with iodoform gauze, and may be kept in the uterus five or six days. After the instrument is removed the cavity of the uterus should be irrigated with an antiseptic solution. The results of this method are not likely to be so lasting as after Dudley's operation.

Treatment short of operative measures consists in giving remedies with a view to the alleviation of symptoms. Counter-irritation in the form of small fly-blisters applied over the seat of pain whenever it commences is one of the most effective remedies for dysmenorrhœa. The promotion of diaphoresis at the outset is helpful. No anodyne, with the exception of morphia, is likely to remove the pain, and morphia cannot be too strictly forbidden, as the symptoms of anteflexion lend themselves to the acquirement of the morphia habit.

9. Retroversion and Retroflexion.—These retro-deviations are of frequent occurrence. In some instances they give rise to no appreciable symptoms, in others the symptoms they cause are very persistent and troublesome.

Retroversion occurs when the uterus rotates round an imaginary transverse axis—the fundus backwards, the cervix forwards. This displacement occurs physiologically whenever the bladder is fully distended, but this is a temporary displacement. Whenever it becomes permanent it is pathological. It occurs as a congenital condition associated with a shortening of the anterior vaginal wall. It is also the position the uterus assumes in senile involution. When a retroverted uterus is abnormally rigid, as when stiffened by metritis, it remains extended, but when it is not rigid intra-abdominal pressure directed against its anterior surface causes it to bend at the isthmus, and the body is bent over on the posterior surface, thus producing retroflexion.

Etiology.—The commonest factor in the production of *retro-displacements* is a relaxed condition of the utero-sacral ligaments. This relaxation may be produced by habitual over-distension of the rectum in a debilitated subject, but the most frequent cause of the condition is the puerperal state. In the puerperium the uterus is large, soft, and heavy, and all its ligaments are lax. While in this state an over-distended bladder may easily push the uterus into retroversion, more especially if it is aided by the intra-abdominal pressure which would accompany any muscular effort on the part of the mother, such as lifting the child. The return of the uterus into anteversion, which should follow the emptying of the bladder, does not take place owing to the toneless condition of the round and utero-sacral ligaments. The consequence is that the latter are stretched and lengthened, and intra-abdominal pressure now acting on the anterior surface of the uterus, the latter remains permanently in retroversion (Fig. 6).

The displacement may be produced in the non-puerperal state in a somewhat similar manner when there is an increase of intra-abdominal pressure, as in vomiting, lifting heavy weights, or overreaching. This can only occur

when the bladder is full. It may also take place without distension of the bladder, as when a patient receives a fall on her sacrum.

Inflammatory action behind, or in the cellular tissue in front of, the uterus, may draw it out of its place and fix it in retroversion; while tumours in front may push it into the abdominal position.

Physical Signs.—The vaginal portion of the cervix is low placed and directed downwards, or downwards and forwards. The vaginal walls are lax. Through the posterior fornix a body is felt, which on bimanual examination is found to be the corpus uteri. The fundus is directed towards the sacrum, the degree of retroversion being determined by the particular portion of the sacrum opposite which it is found. With the exception of congenital and senile cases the uterus is usually enlarged in a uniform manner and of soft consistence. In most instances the mobility of the organ can be made out by the examining finger, though in some the assistance of anæsthesia will be required. In retroversion the rigid body is extended, and in the same straight line with the cervix; while in retroflexion an angle is felt on the posterior aspect of the uterus, generally at the junction of body and cervix.

Symptoms.—Menorrhagia and leucorrhœa are marked symptoms, and are due to the accompanying metritis. When menorrhagia occurs during lactation the uterus will generally be found retroverted. Bearing down is frequently complained of, and may be attributed to the low position and increased size of the uterus. Pain in the back is a prominent symptom, and is due to the stretching of nerve filaments chiefly in the elongated utero-sacral ligaments. Difficulty and pain in micturition and defæcation are also common symptoms. Conception may occur, and in many instances the pregnancy reaches full term. Incarceration sometimes takes place about the fourth month. Women who always abort may carry to term after the uterus has been replaced.

Treatment.—The metritis and other associated inflammatory conditions must first be treated. Rest, tampons, the hot douche, and curettage will be found most serviceable for this purpose. After all tenderness has been removed the displaced organ must be replaced. This is accomplished best of all by bimanual reposition, usually with the patient anæsthetised, or the uterus may be brought to the front with the sound, or by making the patient assume the genupectoral position and pushing the uterus forwards by a finger in the rectum. The retention of the uterus in anteversion is effected by vaginal pessaries or by various operative procedures. The Hodge pessary, or some modification of it, is best suited for the purpose. A pessary acts by keeping the cervix towards the sacrum, and thus fulfilling the function of the utero-sacral ligaments. Many operations have been devised to retain the fundus permanently to the front. We will mention four of the more prominent. *Vaginal fixation*, by which the anterior surface of the corpus uteri is fixed in an incision made in the anterior vaginal wall, while it accomplishes its object, cannot be regarded with favour owing to the risk in subsequent pregnancy and labour, but in women past the child-bearing period it is quite a good method. *Ventral fixation* consists in fixing the body or fundus to the anterior abdominal wall. The risks to pregnancy are the same as those of vaginal fixation, though in a minor degree; and the operation of necessity involves the opening of the peritoneal cavity. *Shortening the round ligaments* by the Alexander-Adams method keeps the uterus in anteversion, but does not fix it, and therefore the fundus is not prevented from ascending during pregnancy. It is performed extra-peritoneally, and is attended by practically no risks. *Shortening the utero-sacral ligaments* through the vagina, as recently planned by Bovée, promises well. Its aim, the restoration of the function of the utero-sacral ligaments, is correct in principle. If this can be attained the operation should, together with that of shortening the round ligaments, restore the normal position of a movable retroverted or retroflexed uterus.

Fixed retroversion or retroflexion requires for its treatment the removal of the cause which produces it before replacement and retention are attempted.

III. Inflammations of the Uterus

INFLAMMATION—
Acute Metritis and Endometritis . 340
Chronic Metritis 341
Chronic Endometritis . . . 343
Chronic Endocervicitis . . . 344
SUBINVOLUTION 346
SUPERINVOLUTION 347

Acute Metritis and Endometritis.—Acute inflammation of the uterus, when compared with the chronic form, is a comparatively rare disease. And while the latter is often confined to one layer of the uterine wall, in acute inflammation we generally find the peritoneum, the muscular tissue, and mucous lining all equally involved at the same time.

Etiology.—It is most frequently met with as a result of septic infection following childbirth or abortion, the infection taking place at the placental site or from some laceration of the cervix. Septic infection may also result from the use of the uterine sound, tents, or intra-uterine pessaries, or follow operations on the uterus, such as the removal of a polypus or curetting of the endometrium. After septic infection, extension of a gonorrhœal inflammation from the vulva and vagina ranks second in importance as a cause of acute uterine

inflammation. An acute general inflammation of the uterus, which probably starts from the endometrium, is occasionally met with during the progress of the exanthematous fevers, *e.g.* scarlatina and typhoid. Sudden suppression of the menstrual flow from chill or excitement is said to cause it, but this is doubtful. Formerly, when strong caustics were so much in vogue in treatment of uterine disease, acute inflammation not infrequently followed their use.

Symptoms.—The disease is generally ushered in by a rigor followed by fever. The temperature may range from 102°-105°, and there may be several chills in the course of the twenty-four hours. The pulse is rapid, 110-120. Locally, there is more or less acute pain in the pelvis, with great tenderness on palpation over the fundus uteri. There may be great rectal and vesical tenesmus. In septic puerperal cases the lochial discharge is generally fœtid and greatly diminished in quantity. In non-puerperal cases the uterine discharge is at first serous and then muco-purulent. In rare instances it may be sanguinolent from the commencement.

Physical Signs.—Vaginal examination reveals a hot, moist vagina, the cervix swollen, the os patulous, and the uterus enlarged, softened, and very tender. These signs, taken in conjunction with the acute character of the symptoms and the history of the case, leave no doubt as to the diagnosis.

The prognosis in puerperal cases is of course very grave. The same is true in cases following gonorrhœa and operative interference, though here there is not the same tendency for the inflammation to become general or for a general infection to occur.

Prophylaxis. –The causes of acute inflammation which have been given indicate how the disease in the great majority of cases may be prevented, viz. by the careful use of antiseptics during labour and in all gynecological operations and manipulations. During menstruation women should avoid exposure to cold and too great physical exertion.

Treatment.—The treatment of acute septic inflammation of the uterus is considered under the head of "PUERPERAL FEVER," and need not therefore be referred to here, for whether puerperal or non-puerperal the disease is to be combated by the same measures, though, as we have stated, with a greater hope of success in the non-puerperal cases.

In the less acute form of acute inflammation, non-septic in origin, the patient must be kept absolutely at rest. Hot fomentations or an ice-bag where the temperature is very high, over the lower abdomen, give great relief to the pain. Hot vaginal douches act in the same way, and tend to relieve the congestion and allay the inflammation. If the pain is very severe morphia may be necessary, when it is best administered in the form of a suppository. To avoid any straining the bowels should be moved by an enema each morning, and for the same reason the catheter should be passed to draw off the urine.

Stimulants as a rule are not indicated or necessary in the non-septic cases. The acute symptoms as a rule pass off within ten days, but there is a great liability for the disease to remain for a long time in a chronic form, so that our prognosis as to complete recovery should always be guarded.

Chronic Metritis.—Chronic metritis is a term applied to a morbid condition of the uterus, which has been described by different authors under various terms, viz., chronic parenchymatous inflammation (Scanzoni); areolar hyperplasia (Thomas); irritable uterus (Hodge and Gooch); diffuse interstitial metritis (Noeggerath); subinvolution (Sir J. Y. Simpson); hyperplasia of the fibro-muscular tissue of the uterus (Virchow), etc. Clinically, it is applied to a uterus which is "enlarged, painful, and tender" (Herman), and is one of the most common, if not *the* most common, diseases of the female sexual organs.

The essential pathological change in chronic metritis is a great increase in the connective tissue elements, not localised as in fibrous tumours, but diffused through the whole organ. This may be the result of a previous acute inflammation, but only very rarely is this so. In the great majority of cases it is the result only of long-standing congestion, and is thus analogous to the fibroid changes produced in other organs under similar conditions, *e.g.* cirrhosis of the liver.

Subinvolution of the uterus and chronic metritis are by some authorities considered to be the same pathological condition, but while subinvolution is undoubtedly a very frequent cause, and in many cases the starting-point of the irritable, painful uterus which we designate under the term chronic metritis, the two morbid conditions are, we believe, quite distinct.

Subinvolution may be present to a very considerable degree, and cause no symptoms except an increase in the amount of the menstrual discharge.

Chronic metritis is always associated with much pelvic pain, dysmenorrhœa, and generally by ill-health, in addition to menorrhagia. It may also occur in women who have never borne children.

Etiology.—Chronic metritis may sometimes, as has been stated, be but the chronic stage or result of an antecedent acute inflammation of the fibrous tissue of the uterus. In but a very small proportion of the cases, however, where the disease is present will we be able to get a history of any acute symptoms occurring much more frequently in parous than in nulliparous women; we will find very often that its onset

is in some way associated with a previous labour or abortion; when we may conclude it had its starting-point in an imperfect involution of the uterus, or was due to some chronic infective process, the real nature of which we still imperfectly understand.

This absence of any acute stage is very characteristic of inflammation generally in connection with the female sexual organs. In the great majority of cases of endometritis, endocervicitis, ovaritis, and salpingitis, the inflammation runs a subacute course from the commencement. The same thing is true of pelvic cellulitis and peritonitis.

Displacements of the Uterus; Retroflexion and Congenital Anteflexion.—The presence of tumours in or near the uterus; stenosis of cervix; laceration of the cervix and cervical catarrh; endometritis, and the repeated application of caustics to the endometrium; exposure to cold during menstruation; improper clothing; excessive sexual intercourse; irritation produced by a badly-fitting vaginal pessary, or from an intra-uterine stem pessary, are all causes of chronic metritis, which probably acts by producing long-standing or repeated congestion of the uterine blood-vessels.

Symptoms.—The prominent symptom is pelvic pain, dull, aching, or bearing down in its character, referred to the lower abdomen or sacral regions. This pain, though more or less constantly present, is always worse on excitation, and is greatly aggravated immediately before and during menstruation. Along with this pain in the pelvis are associated other local symptoms, such as menorrhagia, leucorrhœa, and dysmenorrhœa. Painful defæcation and micturition are frequently complained of, and dyspareunia is always present. The patient is generally sterile, or if she becomes pregnant, aborts in the early months. As a result of the prolonged suffering and enforced sedentary habits, the subjects of chronic metritis frequently pass into a state of semi-invalidism, or become markedly neurasthenic. Chronic metritis is said to be responsible for a large proportion of the sofa and bath-chair population—the nervous, debilitated, dyspeptic women who wander from physician to physician or crowd the watering-places during the summer. No disease is more chronic, none is more difficult to treat successfully. In many cases relief is got at the menopause. Too much reliance should not be placed on this, however. The pelvic pain may continue for years after, while the nervous symptoms very frequently become more marked at the climacteric.

Treatment.—Treatment after childbirth and abortion, which is directed towards securing a perfect involution of the uterus, is the best preventive treatment of chronic metritis, the chronic congestion of the subinvoluted uterus being perhaps the commonest factor in the production of chronic metritis.

The treatment when the disease is already present is very unsatisfactory and its results very uncertain, and in carrying it out great patience is necessary both on the part of the physician and the patient. In long-standing cases months may be necessary to effect even an approximate cure.

From what has been said as to the pathology, probable cause, and symptoms of chronic metritis, it will be seen that the treatment must be both local and constitutional. The chief aim of local treatment is to diminish or prevent the uterine congestion which, if we cannot say definitely is the cause, at any rate we know greatly aggravates the condition. This passive congestion is best relieved by the use of the hot vaginal douche and the glycerine tampon.

There are few cases of chronic metritis which will not benefit very markedly by a six weeks' or two months' course of douching and plugging if the treatment is properly carried out. Where possible it should be done by a trained nurse. For the first week the douche only should be used three or four times daily. The time occupied on each occasion should be not less than ten minutes, and the douche nozzle should be introduced well up to the roof of the vagina. The amount of fluid used is of no importance so long as the parts are fomented sufficiently long. The temperature of the fluid should be at first 110° F., and may be gradually increased to 120°. Should the douching be followed by an increase of the pelvic pain or by faintness the temperature must be lowered. To prevent the too rapid escape of the water from the vagina, and so obviate the frequent filling of the douche can, the patient should keep her hand over the vulvar orifice and allow the fluid to escape at intervals. The douche should be taken in the horizontal position if possible, and should be followed by half an hour's rest in the same posture. It is not necessary to add any medicaments to the douching fluid unless there is any vaginitis or cervical catarrh, when the best solution to use is one of corrosive sublimate (1-3000).

After a week's douching in this way pledgets of wool soaked in glycerine, or in an 8 per cent solution of ichthyol and glycerine, should be introduced into the vagina and packed carefully round the cervix. These should be removed in twenty-four hours and immediately after a douche is given. Twelve hours later another douche is administered, and the glycerine plug again introduced. During a course of such treatment the patient should if possible rest in bed all the time; if this is not practicable, then for three or four hours each day flat on her back. The half-hour's rest after each douche should be strictly enforced.

The hot douche depletes the congestion of the uterus by stimulating uterine contraction

and by causing a reflex contraction in the uterine blood-vessels. The glycerine plug acts by abstracting serum from the inflamed tissues, and its use is generally attended by a copious discharge which necessitates the patient wearing a diaper.

In some cases a complete cure will result from such a course of treatment, especially if the disease is of recent origin. In almost all there will be a marked relief from the general pelvic pain and discomfort. Much of the non-success attending the use of the vaginal douche and the glycerine plug in chronic pelvic inflammatory lesions is due to the altogether imperfect and perfunctory way in which they are employed. A patient who has been treating herself at home with no benefit will often improve rapidly if she is put under the supervision of a nurse who carries out the treatment properly.

Blisters over the hypogastrium and iodine applied to the roof of the vagina may be used in intractable cases, while hot fomentations and warm hip baths are beneficial in acute exacerbation of the inflammation which frequently occurs. Where the uterus is heavy and prolapsed, and bearing-down pain is a prominent symptom, a soft vaginal rubber pessary will often give great relief. Applications of caustics to the endometrium and curettage are contra-indicated so long as the uterus remains tender. At a later stage, when bi-manual examination causes no pain, if there are indications of endometritis, the uterus may be curetted with benefit. The best hope of a permanent cure lies in the patient becoming pregnant and going to full time; the natural cure of chronic metritis which takes place in the puerperium being after all the only efficient one.

General Treatment.—All very active exertion, such as cycling, long walks, dancing, etc., should be forbidden. A moderate amount of gentle exercise in the open air is, however, beneficial. The importance of physical rest for at least three hours each day in the recumbent posture has been already referred to. Sexual rest is equally important. Intercourse should be entirely abstained from during a course of treatment. The value of the mineral waters of Kreuznach, Schwalbach, Wiesbaden, etc., on the Continent, and those of Woodhall Spa and Harrowgate at home, in the treatment of metritis, as well as other chronic inflammatory pelvic lesions, is beyond doubt. It is, however, pretty generally admitted that much of the benefit which so often follows a visit to one or other of these places is a result more often due to the rest—physical, mental, and sexual—obtained there than to the specific qualities of the waters themselves.

Constipation is a common cause of pelvic congestion in the female, and should be carefully guarded against. In correcting it, salines are specially useful in the disease under notice. Epsom salts and Carlsbad salts, or such mineral waters as Hunyadi Janos and Apenta, are well adapted to the majority of cases. They should be given well diluted in the morning half an hour before any food is taken.

Ergot, potassium iodide, mercury, and arsenic are drugs which have all been vaunted as remedies from time to time in the treatment of chronic metritis. With the exception of ergot, their effects in improving the condition are very doubtful. Ergot in many cases does do good. In others, by increasing the normal contraction of the uterus, its use may be attended with an increase of the pain. To get results from the use of ergot it is important to use only freshly made preparations. Much of the ergot which is dispensed is quite inert owing to decomposition.

Careful dieting and tonics are, of course, of great value when the general health is impaired. Alcohol is generally harmful, and should be used sparingly where, owing to extreme debility, it seems necessary. The Weir-Mitchell treatment of enforced rest in bed, isolation from friends, full diet, massage and electricity, is specially adapted to those cases of neurasthenia and hysteria dependent on chronic metritis. In long-standing cases, where all other measures fail, total extirpation of the uterus may be necessary to give relief.

Chronic Endometritis.—Chronic inflammation of the mucous membrane of the body of the uterus is, like chronic metritis, rarely the consequence of an acute affection, but results more frequently from prolonged hyperæmia, and is subacute from its commencement.

Several varieties have been described by pathologists which are difficult in most cases to differentiate clinically. The glandular, where the glands are much hypertrophied and proliferated; the interstitial, where there is great hyperplasia of the connective tissue elements only of the mucous membrane; a mixed form, where both glands and connective tissue are involved. Endometritis fungosa is a rarer variety than these. Here the mucous membrane seems to consist largely of dilated blood-vessels. Endometritis exfoliativa is characterised by a shedding of the endometrium, sometimes in the form of shreds, sometimes of a complete cast of the uterus. It is the same as membranous dysmenorrhœa. Finally, there is an atrophic form of endometritis met with after the menopause in old women, and which is of great importance clinically, from the fact that the foul-smelling discharge which characterises it may often lead to the suspicion of intra-uterine malignant disease.

Etiology.—The causes of chronic endometritis are the same as those mentioned as operating in producing chronic metritis, with which it is very often associated, though in the majority of cases it occurs as a separate affection. It is

found both in parous and nulliparous women, though much more frequently in the former than in the latter; and, as above stated, it occurs in one form in old women subsequent to the climacteric. Scrofula, tuberculosis, syphilis, anæmia, rheumatism, and gout are said to be predisposing causes. This is somewhat difficult of exact proof, but it is equally certain that those cases associated with a tubercular or rheumatic diathesis are the most intractable. Gonorrhœa accounts for a small proportion of cases.

Symptoms.—The symptoms which characterise it are: (1) Menorrhagia. In but few cases is this symptom absent. It may manifest itself by an increase and prolongation, or too frequent recurrence of the menstrual flow. In very rare cases the menstrual flow may be lessened. In these the diminished discharge is of a dirty brown colour, and is much more offensive than normal. (2) Inter-menstrual discharge. This may be leucorrhœal, sanguinolent, or muco-purulent, and is often so acrid as to cause excoriation externally, or be the cause of intolerable pruritus vulvæ. (3) Dysmenorrhœa. This is not a constant symptom by any means, and is probably only present in those cases where there is also some degree of metritis. (4) Pain in the back is commonly present, and is often the most distressing symptom. (5) Endometritis is also a common cause of sterility, and perhaps the most frequent cause of abortion.

Physical Signs.—On bimanual examination the uterus is felt to be enlarged and tender on pressure. The sound passes into the uterine cavity for a greater distance than the normal 2½ inches; its introduction causes pain and frequently a small amount of hæmorrhage. In rare cases the irregularities of the mucous membrane, as in endometritis fungosa, may be detected by the sound. The speculum shows a watery discharge, which has an alkaline reaction, coming from the cervix. Endometritis of the body is, however, generally accompanied by cervical endometritis, and the watery discharge is not often seen alone. More commonly there is a thick, tenacious mucus which results from the cervical affection. Endometritis is very common in a uterus which is retroflexed.

Differential Diagnosis.—Small polypi—mucous and fibroid—early malignant disease—carcinoma, sarcoma, and deciduoma malignum—and the retained products of conception—may all simulate chronic endometritis in their symptomatology and physical signs, and have to be differentiated from it.

This can only be done in many cases by dilatation of the cervix and exploration of the uterine cavity by the finger, or the examination under the microscope of scrapings taken by the curette. In connection with the latter the large amount of embryonic connective tissue which is present in the normal mucous membrane must be kept in mind, or many cases of simple interstitial endometritis will be mistaken for diffuse sarcoma.

Treatment.—Constitutional treatment, if indicated, will be along the same lines as the constitutional treatment of chronic metritis, and need not be repeated here. Local treatment by douching and vaginal tampons of glycerine may, in mild cases, be all that is necessary to effect a complete cure. In others we may apply iodine or some stronger caustic to the endometrium by means of a dressed sound. In the majority a thorough curettage and removal by this means of the diseased endometrium is the best and at the same time the speediest treatment. To be successful, this must be done thoroughly after the cervix has been well dilated. The curetting is generally followed by the application of pure carbolic acid or a mixture of carbolic and iodine to the interior of the uterus.

If done thoroughly and in suitable cases, curettage of the uterus is one of the most satisfactory of gynecological operations. The simplicity of the procedure has, however, led to the great abuse of this operation. (For further details of this operation, *see under* "CURETTAGE," vol. ii.).

Chronic Cervical Catarrh; Cervical Endometritis; Endocervicitis.—This is a chronic inflammatory process affecting the mucous membrane lining the cervical canal, and by many is said to be the most frequent disease of the female pelvic organs. It is frequently associated with corporeal endometritis, but occurs as a separate affection. It is also much more frequently met with in parous women than in nulliparæ.

The disease begins in the glands of the cervix, which in the early stages are found swollen, hypertrophied, and secreting very freely. Later, the mucous membrane itself becomes greatly thickened by proliferation of the areolar tissue and congestion of its blood-vessels. Exfoliation of the columnar superficial epithelium of the cervical mucous membrane also takes place, and a single layer of delicate embryonic epithelium takes the place of that shed. As a result of the great hypertrophy and congestion of the mucous membrane it is thrown into numerous folds, and may extend outwards from the external os over a considerable extent of the vaginal surface of the cervix, the superficial squamous epithelium of which gives place to this new formation. The minute folds into which this hypertrophied membrane is thrown gives it to the naked eye a granular appearance; and, as the thin epithelial covering allows the subjacent vascular tissue to shine through, the appearances presented on examination by means of the speculum are very similar to those of an ulcer, for which condition they were long mistaken. The red,

raw-looking patches are in reality newly formed glandular secreting surfaces, and result from an ectropion of the cervical mucous membrane. True ulceration of the womb is one of the rarest of diseases.

The inflammatory process is not confined to the glands and mucous membrane, but extends also to the connective tissue of the cervix, so that in long-standing cases the cervix becomes much enlarged and thickened. Finally, it should be stated that the mouths of the inflamed glands frequently become obstructed, the secretion becomes inspissated, and retention cysts are thus produced. These are felt as hard, pea-like nodules, and constitute the so-called Nabothian follicles, and account in many cases for much of the enlargement and irregular shape of the cervix in cervical catarrh. On incision of these follicles, as much as a teaspoonful of thick, glairy mucus may escape. When the glands are extensively involved we get a condition which has been described as cystic degeneration of the cervix.

Etiology.—Laceration of the cervix produced in parturition is undoubtedly the most important cause of cervical catarrh. As a result of the tear, the mucous membrane is exposed to friction and irritation which leads to inflammation. It may be secondary to a vaginitis, the inflammation spreading upwards, or be an extension downwards of a corporeal endometritis. All the causes, therefore, of endometritis are also causes of cervical catarrh. It is also very commonly associated with backward displacements of the uterus. In nulliparous patients, gonorrhœa is not an uncommon cause.

Symptoms.—In a simple, uncomplicated case leucorrhœa is the outstanding symptom; pain and weakness in the back, irregular menstruation, and sterility are also generally present. To these may be added the symptoms of endometritis, as the two conditions so frequently occur together. Formerly, it was supposed that many reflex disturbances, such as dyspepsia, neuralgias, convulsions, persistent salivation, profuse salivation, neurasthenic conditions, etc., had their origin in cervical catarrh. Now, however, it is generally believed that the importance of this condition in producing such symptoms has been greatly overestimated, and that if they do occur secondary to pelvic disease the cause is to be sought for rather in an accompanying metritis or diseased state of the appendages than in the cervical affection.

Diagnosis.—The examining finger, on vaginal examination, feels the enlarged, thickened, and generally lacerated cervix, scattered over which are the prominent Nabothian follicles. The everted mucous membrane is soft and velvety, and bleeds readily. The vagina is unusually moist from the excessive secretion. The speculum shows the fissured os surrounded by a red granular surface which looks like an ulcer, the true nature of which has been already stated. If the everted lips of the torn cervix are approximated by means of tenacula the greater part of this red surface will disappear and the extent of the laceration become more evident. A thick layer of tenacious mucus generally covers the inflamed surface.

Differential Diagnosis.—The leucorrhœa resulting from cervical catarrh has to be differentiated from that caused by vaginitis and corporeal endometritis. Examination by means of a Sim's speculum will, as a rule, be all that is necessary in the case of the former. Menorrhagia, with increase in the length of the uterine cavity, tenderness, and undue vascularity of the interior discovered on passing the sound, indicate the presence of endometritis. Of much greater importance is the differential diagnosis from early carcinoma of the cervix. This may be exceedingly difficult, for it has to be remembered that the simple condition not infrequently passes slowly into malignancy. Where there is any doubt, no time should be lost in excising a portion of the diseased tissue and having it examined microscopically.

This is really the only certain way of coming to a correct diagnosis in an obscure case. The age of the patient cannot be relied on as affording any reliable information. Cancer is often found in young women. The induration and distortion of the cervix produced by inflammation may both be closely simulated by an early malignant infiltration. Pain, too, is frequently absent in early cancer. In the later stages of malignant disease, when the tissue has begun to break down, there should be, of course, no difficulty. But we cannot afford to wait for this before forming a definite diagnosis, for it is only if recognised very early that malignant disease of the cervix offers any chance of successful treatment. (*See also* "MALIGNANT DISEASE OF THE UTERUS," p. 363.)

Treatment.—Constitutional treatment here, as in other pelvic diseases, is of great importance, and should never be neglected. The patient should be encouraged to take outdoor exercise, and very often a change of air will be beneficial. Tonics do good, and a strict attention to the diet and action of the bowels are necessary here as in endometritis. Complete sexual rest should be enjoined.

Locally, astringent douching of the vagina is of great value. The best astringent douche is corrosive sublimate solution, 1-3000; or 1-2000 sulphate of zinc (℥j. to the pint) or copper sulphate (℥ij. to the pint) may be used instead. The temperature of the fluid used need not be over 112° F. Such a douche in a very short time has a marked effect on the leucorrhœa, and causes a shrinking and gradual disappearance of the catarrhal patches on the cervix. In nulliparæ, if the os externum is narrow, it should be notched to permit the escape of the

mucus and allow the astringent fluid to reach the inflamed surface of the cervical canal. Retention cysts in the cervix should be punctured and their contents evacuated. The glycerine plug is useful here also. In many cases of cervical catarrh no further treatment than this is necessary. If, however, in spite of the astringent douches the leucorrhœa continues, direct application of pure carbolic or iodine may be made to the cervix. This should be done thoroughly once, and not repeated at intervals as was formerly done. Failing improvement after a single cauterisation, it is better to have recourse to operation. This may consist of a thorough scraping of the diseased surface, its removal by Schroeder's method, repair of the laceration by Emmet's operation, or, if there is great hypertrophy and induration, amputation of the vaginal portion of the cervix.

SUBINVOLUTION OF THE UTERUS.—The weight of the uterus after delivery is from one and a half to two pounds, and its length is about six inches, the fundus reaching as high as the umbilicus. In from four to six weeks, as a result of certain processes of absorption and transformation of tissue, the exact nature of which is still very imperfectly understood, it is returned to its normal size and weight and position in the pelvis which it occupied before pregnancy. The retardation or arrest of this process of involution after premature termination of pregnancy or delivery at term is known as subinvolution, a term first employed by Sir J. Y. Simpson.

Etiology.—The physiological process of involution is brought about by the diminished blood-supply to the uterus resulting from the intermittent contractions and permanent retraction of its muscle fibres after delivery. The causes of subinvolution are to be found, therefore, in conditions which tend to produce vascular engorgement, or which will interfere with the normal contraction of the uterus. First in importance, therefore, is the occurrence of pelvic inflammation. In all such cases, involution is delayed and always arrested before the process is complete. The presence of pelvic tumours, backward displacements of the uterus, chronic constipation, hepatic and cardiac disease, and resuming the erect posture and active work too soon after delivery, all operate in the same way in retarding or arresting complete involution by causing congestion of the uterine vessels. Imperfect uterine contraction after labour is met with in cases where there has been undue hæmorrhage during delivery, where the labour has been unduly prolonged, in cases also where portions of the membranes or placenta have been left behind, and where there is retained blood-clot. An over-distended bladder may interfere with normal involution. Finally, imperfect contraction of the puerperal uterus is frequently met with in those cases where the normal stimulant to such contraction is not forthcoming owing to the patient not suckling the child. Premature labour, plural births, parturition late in life, chronic debilitative disease, such as phthisis, are often alleged but much less certain causes of subinvolution.

Diagnosis and Symptoms.—During the lying-in period, observations should be systematically made as to the position of the fundus and size of the uterus. An early recognition of any arrest or cessation of the normal diminution is of the utmost importance, as appropriate treatment at this time may avert much subsequent suffering and discomfort. Considerable variations occur, but for practical purposes it may be stated that the fundus uteri on the day after delivery will be found at or slightly above the level of the umbilicus; on the third and fourth days, just below the umbilicus; on the fifth and sixth days, from two to three fingers' breadth below the umbilicus; on the seventh and eighth days, midway between umbilicus and symphysis; and on the tenth and eleventh days, at the level of the symphysis. By the end of the sixth or eighth week the process of involution is complete. The parous uterus is always slightly larger than that of a nullipara. Delayed or arrested involution is associated with an increase and prolongation of the bloody lochia, and in those cases where the cause is a piece of retained placenta or membrane the discharge may remain coloured for weeks. The diagnosis of subinvolution is, however, generally not made until long after the patient has passed out of the puerperal state. The symptom which, in the majority of cases, causes the patient to seek advice is an undue loss of blood at the menstrual periods. This, in fact, is frequently the only symptom. In others there is also a feeling of weight and discomfort in the pelvis, or actual bearing-down pains. Leucorrhœa is present when there is endometritis. Rectal and bladder troubles may be complained of, especially when the uterus is displaced backwards. Dysmenorrhœa exists only when there is metritis or congestion of the ovaries.

As regards the reproductive system, it has to be noted that subinvolution tends to cause sterility, probably on account of the diseased state of endometrium, which is so frequently also present. In the event of a fertilised ovum finding a lodgment in such a uterus, there is a great tendency to abortion. The cause of the so-called "abortion habit" is very often to be found in subinvolution.

Physical Signs.—On vaginal examination the cervix will be found enlarged and thickened often at a lower level than normal, the heavy uterus tending to prolapse. Bimanually the uterus is found enlarged and often retroflexed. The sound passes into the uterine cavity more than 2½ inches, and its introduction generally causes pain.

Subinvolution has to be differentiated from the following conditions, in which there is enlargement of the uterus:—

1. *Pregnancy before the Third Month.*—The amenorrhœa and morning sickness, and the soft, boggy consistence of the uterus in early pregnancy, will in most cases be sufficient to enable one to distinguish an early gestation from the hard, subinvoluted uterus. Subinvolution, however, in a lactating woman, when menstruation is in abeyance, may very easily be mistaken for an early pregnancy by one who is not skilled in the bimanual examination of the uterus. When any doubt exists, a definite diagnosis should be withheld for a month, by which time the growth of the uterine swelling and the development of some of the other signs of pregnancy will make the case clear.

2. *Small Fibroid Tumour.*—A small intramural or submucous fibroid which does not produce any distortion in the form of the uterus. Not only an increase in its size may so closely simulate in its symptoms and physical signs subinvolution, as to make a correct diagnosis impossible until the case has been under observation for some time; the hæmorrhage is likely to be more severe in the case of the fibroid, especially when it is submucous or polypoidal.

3. *Chronic Metritis.*—This condition is by many authorities considered to be the same pathologically as subinvolution, but while it is probable that the one condition frequently merges into the other, there is sufficient evidence clinically to warrant their being considered as separate diseases. In subinvolution the uterus, though enlarged, is not tender. In chronic metritis there is much greater pelvic pain, and dysmenorrhœa is always a marked symptom. Menorrhagia is always present in subinvolution, while it may be altogether absent in chronic metritis in its later stages. Finally, we may meet with well-marked metritis in nulliparous women.

Treatment.—From what has been said as to the etiology of subinvolution, it will be seen that preventive treatment is of the first importance. Strict antisepsis is to be observed during labour and in the lying-in period. Care should be taken that no part of the placenta is left in the uterus after labour or abortion. The patient should not be allowed up too soon, and constipation in the puerperium is to be guarded against. As regards the measures to be adopted when the condition is already present, these are all to be directed towards relieving the congestion and engorgement of the uterus, which is always present; and though it is questionable if any treatment will succeed in making the uterus smaller when the puerperal period has passed, still, many of the symptoms may be relieved and a partial cure at any rate effected. General or constitutional treatment is often more indicated than local remedies. Mineral and vegetable tonics, iodide of potassium, ergot, given over long periods, and the mineral waters of Kreuznach, etc., are all useful. In weak, debilitated women, where the general nutrition is poor and little food is taken, cod-liver old and iron are indicated. In all cases a saline aperient draught each morning does good.

Local remedies include the use of the hot vaginal douche—to which some astringent may be added if there is also a relaxed and subinvoluted condition of the vagina and pelvic floor—and the glycerine plug. These remedies have largely replaced the former treatment of applying caustics to the endometrium and the application of leeches to the cervix, and, if properly carried out as directed under the local treatment of chronic metritis, rarely fail to do good. Electricity, used in the same way as in the treatment of uterine fibroids, is also a valuable therapeutic remedy. Under its use the uterus often becomes distinctly smaller, and the menorrhagia lessens if not entirely disappears. When the uterus is displaced, the first step in treatment will, of course, be the correction of the displacement and the retention of the uterus in its normal place by operation or the use of a vaginal pessary. When endometritis is present, the uterus should be curetted; and when the cervix is deeply lacerated and inflamed, attention must be directed to the cure of these conditions. Amputation of the cervix in subinvolution was formerly a popular method of treatment. It has, however, decidedly fallen into disuse. Should a patient with a subinvoluted uterus become pregnant, the greatest care should be taken to prevent an abortion, for it is in the succeeding puerperium that we will look for the only safest cure of this troublesome condition.

Superinvolution of the Uterus.—Very rarely the process of involution after delivery does not stop at the restoration of the uterus to its normal non-pregnant size, but continues, and as a result the organ remains permanently small. To this condition Sir J. Y. Simpson applied the term superinvolution. It is also known as puerperal atrophy.

Etiology.—This is obscure. Prolonged lactation is said to cause it, and, from the relative frequency with which the condition is met with among the poorer classes, this is probably correct. Excessive loss of blood during labour is said to favour its occurrence, and it is more commonly met with in weak, anæmic patients than in those who are well nourished and full blooded. A form of atrophy of the uterus in the puerperium may be caused by an obstruction to its proper blood-supply in the cicatrisation of inflammatory exudations in the broad ligament. This, however, though puerperal, is hardly the same condition as superinvolution.

Symptoms.—Amenorrhœa, relative or complete, and sterility are the only symptoms of this rare affection. The diagnosis is made by

bimanual examination of the uterus, when its small size will be noticed. If the sound is used, it will be found to go into the uterus only for a distance of 2, or it may be only 1½ inches. The prognosis as regards the possibility of a patient with superinvolution becoming again pregnant is decidedly unfavourable, but should always be very guarded. The writer has had under his care a patient whose uterine cavity was less than 1¾ inches in length, and who had not menstruated for more than one day at a time, and for months not at all, who was subsequently delivered of a healthy child at term.

Treatment.—This is very unsatisfactory. The galvanic stem pessary introduced by Sir J. Y. Simpson for stimulating the atrophic uterus to further growth probably does no good, and its use is not free from danger.

A more rational and safer treatment is the direct application of electricity by means of one pole inside the uterus, the other over the abdominal wall.

IV. Non-Malignant Tumours of the Uterus

FIBROIDS 348
Classifications 348
Symptoms 350
Complications 351
Degenerations 352
A. *True* 353
B. *Transition to other Growths* . 354
C. *Cystic Changes in Fibroids* . 354
Indications for Operation . . 354
FIBRO-CYSTIC 355
POLYPI OF THE UTERUS 355
Fibroid 356
Mucous 356
Malignant, etc. 357
ADENO-MYOMATA 357
DIFFERENTIAL DIAGNOSIS . . . 357
TREATMENT 360
Medical 360
Electrolysis 360
Surgical 360
(*a*) *Vaginal* 361
(*b*) *Abdominal* 361
TUBERCULOSIS 362
HYDATIDS 362

SIMPLE TUMOURS, "FIBROIDS," ETC.

"FIBROIDS."—Under this heading are included all those tumours of the uterus in which white fibrous tissue and muscular elements in varying proportions form the bulk of the tumour. It is simply a convenient term to describe a group of pathological growths, for under it are included tumours which have very dissimilar composition, histories, and symptoms. The word fibroid in this article is used in this sense only. Several classifications of fibroids are in use. In one the composition and manner of growth supply the basis, and the growths are described as:—

1. *Fibro-Myomata, or Multiple and Multinodular Fibroids.*—In these tumours the white fibrous tissue predominates over the muscular elements. They are hard, and are usually present in numbers in the same uterus.

2. *Myo-Fibromata, or Uninodular Fibroids.*—These tumours are softer, because they have more muscular tissue in their composition. They should be subdivided into—(*a*) the encapsuled; (*b*) the non-encapsuled.

The encapsuled sub-variety is often many whorled and comparatively hard; while the non-encapsuled consist not of whorls but of an intricate meshwork of muscular and fibrous tissue more or less inseparably blended with the normal musculature of the uterus.

A second classification is based on the position of the tumours with reference to the wall of the uterus, thus:—

(1) Sub-peritoneal: (*a*) sessile; (*b*) pediculated.
(2) Interstitial.
(3) Sub-mucous: (*a*) sessile; (*b*) polypoid.

Frequently in cases of multiple fibroids all these varieties are illustrated.

A third classification has reference to the point of origin of the growth with reference to the region of the uterus—such as fibroids of the body, fibroids of the cervix.

Pathologically the first classification is the most important.

1. *The hard, multiple fibro-myomata* consist chiefly of white fibrous tissue arranged in whorls in the interstices of which lie unstriped muscle fibre cells with elongated nuclei resembling the muscle cells of the normal unimpregnated uterus. The whorls vary in size, from those seen only through the microscope, to others having a diameter of several centimetres. A varying number of whorls packed together, and usually surrounded by a loose musculo-fibrous capsule, constitute a tumour. The blood-vessels ramify chiefly in the capsule, the interior of the growth being almost non-vascular.

The naked-eye appearance of a fibro-myoma is that of a firm white mass, the cut surface of which shows the whorled arrangement of its fibres and has been compared to "white-watered silk."

The capsule really consists of altered and condensed uterine tissue. It is therefore attached loosely to the growth, but intimately to the uterus. This peculiarity often facilitates the enucleation of the morbid from the normal tissues.

The fibro-myomata are probably all interstitial in origin. They commence in the musculature of the uterine wall and grow in the direction of least resistance, being pushed by the contractions of the normal muscle outwards towards the peritoneum or inwards towards the mucous membrane lining the uterus. They may be found deeply embedded in the wall, or covered only by peritoneum or by mucous membrane.

The fibro-myomata, like their component whorls, differ greatly in size. They vary from a minute size up to a huge tumour weighing more than 30 kilogrammes, or upwards of 70 lbs. The numbers vary in each case examined; there may be two or three separate tumours or there may be hundreds.

2. *The Myo-Fibromata.* — *Synonyms*: soft fibroids; uninodular fibroids; monomata; non-encapsuled fibroids.

These tumours appear as pinkish masses of varying degrees of softness embedded in the uterine wall.

They contain a larger proportion of muscular fibres and relatively less fibrous tissue than do the multinodular or hard fibroids. The more abundant the muscular tissue the softer and the pinker the mass. They are also richer in blood-vessels, which permeate their tissues and are not confined to their periphery. These growths frequently have no capsule, they cannot be readily enucleated from the surrounding uterine tissues, and they are usually deeply embedded because they stimulate the normal tissue to over-development.

Some of the uninodular fibroids are so soft that clinically they are mistaken for cystic growths. Their tissues are not arranged in whorls, but from an interlacing network more or less continuous with the normal tissues of the organ.

The life-history is different in the two great classes of fibroids. Both varieties may exist from very early days, but occur most frequently during the child-bearing period of life. The hard fibromata follow closely the life-history of the uterus itself. They develop during its period of functional activity; they frequently enlarge to a noticeable degree before and during the catamenia, becoming smaller during the intermenstrual epoch; they often grow enormously during pregnancy, in correspondence with the remarkable physiological hypertrophy and hyperplasia of the uterus; after parturition they share in the rapid involution of that organ. In rare cases the tumour may even disappear; and, lastly, although the menopause may be long, and indefinitely, delayed in the presence of fibroids, yet when it is established the tumour may, like the uterus, undergo senile atrophy. Lastly, these tumours often dwindle after the artificial menopause caused by ablation of the ovaries and tubes.

The soft myo-fibromata have a life-history entirely independent of that of the uterus. It is true that they are very rare before puberty, but so are other tumours. If a young girl has a fibroid at all it is usually of the soft, solitary variety. They are often unaffected by menstruation; alter little during pregnancy and after parturition; they grow on steadily unaffected by the menopause, and, indeed, often appear for the first time after the reproductive period of the uterus is over. Their growth is unaffected by the removal of the uterine appendages. This independence of growth and other characters show that soft, non-encapsuled fibroids are more nearly allied to malignant growths than are the hard fibro-myomata.

With regard to the destiny of an individual fibroid, it may remain interstitial and evolute, involute, or degenerate *in situ*. It may be gradually pressed towards the peritoneum, and be eccentrically extruded so as to form a rounded mass raised above the general peritoneal surface of the uterus. If the process continue the fibroid may be entirely expelled from the uterus and hang free in the peritoneal cavity. Eventually such a fibroid may be entirely detached so as to be found lying in Douglas's pouch; or it may acquire attachments to various pelvic or abdominal organs. Fibroids attached to the uterus by a long pedicle sometimes cause obstruction of the bowels by strangling a coil of intestine, or they may lead to severe pressure symptoms by falling into Douglas's pouch when the patient stands up. The recognition of this accident is facilitated by the cessation of symptoms when the fibroid is pushed up, and when the patient lies down or assumes the knee-face position.

In another class of case the direction of growth and the uterine contractions may force the tumour concentrically towards the uterine cavity, so that under favourable circumstances it becomes polypoid. The pedicle at first consists of a layer of uterine muscle covered with mucous membrane, but after a time it is formed by attenuated mucous membrane only. Having thus become pedunculated, the growth is called a polypus. At any stage this process of pediculation may be arrested, and the bleeding and other symptoms may become so severe as to destroy life unless relieved by operation.

Thus the submucous fibroid is liable to cause excessive hæmorrhage and discharge owing to the accompanying congestion of the uterine mucosa; it may become infected and undergo sloughing, gangrenous, or suppurative changes. It may undergo an imperfect process of enucleation in the course of which its vitality is lost, its capsule ulcerates, and it is discharged piecemeal, the patient meanwhile developing symptoms of sepsis. The danger is less, and the chance of complete spontaneous expulsion is greater, when the suppurative or other destructive change involves the capsule only. The maximum of danger arises when the substance of the tumour undergoes one of the destructive degenerations. The whole system of muscles connected with the uterus is liable to the development of "fibroids." They have been found in the round ligament, the ovarian ligament, and in the broad ligament, not merely as extensions of a tumour originating in the uterus, but as independent growths.

Symptoms.—(*a*) Symptoms may be due to the tumour itself. These seldom arise so long as its tissues are in good health.

(*b*) The commonest symptoms are due to alterations in the uterus and other organs caused by the tumours, and to secondary changes produced in them.

Hæmorrhage.—The commonest symptom of both hard and soft fibroids is hæmorrhage. It occurs both as menorrhagia and metrorrhagia, and varies from a slight increase in quantity or frequency of the normal loss to dangerous floodings. The hæmorrhage proceeds from the mucous membrane of the uterus, and may be due to its abnormally increased area, to the supervention of endometritis, or to the coexistence of fibroid or mucous polypi. The hæmorrhage is most abundant when the tumour is immediately beneath the mucous membrane, and may be slight, or even absent, when the growth is entirely subserous. So long as the tumour remains in the uterus the hæmorrhage usually observes the monthly rhythm and may be called menorrhagia. Should the tumour be extruded into the vagina, the hæmorrhage, whether slight or profuse, tends to be remittent or continuous and is a metrorrhagia. Hæmorrhage sometimes, but rarely, proceeds from the capsule of the tumour, as in cases of submucous fibroids, where the mucous membrane has been perforated by the growth, so that the tumour presents at the os uteri. In a case recorded by Matthews Duncan the capsule of the tumour had calcified; subsequently this shell cracked, and wounded a large sinus, producing an immediately fatal hæmorrhage.

The amount of hæmorrhage varies greatly in different cases, some patients with a large tumour losing little, while others in whom the growth is small suffer absolute floodings. The character of the growth does not explain these variations, as they occur alike in uninodular, multinodular, and multiple fibroids. Certainly the approach of the growth to the mucous membrane appears to be accompanied by increased hæmorrhage, while the symptom is usually less formidable, and may even be absent, when the tumours are subperitoneal only.

Pain is not so common a symptom in fibroids as in hæmorrhage, and when present is often associated with some morbid change in the tumour, or with injurious effects produced by it on other organs. It is usually described as a dull grinding pain, sometimes as a sense of over-fulness or twisting, and sometimes as colicky or paroxysmal. This intermittent rhythmical pain is most frequent when the tumour is pressing on, or through, the cervix. It is natural that it should thus simulate the pains of the first stage of labour.

Pain may be due to the following causes:—

1. The involuntary extrusive efforts of the uterus by which an intramural growth is forced towards the mucous or the serous surface.

2. The dilatation and stretching of the cervix uteri during the extrusion of a fibrous polypus.

3. The incarceration of a fibroid uterus, or of a pedunculated subserous fibroid, in the bony pelvis, causing pressure on adjacent organs and nerves.

4. Interference of the growth, or its pedicle, with other pelvic or abdominal organs by which morbid changes are caused in them.

5. The sense of weight and bearing down usually present is aggravated into pain during the catamenia. This is most marked in the case of submucous growths, and is then probably due to exaggeration of the normal congestion. The same symptoms may occur even when the growth is subserous, and is then explained by the fact that the swelling due to normal increase of vascularity is hindered by the tension of the capsule.

Any marked increase of pain—or its appearance as a new symptom—demands careful investigation, as it is frequently the first warning of degenerative changes occurring in the tumour (*see* "Indications for Operation," p. 354).

Discharge of a watery or mucoid nature, not blood-stained and not offensive, is a common symptom. It is due to concurrent congestion of the general mucosa of the uterus, or sometimes to over-stimulation of the cervical glands. Purulent, blood-stained, and offensive discharges are usual in sloughing, inflammatory, sarcomatous, and other destructive degenerations, also when there are certain complicating or coexisting diseases, such as purulent endometritis or epithelioma.

Bladder symptoms, especially incontinence or retention of urine, are common in cases of fibroid growths. Cystitis is not common. These symptoms are due to pressure. Retention generally results from pressure of an intra-pelvic mass on the urethra, while incontinence is more commonly caused by pressure of an intra-abdominal growth on the bladder.[1]

Rectal symptoms of fibroids (which are much less common than bladder symptoms) are also due to pressure. Constipation may exist from direct pressure of a growth when intra-pelvic, or from increase of general abdominal pressure in the case of large tumours. Hæmorrhoids are caused by pressure on the veins of the rectum. The patient may also complain of tenesmus and of mucoid discharge from the bowel.

Variations in size may be very slow, as in the majority of the hard fibromata, or relatively rapid, as in the softer variety. Sudden increase

[1] Hydro-ureter, hydro-nephrosis, pyo-nephrosis, and other destructive changes in the kidneys are less common in cases of fibroids than in cases of cancer uteri. They do, however, occur, and generally in cases in which the broad ligaments are invaded by the growths.

in size should always excite suspicion of some untoward change in the growth. The rate of growth is uncertain, as there are no means of fixing the time of its commencement. Each case must be watched carefully and judged on its merits.

The size of the tumour may also vary from time to time spontaneously to a remarkable degree. A decrease may be observed after rest in bed, more frequently after a profuse period, and after parturition, sometimes after a merely exploratory laparotomy.

Sterility is frequent in women afflicted with fibroids, and may be explained in part by the endometritis which is often present, and in part by the frequent complicating diseases of the ovaries and tubes. In another group of cases conception occurs, but abortion or miscarriage supervenes. Lastly, pregnancy may be ended intentionally or accidentally by surgical interference. On the whole, fibroids appear to be more common in sterile than in fertile women.

A venous hum, resembling the souffle heard during pregnancy, may be sometimes recognised in cases of fibroid tumours. Probably it is due to the rushing of the blood in the large sinuses of the capsule.

One or more of these symptoms, such as hæmorrhage, pain, and discharge, may combine to present us with a typical clinical picture.

In the early stages of a fibroid's career there may be no distinctive symptoms, and the small tumour may be discovered by accident when the patient is examined for a supposed displacement, or to ascertain whether an early pregnancy exists. In some cases this absence of marked symptoms persists, and the patient remains in good health, even though a large tumour is present. In many cases, however, the patient gradually becomes anæmic, with pale skin, white lips and mucous membranes, the ears look as if modelled in pale yellow wax. She is breathless on exertion, and complains of faintness, palpitation, and dyspepsia. If the disease continues unchecked and pain coexists with hæmorrhage, the woman is still more wretched. She suffers from loss of sleep and rest, from want of appetite, and from nervous debility. At last, even if the tumour undergoes no degenerative change, and no serious complication appears, the patient becomes a chronic bed-ridden invalid.

Complications *of Fibroids.* — Most authors agree that the percentage of complications in cases of fibroids is large.

Twambly (*Boston Medical and Surgical Journal*, May 1897) estimated that 50 per cent of the cases of interstitial fibroids were accompanied by disease of the Fallopian tubes.

Meredith, analysing Tait's cases (*Brit. Med. Jour.*, 1890), found 54 per cent tubal disease and 46 per cent chronic ovaritis. Lately, three analyses gave the following results:—

Dr. Ch. Noble. 218 Consecutive Cases.		Dr. Ch. Cullingworth. 100 Consecutive Cases.		Mrs. Scharlieb. 100 Consecutive Cases.	
Hydrosalpinx	13	Hydrosalpinx	5	Hydrosalpinx	4
Pyosalpinx	8	Serious adhesions	12	Pyosalpinx	1
Hæmatosalpinx	1	Dilated ureters and hydronephrosis	1	Adhesions	6
Salpingitis	7	Dilated ureters, hydronephrosis, suppurative pyelitis, etc.	1	Cysts or cystomata	23
Ectopic gestation	3	Ectopic gestation	1	Parovarian cysts	2
Ovarian cysts	27	Twisted pedicle	2	Thrombosis, before operation	2
Ovarian abscess	1			Varicose veins of pelvis	4
Parovarian cysts	2			Fibroma of Fallopian tube	1
				Papillomata of appendages	1
				Incarceration of tumour	5
				Pregnancy	1
				Appendicitis	1

These pelvic complications may bear relation to fibroids as being coincidents or effects—probably they are most frequently effects. The tumour interferes with the pelvic circulation and frequently dislocates the organs. When it is situated in the fundus or the posterior wall of the uterus it may cause retroversion or retroflexion, and the appendages are thus dragged out of their normal position.

Pregnancy is one of the gravest complications of fibroids, partly from the enhanced risk of mechanical obstruction during parturition, partly from the increased risk of post-partum hæmorrhage, and partly from the danger of bruising or other injury impairing the vitality of the tumour (*see* "Indications for Operation," p. 354).

Adhesions are a fairly common, and may be a very formidable complication. They occur after peritonitis however excited, (1) by extension of inflammation from the tumour itself, or (2) in consequence of inflammation of neighbouring organs, and (3) in cases where treatment by electrolysis has been tried. Adhesions are common, tough, and abundant where the sharp electrode has been plunged into the tumour or into the normal uterine tissue in which it is embedded.

Phlebitis, phlegmasia dolens, thrombosis, and embolism occur both during the existence of fibroids and after operation for their removal.

These complications are due to pressure on the veins, and other interference with the circulation; to septic absorption when the tumour has undergone degenerative changes; to feebleness of the heart's action; and to altered constitution of the blood, especially in cases where there has been much depreciation of vitality as a result of excessive hæmorrhage, severe pain, etc.

Parotitis is not common, but when it occurs in connection with degenerate fibroids it is usually suppurative, as in other cases of abdominal disease or operation.

Heart disease is a fairly common complication of fibroids. It especially occurs as dilatation, hypertrophy, fatty and brown degeneration of the musculature.

Fatty enlargement of the liver has been observed, and is noted by Dr. Bantock.

Derangements of the urinary apparatus are among the most serious and common of the complications of fibroids, and are most likely to occur when there is incarceration of the uterus and tumour, or of the tumour alone, within the bony pelvis, especially when the broad ligaments are invaded. The symptoms then resemble those of the incarcerated retroverted gravid uterus (vol. viii. p. 267).

Retention and incontinence of urine have been already mentioned, and although secondary changes in the kidneys, due to disease of the bladder or to pressure of a fibroid on one or both ureters, are not so common as in cancerous disease of the pelvic organs, they occur with sufficient frequency to cause great anxiety to the surgeon.

Pressure on the intestines, especially on the *rectum*, already mentioned (p. 349).

Pseudo-sciatica and other neuralgic pains due to pressure on the pelvic nerves ought to draw the surgeon's attention to the condition of the pelvic viscera. Not infrequently a pain in the course of one of the pelvic nerves, which has resisted diagnosis and treatment, will be explained by the discovery of a fibroid or other tumour. Relief will follow treatment based on a correct diagnosis.

DEGENERATIONS *of Fibroids*.—This term is limited to morbid alterations in the tissues of the tumours, and does not apply to coexisting disease. Thus epithelioma of the cervix is a complication but not a degeneration of a coexisting fibroid situated in the body of the uterus.

The following table appears to include all the known forms of "degeneration," and is satisfactory in so far that the classification is fairly accurate both pathologically and clinically:—

A. *True Degenerations*—
1. Simple aseptic necrobiosis.
2. Suppuration.
3. Sloughing.
4. Œdematous change and myxomatous degeneration.
5. Calcareous degeneration.
6. Fatty degeneration.

B. *Transition to other Forms of Growth in Fibroids*—
1. Sarcomatous growth.
2. Carcinomatous growth.
3. Teleangiectatic growth.
4. Lymphangiectatic growth.

C. "*Cystic*" *change in Fibroids*—
This is not a pathological entity, but includes cystic development caused by—
1. Cavities (pseudo-cysts) due to the softening produced by necrobiosis, suppuration, œdematous and myxomatous change, or as the result of hæmorrhage into soft fibroids or sarcomata. These cavities have no epithelial lining.
2. Cavities formed by teleangiectasis or lymphangiectasis, lined by endothelium.
3. True cystic fibroids, in which the cysts are formed by glandular elements within the tumour and are lined by columnar epithelium.

Under these headings it is possible to group the degenerations noted in practice by several authors, and it is to be hoped that a more careful and systematic examination of all tumours removed by operation or post-mortem will lead in time to a fuller understanding of these important changes, and consequently to wiser advice and better treatment.

	Noble. 208 Cases.	Martin. 205 Cases.	Cullingworth. 100 Cases.	Scharlieb. 100 cases.
A. *True Degenerations*—				
1. Simple necrobiosis	12	...	15	
2. Suppuration	...	10	3	2
3. Sloughing	...	...	...	3
4. Œdematous and myxomatous	5	11	27	5
5. Calcareous	5	3	1	2
6. Fatty	...	7		
B. *Transition to other Forms of Growth in Fibroids*—				
1. Sarcomatous	2	6	1	6
2. Carcinomatous				
3. Teleangiectatic	...	2	...	1
4. Lymphangiectatic				
C. *Cystic Changes in Fibroids*—				
1. Pseudo-cysts (cavities due to softening)	5	1	5	7
2. Cavities formed by teleangiectasis, etc.				
3. True cystic fibroids				

The above table is evidently very imperfect, as, for instance, none of the observers have

sufficiently indicated the varieties of cysts; it is published only in the hope that in future classification may become more accurate.

A. *True Degenerations*

1. *Simple aseptic necrobiosis* occurs in tumours in which the blood-supply is absent or deficient, but to which septic germs have not acquired access. The tumours in such cases have probably been badly supplied with nourishment from the first, and consequently a comparatively slight interference with the vessels stops the supply altogether.

2. *Suppuration.*—The change may depend on inflammation commencing in the tumour itself, or on extension to it of morbid changes from neighbouring organs, such, for instance, as the vermiform appendix, the ovaries, or tubes. In this degeneration some micro-organism plays a part, for instance the streptococcus, staphylococcus, gonococcus, the bacillus of tubercle, or the bacillus coli communis. The process must not be considered identical with sloughing or gangrene.

3. *Sloughing* may attack fibroids in many ways by interference with their vascular supply, mechanical injury, or the invasion of septic organisms. These conditions, singly or combined, are seen under the following circumstances among others:—

(1) As a result of bruising or other injury during childbirth, either from the muscular contractions necessary to the process, from pressure of the child's head, or as the result of assistance, manual or instrumental.

(2) As one effect of puerperal septicæmia—the micro-organisms or their toxins obtaining access to the tumour and interfering with its vitality.

(3) As the result of operations intended for the removal of the tumour.

(*a*) Submucous enucleation, where the capsule has been injured and the uterus left perforce or designedly to expel the tumour.

(*b*) Electrolysis, where the cauterising effects of the intra-uterine electrode injures the endometrium and opens the way for pathogenic micro-organisms, or compromises the vitality of the tumour by interfering with its supply of blood. In cases where a sharp electrode is plunged through the endometrium and the intervening tissues into the tumour, the injury is designed to produce a local disintegration of tissue which in so badly vascularised a tumour is likely to lead to generalised destructive changes.

(*c*) Result of extension from neighbouring organs, as in cases of disease of the pelvic and adjacent abdominal organs; especially in suppurative diseases of the ovaries, tubes, and appendix vermiformis. In some cases where no evident disease can be found it seems probable that infection by the bacillus communis coli may occur.

(*d*) Infection through the blood-supply, as in the acute exanthemata, influenza, etc.

(*e*) From incarceration of the tumour and uterus (resembling the condition of incarcerated retroverted gravid uterus), or from the incarceration of a pedunculated subperitoneal fibroid which has slipped behind the uterus and gives rise to urgent and distressing symptoms resembling those of strangulation of a viscus.

4. *Œdematous and Myxomatous Degeneration.*—Probably the term myxomatous is not always correctly used. In the majority of cases the fluid contained in the interstices of the tumour is serum rather than mucus. In some tumours, no doubt, there is myxomatous degeneration, but this is a different sort of change, and should not be confounded with an ordinary œdema (*see* Stanmore Bishop's *Uterine Fibro-Myomata*, 1901).

5. *Calcareous degeneration* occurs (1) in the capsule (calcification *en coque* of Matthews Duncan), also (2) throughout the tumour, commencing from the centre and extending to the circumference. The deposition of salts, chiefly of phosphate of lime, may be so general that the sawn surface of the tumour presents a uniformly hard surface capable of taking a high polish, or it may be partial, so that the cut surface presents a coral-like appearance, the calcified portions persisting while the fluid or soft solid which filled the interstices has disappeared. By calcareous degeneration are formed those curiosities of ancient medical literature known as "womb stones."

Calcification is due to deficiency in quantity or quality in the supply of arterial blood and is a true degeneration, such as is seen in the case of calcification of arteries. It is worthy of note that this change appears to be common in fibroids of long standing that have been extruded from the peritoneal surface of the uterus and have maintained a precarious existence by adhesion to other organs.

6. *Fatty degeneration* may be primary, and in a sense "physiological" and curative, as when the tissues of a "fibroid" share in the physiological involution of the uterus and other pelvic organs after parturition and after the menopause, when some degree of fatty change appears to precede involution.

Fatty degeneration of fibroids may also be secondary and purely pathological; for instance, when it occurs as part of, or in the course of, other degenerations, such as the inflammatory, the necrotic, and the sarcomatous.

7. Lastly, some cases of gangrenous and other necrotic degenerations of fibroids are

recorded as having occurred in women in good health and in whom no cause of injury to the tumour could be discovered. (*See* Cullingworth, *Journal of Obstet. and Gynæcol.*, January 1902.) The explanation is difficult; probably the blood-supply to the tumour was inadequate for its nourishment, and infection from some unrecognised source reached the dying tissues.

B. *Transition to other Forms of Growth in Fibroids*

1. *Sarcomatous change* is recorded by all observers, and appears to be commoner than was formerly thought to be the case. It occurred twice in 208 cases reported by Ch. Noble, six times in 205 cases reported by A. Martin, once in 100 cases reported by Dr. Cullingworth, and six times in 100 cases reported by Mrs Scharlieb. The difficulty seems to be to determine whether the connective tissue becomes replaced by sarcoma cells, or whether there is a sarcomatous invasion of the muscle fibre cells (Discussion Obst. Society of London, 1898, *Obst. Trans.*, pp. 178 and 227).

2. *Carcinomatous change* is rare in fibroids, and from histological considerations is less to be expected than the sarcomatous. Still, epithelial structures have been observed in fibroids, and are probably due in some cases to the nipping off by the growth of portions of the uterine glands which normally pass deep into the tissues. In other cases the epithelial tissues may be derived from aberrant remnants of the Wolffian body or the Müllerian duct.

The association of fibroids of the uterus with carcinoma or epithelioma of the body or cervix, though not uncommon, appears to be accidental. Of course the existence of the one disease is no bar to the development of the other, while both are common.

3. *Teleangiectatic Change.*—In some cases not only are the ordinary vessels in the capsule and the uterine vessels themselves of enormous size, but the capillaries in the tumour appear to have become transformed into a network of sinuses somewhat like that existing in a pregnant uterus. They have no definite walls except a lining of endothelium, and are adherent to the muscular bundles. The portions of tumour that are thus degenerated are said to look "like a sponge engorged with blood." They furnish a loud venous hum such as is heard over the placental site, and may tend to obscure the differential diagnosis of the tumour.

4. *Lymphangiectatic changes* also occur. The lymph spaces between the bundles of fibres become dilated and filled with lymph, giving rise to one variety of fibro-cystic tumour.

C. "*Cystic*" *Change in Fibroids*

The term "cystic" fibroid has been applied to many different forms of cavities within fibroid tumours. The true cyst is that which is lined with columnar epithelium and which originates in some granular elements enclosed within the tumour such as portions of uterine glands, or possibly from aberrant remains of fœtal structures. These cysts contain a secreted fluid, may attain a large size, and may be multiple. The cavities or pseudo-cysts which are formed by fatty, necrotic, myxomatous, or suppurative changes may also attain a large size, but their walls are ragged, irregular, and without epithelial lining, while their contents may be soft, necrotic, or gelatinous material, altered blood, or pus, according to the nature of the change which produced the cavity. The cavities which occur in teleangiectasis and lymphangiectasis have been described above.

Indications for Operations on Fibroids.—These will be discussed under the following headings:—

1. Hæmorrhage.
2. Pressure symptoms.
3. Rapid increase in size of the tumour.
4. Coexisting pregnancy.
5. Coexisting complications (such as disease of the pelvic organs or other viscera).
6. Sloughing and other degenerations of the tumour itself.
7. The patient's social position and moral considerations.

1. *Hæmorrhage* was long considered almost the only indication for operations on fibroids. Operations, except for removal of fibroid polypi already extruded into the vagina, were seldom proposed until the profound anæmia and general debility were so marked that, operation or no operation, the patient was nearly sure to die.

Hæmorrhage may kill the patient directly, as in the case recorded by Matthews Duncan, but this is not common. Women have a remarkable, but not unlimited, power of making fresh supplies of blood. Frequently the fatal result is indirect, the woman being incapable in her exhausted and anæmic state of repelling attacks of illness or of convalescing from them. She is also liable to serious cardiac and vascular depreciation.

2. *Pressure symptoms* are most formidable when the tumour is incarcerated in the pelvis They resemble those which occur in a case of retroverted or retroflexed gravid uterus. There is frequently retention of the urine due to pressure on the urethra or on the neck of the bladder. From time to time a little paralytic dribbling may occur, but the bladder is not relieved and requires the use of the catheter. Pressure on one or both ureters as they cross the pelvic brim may lead to hydro-ureter, and to further serious mischief in one or both kidneys (hydro- and pyo-nephrosis). Pressure on the rectum is seldom as painful or as prohibitive to function as in the case of pressure on the bladder, but it causes pain, constipation, tenesmus, con-

gestion of the rectal mucous membrane and hæmorrhoids. Pressure on the pelvic nerves leads to various neuralgiæ, and especially to one form of pseudo-sciatica. Indeed, pain in the course of this nerve and its branches should always lead to careful examination of all the pelvic organs. Pressure on the ovaries and tubes, especially when they are prolapsed, produces a peculiarly sickening and intolerable pain. It also probably leads to many of the diseased conditions of these organs which are found in connection with fibroids.

When the fibroid has risen into the abdomen, pressure on the bladder more frequently causes incontinence than retention, and is often a great source of trouble and annoyance. The presence of a large fibroid in the abdomen affects the bowels, the liver, and other organs by the limitation of space and distortion of relations. Flatulent distension is sometimes specially distressing.

3. *Rapid increase in the size of the tumour*, especially when combined with pain, is one of the most important indications for operation. These symptoms arise in cases where the tumour is undergoing necrotic, suppurative, œdematous, myxomatous, cystic, sarcomatous, or carcinomatous change. The rapid increase in size is generally accompanied by constitutional symptoms, such as rise of temperature and pulse, with other signs of distress, and when the tumour is gravely degenerate the classical signs of septicæmia will usually develop.

4. *The coexistence of pregnancy*, though it does not invariably necessitate operation, is a very grave complication, the mortality being estimated at 36 per cent, including the cases in which spontaneous miscarriage occurs. Pregnancy must be considered both as an absolute and as a relative indication for operation. It is an *absolute* indication when, from the position of the tumour, delivery *per vias naturales* is impossible owing to mechanical obstruction, also when severe pressure symptoms develop. In some of the cases of retroverted gravid uterus plus fibroids with pressure symptoms, it may be possible to push the mass out of the pelvis, and, by posture aided by a suitable pessary, to retain it above the brim until the danger of incarceration is passed. Such an attempt at reposition must be made with great gentleness and caution, for many surgeons have reported cases where a rapidly fatal result followed successful attempts at reposition—death under these circumstances being caused by contusion of the tumour or by torsion of its pedicle.

The coexistence of pregnancy and fibroids is a *relative* indication for operation, when mechanical obstruction is not feared, but where, from the position of the tumour, or from its size, the risks of parturition, whether premature or at full term, are great. The risks of hæmorrhage before, during, and after delivery must be considered; also the risks of injury to the tumour and consequent increased liability to septic infection.

Operations on fibroids during pregnancy do not always involve premature delivery nor the loss of the fœtus. In many cases of subserous fibroids removal can be effected with little or no injury to the uterus, and the mere fact of opening the abdomen seldom interferes with the normal course of gestation. In some cases hysterectomy may be indicated early in pregnancy, but only when the mother's life is obviously in danger. In another group of cases the operation can be postponed until full term. The child is then delivered by Cæsarean section, the uterus being removed or retained as seems most desirable in each case. *See* valuable papers by Cullingworth and Bland Sutton.

5. *Other complications*, some such as appendicitis, suppuration of ovaries or tubes, ovarian cysts, extra-uterine gestation, carcinoma of the body or cervix, are clear indications for operation in themselves apart from the coexistence of fibroids. Some complications, such as displacement of the uterus, phlebitis, and thrombosis, heart-disease, insanity, and other nervous disorders, need careful consideration. Such factors as the patient's position in life, her ability to earn her living, and her general health, would count for much in these more doubtful cases.

6. *Degenerations of Fibroids.*—Of these the sarcomatous, suppurative, necrotic, gangrenous, cystic, and œdematous forms must be accepted as absolute indications for removal of the uterus and the tumour, unless the patient's general condition is so bad as to prohibit operation. Calcareous and fatty degenerations are sometimes absolute, sometimes relative indications, while the involution changes after parturition or the menopause, being conservative, do not call for surgical interference.

7. *Inability to work* in the case of women who must earn their living is generally accepted as an indication for operation, and surely it must be conceded that a woman, whose happiness and usefulness depend on her activity, has a right to demand relief when she prefers the risk of operation to the death in life of a "sofa existence."

8. Another indication which must weigh with surgeons is the misery inflicted by a large, heavy, and disfiguring tumour, which interferes with both the duties and pleasures of life, and excites much painful comment, and sometimes injurious suspicion.

Fibro-Cystic Tumours of the Uterus (*see ante*, p. 354).

Polypi of the Uterus

The term uterine polypus simply denotes the pediculation of a mass with relation to the cavity of the uterus, and gives no information as to its

nature or condition. The chief varieties of uterine polypi are—

1. The fibroid.
2. The adenomatous (so-called mucous).
3. The malignant — sarcomatous, carcinomatous.
4. The placental and fibrinous.
5. The papillomatous.
6. The tuberculous.

1. *Fibroid polypi* are submucous fibroids in process of extrusion. They appear to originate as interstitial fibroids. They grow in the direction of least resistance, and are also pressed towards the cavity of the uterus by the contractions of its musculature. Thus they become submucous, and, if the circumstances for their extrusion are favourable, they become increasingly prominent in the uterine cavity. They then approximate to the condition of a foreign body, and are gripped with increasing power by the uterine walls. Gradually they are extruded from their muscular surroundings, and their investment of mucous membrane is stretched and thinned at the base so as to form part of the pedicle. The mass is pressed against the internal os and gradually dilates it in the same manner as does an ovum. This resistance overcome, the same process is applied to the cervical canal until the polypus is forced through into the vagina. The main factors are the contractions of the musculature, the stretching of the mucous membrane (which sometimes gives way so that the fibroid is more or less denuded in its descent), the opening of the cervix, partly due to "polarity" and partly to softening from increased supply of blood and mucoid discharge. The whole process is analogous to that of parturition, although the parallelism of details must not be pressed. One feature of the process is that a polypus may be extruded during the increased action and softening peculiar to menstruation, and withdrawn into the cavity of the uterus during the intermenstrual period (intermittent polypus). The possibility of extrusion through the vulva depends on the length of the stalk, or on the dragging down of the uterus in the process of extrusion.

The histology and pathology of uterine fibroid polypi is identical with that of fibroids in general, but they are usually of the "hard" variety. The mucous membrane covering a polypus usually retains the columnar ciliated type and its glandular arrangement while the polypus is intra-uterine. It becomes stratified and squamous after the extrusion of the growth, except in recesses and crannies which are not exposed to pressure from the vaginal walls.

They are nourished by their envelope of mucous membrane, which, as it is stretched and thinned, becomes progressively less vascular. Large blood-vessels are seldom found in the pedicle. To this comparative deprivation of blood-supply is due the increased tendency to sloughing and gangrene observed in submucous and polypoid fibroids. Hence if delayed in the canal of the cervix, or at the os uteri, the part of the tumour beyond the constriction is deprived of nourishment and exposed to the invasion of micro-organisms; it is therefore in the most favourable condition for degeneration.

In size, fibroid polypi vary from that of a walnut, or even less, to that of the uterus at full time; in one case such an extruded tumour was mistaken by an Indian midwife for a monstrous birth. The extrusion through the pelvis occupied a week, and as it was completed the patient died. The tumour was the size and shape of a uterus at term.

Usually fibroid polypi lie free in the vagina, or in the circle of the os uteri; exceptionally, adhesions form, making it difficult to diagnose these polypi from other morbid conditions, such as inversion and prolapse of the uterus and malignant growths.

Symptoms of Fibroid Polypi.—The most important are hæmorrhage and pain. The hæmorrhage, which is menorrhagic so long as the polypus is intra-uterine, becomes metrorrhagic after its extrusion into the vagina. The pain is caused chiefly, like the pain in labour, by the contractions of the body of the uterus, partly by the dilatation of the cervix and the os uteri, and by the pressure exerted on them by the protruding mass. The polypus itself is somewhat sensitive so long as it retains its envelope of mucous membrane. Pressure symptoms are rare. Deterioration of the general health follows from the frequently abundant and long-continued hæmorrhages, which cause profound anæmia, with its symptoms and dangers. A sloughing or suppurating polypus will give rise to the usual septic symptoms, and in cases of "enormous" polypi, like the one described above, death from exhaustion may occur.

The congestion and irritation caused by polypi are evidenced by leucorrhœal discharge, which is sometimes watery and profuse. It may become offensive from retention in the vagina and in cases where the growth is degenerate.

Women with large polypoid myomata are usually, but not always, sterile. If pregnancy occur, the polypus may cause abortion, or possibly obstruction during parturition unless previously diagnosed and removed.

2. *Mucous polypi* usually grow from the mucous membrane of the cervical canal and protrude through the os uteri. More rarely they are found on the vaginal surface of the cervix. They consist of hypertrophied mucous membrane, including some connective and muscular tissue. They usually retain the columnar ciliated epithelium of the endometrium with its glandular arrangement, but sometimes the epithelium shows a stratified structure. When

these polypi are cystic they are known as "channelled polypi," when the connective tissue is dense the term "fibro-cystic polypus" is applied. Probably many cases of endometritis, polyposa, fungosa, and hæmorrhagica are really cases of multiple polypi (or pedunculated adenomata) of the endometrium; and just as these soft cervical polypi occasionally undergo malignant change, so the cognate condition of the endometrium may sometimes be precedent to one or more varieties of malignant disease of the body of the uterus. Localised hypertrophy of the cervical glands certainly constitutes adenoma cervicis benigna or maligna in different cases (*see* Williams on *Cancer of the Uterus*, London, 1888, pp. 40-44).

3. *Malignant polypi* (*see* p. 371).

4. *Placental polypi* and *fibrinous polypi* may be considered together; for while in the former the fibrin of the blood is deposited on a shred of placenta or membrane, in the latter it is deposited on a ligature or suture left exposed after an operation such as hysterectomy. They are not neoplasms but accidents. The diagnosis is easy, the soft jelly-like clot, usually red, but sometimes decolorised, being unlike any ordinary growth. The foreign body which caused the coagulation being removed, the whole process comes to an end. The one symptom is hæmorrhage, moderate in amount and persistent in character.

In both cases the previous history of the case is likely to help to a correct diagnosis, the placental polypus occurring only after miscarriage or labour at term.

5. *Papillomatous polypi* are usually malignant. A papilloma of the cervix is rarely innocent; and even when apparently simple it demands removal with the portion of cervix from which it grows, as too often it recurs and eventually demonstrates its essentially malignant character.

6. *Tuberculous polypi* are rare, and probably often escape diagnosis because their existence is not borne in mind.

Angiomata and *teleangiectatic tumours* are varieties of the fibro-myomata (*see* p. 348).

Adeno-myomata

This is believed to be a rare form of "fibroid," but until all tumours removed by operation, or post-mortem, are carefully examined by the microscope as well as by the naked eye, it is not possible to know anything definite about their histology, and many cases of adeno-myomata may pass unrecognised.

The glandular structures found in "fibroids" are derived from (*a*) ingrowths of the glands of of the uterine mucosa; (*b*) remains of the Wolffian body.

When the glandular elements of a fibro-myoma are derived from the glands of the mucosa the tumour is more or less central, abutting on the cavity of the uterus.

When the glandular elements are derived from remains of the Wolffian body, the tumour is situated near a uterine cornu, or at any rate peripherally. Similar tumours are occasionally found in the Fallopian tube.

The fibrous, muscular, and glandular elements coexist in varying proportions in individual tumours. When the glandular element predominates, the tumour may be very soft, full of cysts, and sometimes teleangiectatic or angiomatous.

The chief interest of these adeno-myomata uteri lies in their obviously probable connection with carcinomatous degeneration of "fibroids," rare though that degeneration is. Such a degeneration has been discussed by Kelly, Senn, Macnaughton Jones, Hektoen and Riessman, and other authorities. The whole subject wants much careful pathological research.

Kelly, in his *Operative Gynæcology*, vol. ii. p. 385, under the heading "Adeno-myoma uteri diffusum benignum," says: "In the Johns Hopkins Hospital *Reports*, vol. vi. p. 133, Dr. T. S. Cullen describes one of my cases constituting a new variety of myoma, under the name of 'Adeno-myoma uteri diffusum benignum.' I have also had two more cases during the past year. These adeno-myomata are rather more interesting from the pathological than from the clinical standpoint, because their true character cannot be recognised before operation, as their symptoms do not differ from those of simple myomata."

Hektoen and Riessman, in their *Text-Book of Pathology*, vol. ii. p. 1040, say: "Adeno-myomata are a rare form of myomata of the uterus containing gland-elements. The epithelial canals in these tumours are either derived from the uterine mucosa or the remains of the Wolffian body," etc.

In Stanmore Bishop's *Uterine Fibro-Myomata*, p. 95, we find the following: "In certain cases very distinct epithelial relics are found in myomata. These seem to represent remains of the primitive epithelium of Müller's duct. Relics of the Wolffian duct are found in the uterine wall. . . . Max Voigt has detected distinct glandular structures in two myomata. Hauser and Diesterweg trace these glands to Müller's duct, Nagel and Breus to the duct of the Wolffian body." These remarks explain how a fibroid sometimes becomes cancerous, a change which they believe occurs.

"Von Recklinghausen, following von Babes, who in 1882 detected true epithelial growths in the interior of uterine myomata, traces these growths, tubular or glandular bodies lined with epithelium, to the Wolffian duct. Coblenz insisted on the pathological importance of the uterine portion of that duct," *et seq.*

Differential Diagnosis of Uterine Fibroids

May be considered under two heads:—

1. While the tumour is still of moderate size

and contained in the pelvic cavity it may be confounded with—

(*a*) Early normal pregnancy. (*b*) Molar pregnancy. (*c*) Extra-uterine pregnancy. (*d*) Threatened or incomplete abortion. (*e*) Sub-involution after parturition. (*f*) Retroverted uterus, gravid or not gravid. (*g*) Retroflected uterus, gravid or not gravid. (*h*) Prolapsed, enlarged, or adherent ovaries and tubes. (*i*) Small cyst, deep in broad ligament. (*j*) Inverted uterus (mistaken for polypoid fibroid). (*k*) Uterus infected with echinococcus. (*l*) Malignant disease of the body of the uterus.

2. When the tumour is wholly or in part abdominal it may be confounded with—

(*a*) Pregnancy, from end of fourth month to term. (*b*) Extra-uterine abdominal pregnancy. (*c*) Ovarian tumours, cystic and solid. (*d*) Broad ligament tumours. (*e*) Hæmatocele. (*f*) Perimetritis and parametritis. (*g*) Splenic, hepatic, and renal tumours. (*h*) Retro-peritoneal growths (chiefly sarcomata and lipomata). (*i*) Distended bladder. (*j*) Sarcomata of uterus. (*k*) Hydatid cysts. (*l*) Tumours formed by intestines and omentum matted together, usually by tuberculous disease.

Some of the above conditions admit of the very easiest elimination, others can be diagnosed with ordinary care, but others may defy the most painstaking examination and the closest reasoning.

In all cases where the diagnosis lies between a uterine pregnancy (the fœtus being alive) and a fibroid, time is an important factor; the question will be settled by the rate of growth and by the gradual development of the "certain signs of pregnancy." Discharge of blood, intermittent or persistent, during the early months may cause doubt, but either the case ends in miscarriage, or the steady growth in due relation to time, the detection of the fœtal heart sounds and movements, the procuring of ballottement, and the alternate contraction and relaxation of the tumour will shortly settle the question. On the other hand, amenorrhœa, instead of hæmorrhage, may accompany the existence of a fibroid, but its slower growth and the absence of the certain signs of pregnancy will afford grounds for a correct diagnosis.

The difficulty is greater when the embryo has perished and is retained in utero; but where there are no signs of life we may, after judicious delay, dilate the cervix and explore the uterus under anæsthesia.

The maximum difficulty in the differential diagnosis of fibroids and pregnancy is when they coexist. Here, again, time is the great elucidator, for owing to the usually accelerated increase in size of fibroids during pregnancy the uterus will soon be larger than is normal for the supposed time of gestation, and careful palpation will probably reveal one large boss or several bosses that are manifestly not parts of the fœtus.

In molar pregnancy, in abortion, and in sub-involution, where any doubt as to the accuracy of the diagnosis exists, the dilatation and exploration which are indicated as treatment will also reveal the true condition; which, however, would not be of immediate importance unless hæmorrhage or some other urgent symptom were present.

Swellings in Douglas' pouch may call for immediate recognition and treatment, as in the case of extra-uterine pregnancy; of retroverted or retroflexed uterus, especially if gravid; of prolapsed and enlarged ovaries and tubes; of an incarcerated fibroid. The history of the incarcerated fibroid would probably be longer than that of the various conditions of pregnancy. It would usually include attacks of hæmorrhage, while it would not be accompanied by mammary signs. The detection of the position of the uterus and the direction of its canal would be evidence for or against the tumour being the uterus itself, a fibroid, or some disease or displacement of the adnexa. The investigation may not be entirely free from risk, but non-recognition and consequent inadequate treatment of some of these confusing conditions may lead to a fatal result.

A fibroid polypus has been mistaken for a chronically inverted uterus. The diagnosis may be made on bimanual, or combined rectal and vesical, examination; the body of the uterus being thus detected in its normal position apart from the procident mass. The sound can be passed into the cavity of the uterus and all round the stalk of the polypus, unless the body of the polypus be adherent to the uterus, which is not common. Lastly, the polypus is comparatively or quite insensitive, which is not the case with the inverted uterus.

A prolapsed uterus is recognised at once by the position of the os uteri, into which the sound or a probe may be passed.

The uterus infested by echinococci presents some resemblance to the fibroid-bearing uterus, especially while the cysts are covered with uterine muscle and protrude as rounded bosses into the uterine cavity. The history and symptoms would probably help little, and the diagnosis would rest chiefly on the extrusion of one or more cysts and on the nature of the discharge should they burst *in situ*.

A cyst, whether parovarian or parasitic, deeply situated between the layers of the broad ligament, may be so immovable and so tense as to greatly resemble a fibroid growing in the same position. In neither case is hæmorrhage a symptom unless other diseases, such as uterine fibroids or endometritis, coexist. In both conditions pain and pressure symptoms may be present, and generally the recognition of a "tumour in the broad ligament" has to be the provisional diagnosis until growth, or the supervention of serious symptoms, at once proclaims

the nature of the trouble and demands operative assistance.

When the tumour is wholly or in part abdominal it should be easy to diagnose it from an uncomplicated pregnancy of the same volume, but it will frequently be impossible to assert or to deny the coexistence of fibroid disease with a comparatively early pregnancy. Some change in symptoms may occur, especially in the arrest of hæmorrhage if it existed before the pregnancy began. Mammary signs and softening of the cervix may exist in fibroid disease apart from pregnancy, and are in no case certain signs of gestation. Frequently the diagnosis cannot be certain until the mid-term of pregnancy brings the certain signs, or unless one or more fibroids can be felt per vaginam or per abdomen, and recognised apart from the enlarged, softened, and manifestly pregnant uterus.

Extra-uterine pregnancy, when advanced, will be easily distinguished from fibroids if the embryo is still alive, but difficulty arises when the child has perished and involution changes have occurred, or when extra-uterine pregnancy coexists with fibroids of the uterus. The symptoms of pregnancy may be much overshadowed by those of the fibroid. Hæmorrhage from the uterus may occur, while pressure symptoms and distress from increasing size would be aggravated by the coexisting pregnancy. If the history is well known and trustworthy it will be of great assistance, and probably the symptoms would demand interference for the removal of one or both conditions.

Ovarian and broad ligament cysts might be supposed to be easily diagnosed from fibroids, but in some cases the smooth, soft myoma greatly resembles an ovarian cyst on palpation, often yielding a sense of fluctuation, while the great test of passing the sound frequently fails to correct these faulty observations. It is assumed that the uterine cavity is elongated in cases of fibroid enlargement, and that, as it is unaltered in cases of ovarian cyst, the passing of the sound will give a diagnostic result. Unfortunately the distortion of the canal in many cases of fibroids, and its absolute blocking by submucous growths or adhesions in others, may completely prevent the passage of the sound and falsify the conclusions obtained. The duration of history should assist the diagnosis, but patients, even when honest, are often unobservant and inaccurate, and will give one year's history for a tumour that has obviously been growing five or more years. Lastly, abnormal hæmorrhage, although regarded as typical in cases of fibroids, does not always occur when they are present, whereas it may be present in ovarian cysts, especially in cases of dermoid cysts, and if tubal disease or endometritis coexist. Here again time is the great elucidator. Having found the tumour, its rate of growth can be observed, and should this be rapid, operation may be forced on the surgeon whether the tumour may be ovarian or uterine.

The solid tumours of the ovary are much less common than the cystic, and also less common than uterine fibroids. They can sometimes be diagnosed by their position, or by their manifest separation from the uterus, but these tests are not infallible. Inasmuch as solid ovarian tumours are seldom innocent an immediate diagnosis is necessary, and must be made even by exploratory incision if necessary.

Hæmatocele when recent is easy of diagnosis from fibroids, but sometimes in an old case where the blood has coagulated and a firm tumour results the distinction is difficult.

Splenic, hepatic, and renal tumours, if seen before the abdomen is filled by the growth, should be easily distinguished from fibroids of the uterus which grow from below upwards. The cases threatening confusion are rare, but their possible existence must be remembered.

Retro-peritoneal growths, chiefly sarcomatous, may usually be recognised by their comparatively rapid growth, by the unaltered length of the uterine cavity, by the absence of hæmorrhage, and by the immobility of the tumour. Sometimes these criteria fail, as when a fibroid is undergoing destructive changes or sarcomatous degeneration, and therefore increases rapidly in size, at the same time becoming less mobile. In these cases an exploratory incision may be the only means of diagnosis, and is certainly advisable when there is a doubt, for where the growth proves to be removable the exploratory incision is the first step of the major operation. On the other hand, should the growth be unfit for operation no great danger will be incurred as a result of exploration, provided strict asepsis is preserved.

Sarcomata of the uterus may well be mistaken for fibroids. The question often is whether any given tumour was a sarcoma from the first, or whether the malignant growth has supervened in a pre-existing fibroid. The practical question here is less of diagnosis than of treatment.

Distended bladder so frequently occurs in abdominal tumours that a catheter should be passed as a routine practice in all cases before operation, and even before an opinion is expressed.

Hydatids of the abdomen would appear more likely to be confused with cystic than with solid tumours, but occasionally they are found to fill the abdomen with a mass of hard round bosses yielding no general fluctuation, and closely simulating a multinodular fibroid. Unfortunately, as in other cases, neither the history nor the symptoms avail much. Both conditions are alike compatible with apparently perfect health, and in both the supervention of suppuration and other changes may bring the patient to death's door.

Perimetritis, parametritis, and localised effusions into the peritoneum can seldom be mistaken for fibroids. The upper rounded margin may closely simulate a tumour of the broad ligament, or of the space between the bladder and the uterus. On the other hand, the lower part of a fibroid, even when incarcerated in the pelvis, will seldom show so uniform a surface as occurs in para- and perimetritis; and the position of the uterus pushed up or to one side by a fibroid is seldom like its quasi-central position when it is embedded in an outpouring of plastic inflammatory products. The history, too, is widely different—cellulitis usually follows parturition, operation, or infection, and is accompanied by a rise of temperature and pulse, and by a general systemic reaction.

The difficulty is greater in old cases of cellulitis where shrinking and partial absorption have occurred, sometimes leaving an isolated hard mass in a broad ligament. Here the best clue is afforded by the less rounded outline and less sharp definition of the inflammatory mass, and also by the absence of any pedicle such as might be made out in a pedicled fibroid, though the pedicle might be simulated by strands of old inflammatory tissue between the intra-ligamentous mass and the uterus.

Treatment of Fibroids

In some cases the treatment is to "let it alone"; but considering the many complications, degenerations, and dangers that may occur, the patient should be carefully examined at suitable intervals, and accurate notes kept of all the details of her case, so that rapid growth, attacks of pain, and other warnings of danger may receive attention.

Medical Treatment.—Medical treatment of fibroids has been almost entirely limited to efforts to control hæmorrhage and to relieve pain.

The drug most in use is ergot in one of its forms, administered either subcutaneously or by the mouth. Subcutaneously—sclerotinic acid or ergotinin; by the mouth—ergole or ergot in doses of 1 to 3 grains three times a day for three months, with possibly large doses of the liquid extract of ergot (one-half to one drachm combined with ten grains of bromide of potassium) during the periods if the hæmorrhage and pain are excessive.

Another drug used somewhat largely in America and also in Europe is hydrastis canadensis. This may be ordered in doses of $\frac{3}{8}$ gr. hydrastinine three times a day, or may be taken three times a day in doses of 20 to 30 drops of tincture of hydrastis.

Hazeline has been found useful, especially in cases where the hæmorrhage is caused by passive congestion of the uterine mucous membrane. It should be given in doses of 20 to 40 or even 60 drops combined with 10 of tincture of nux vomica and an ounce of chloroform water three times a day. In pill it can be given as extract of hamamelis gr. j., ext. nux vomica $\frac{1}{3}$, and ext. gentian, or pil. rhei co. gr. j., three times a day. Preparations of thyroid gland and of the suprarenal capsule have also been tried. Adrenalin hydrochloride in 3-minim doses checks hæmorrhage, and calcium chloride is sometimes effectual.

It is to be borne in mind that these drugs are directed against one symptom only, and that even if they check the hæmorrhage they have no power to relieve pressure effects, to prevent degeneration, or to deal with complications. Ergot, the most powerful of the three, is scarcely a drug to be used for months and years. It is chiefly useful in cases of submucous fibroids or polypi, for by stimulating the muscular contractions it may assist in the extrusion of the mass.

The relief of pain is a difficult question. It is sometimes a very marked and exhausting symptom, and yet in a disease which may run a very chronic course it is wrong to teach the patient the morphia habit, or to permit her to depend on any drug in the long list of analgesics and hypnotics.

Electrolysis as applied to fibroids has been long known and has been largely used, especially by Apostoli and his school in France, and in Great Britain by Dr. Keith and his sons. When the mortality of direct operations on the tumour or the uterus was heavy, electrolysis was gladly hailed, and it had a fair and prolonged trial.

It has greatly disappointed the hopes of both surgeons and patients, and is now but little used in England. It is a tedious, painful, and very uncertain remedy. It is not free from danger, especially if the sharp electrode is plunged into the tumour or into the musculature of the uterus. It is so used with the intention of producing a limited and aseptic necrosis of the part of the tumour into which it is plunged, but the process may be neither limited nor aseptic, and sometimes leads to destructive changes which may entail prolonged invalidism or even septicæmia. When the blunt electrode only is used, the effects of the electrolysis are confined to the mucous membrane, and in cases where there is hæmorrhagic endometritis some improvement may be obtained.

Surgical treatment includes operations applicable to all the varieties of fibroids. To begin with the simplest operations on fibroid polypi—when of moderate size and extruded into the vagina these may be easily removed by avulsion or torsion of the pedicle. If the pedicle is thick, it is well to circumcise the mucous membrane and then to twist the fibrous pedicle remaining. This little operation, like all others, should be done with full aseptic care. In large or "enormous" polypi, where the pedicle cannot be

reached, the bulk of the tumour should be reduced by morcellation, using strong toothed forceps and scissors curved on the flat, until finally the stalk is reached. In cases in which the polypus is not yet extruded it may be necessary to dilate the cervix uteri, or to make bilateral incisions in it so as to bring the lower pole of the polypus within reach. In no case should the ecraseur be used, as it has a tendency during tightening the wire loops to grasp a portion of the uterine wall, which would thus be seriously injured.

Enucleation of Submucous Fibroids — This operation has a very limited range, and is never advisable unless the tumour is apparently single, and so projects into the uterine cavity as to make its enucleation at one sitting a probable result. We have properly abandoned the dangerous practice of incising the mucous membrane and the capsule, and then waiting for the uterus to expel the tumour, assisted when necessary by traction and drugs. This method causes the devitalisation of the tumour, and often leads to suppuration within the capsule, and to sloughing of the tumour which may extend to the surrounding tissues. In these cases the products of decomposition are carried by the uterine lymphatics into the general circulation, and septic infection is the result. The operation is so uncertain and so dangerous that even were the mortality of hysterectomy and myomectomy double what they are it would still be condemned.

Morcellation of fibroids and their removal per vaginam has long been practised in America, but it has been chiefly brought before the profession by Péan and his pupils. He thought it a suitable operation for the removal of even the largest tumours, but it frequently fails in such cases, and then total hysterectomy has to be performed by a tired surgeon on an exhausted patient. Morcellation should be limited to cases in which the tumour is wholly uterine and does not extend into the broad ligaments; where the total mass does not exceed that of a five months' gestation; where the vagina is wide enough to permit of the necessary manipulations; and, finally, where the tumour is healthy.

Method.—The patient lies in the lithotomy position. After thorough cleansing of the vulva and vagina, Auvard's speculum is introduced and the uterus lowered by pressure from above and, if necessary, by seizing one or both lips with forceps whose grip depends on apposition and not on teeth. The cervix is then freed from its vaginal attachments and incised up to the lower pole of the tumour, so as to make two lips up to its level. Two pairs of Museux toothed forceps are then fixed on the tumour, and with scissors curved on the flat, or with a bistoury curved on the flat, the mass of tissue commanded by the lower forceps is removed. The forceps are reapplied higher on the tumour, and the process repeated as often as is necessary. A finger in the uterus and the other hand on the abdomen give information as to progress, and warning as to possible opening through into the peritoneal cavity. Sometimes the upper part of the tumour may be shelled out with little difficulty—a large, ragged, and sometimes bleeding cavity is left; hæmorrhage must be arrested by the application of long pressure forceps, and both it and sepsis prevented by irrigation with 2 per cent carbolic lotion or iodine water, or a solution of formalin, 1 per cent, at a temperature of 110° to 120° F. Gauze soaked in 5 per cent solution of adrenalin chloride is extremely effectual in stopping hæmorrhage. The cavity is then packed with strips of iodoform gauze. Shock, if present, is met with the usual remedies, and the patient put to bed. This method appears to work well if the limits as to size and condition of the tumour are observed. It has the advantage of leaving a functionally useful uterus; but it must fail in the following cases: when there are many separate fibroids scattered throughout the uterus, when they invade the broad ligaments, when the size of the tumour is excessive, and, above all, when suppuration, necrosis, sloughing, or other destructive changes have occurred. The risk of sepsis is then very great.

Vaginal hysterectomy, described under "Uterus, Malignant Tumours," at p. 375, is of service in tumours that are not too large to admit of extraction of uterus and tumour through the vagina. It is the operation of election in multiple fibroids of the uterus, exclusive of the subperitoneal variety, when the size is not excessive, when the broad ligaments are not invaded, when the tumours, so far as can be ascertained, are healthy, and when there is no known pelvic complications of an inflammatory or septic nature.

The process of morcellation as described above can be combined with the classical operation of vaginal hysterectomy, and the uterus so reduced in bulk can be removed, leaving behind no ragged cavity to start a septic infection.

Colpotomy, or incision through the vaginal roof, in order to reach and remove "fibroids" has a very limited range; it can only be useful in cases of subserous growths, and comes into competition with vaginal hysterectomy on the one hand and with operations per abdomen on the other. It also has the disadvantage of affording imperfect demonstration of, and access to, coexisting disease such as pyosalpinx.

Abdominal Operations for Fibroids

1. *Abdominal hysterectomy* (*see* p. 374).

2. *Abdominal myomectomy*, the removal of the fibroids alone through an abdominal incision. This is the operation of election in cases where there is one tumour or a few tumours too large for removal per vaginam, where the growth is

intra-ligamentous, and where it is subperitoneal. It is unsuitable where there are many small growths, and where they are submucous.

The abdomen being opened if the tumours are subperitoneal, the peritoneum over the growths is incised, the tumour shelled out, the flaps trimmed, and hæmorrhage having been arrested they are sewn together with Lembert's suture.

If the growth be a solitary soft myo-fibroma embedded in the hypertrophied musculature of the uterus, the operation is more difficult, but it is well worth doing in suitable cases. The uterine tissue is incised down to the tumour, which is shelled out by the fingers, aided by scissors or scalpel if necessary. Hæmorrhage, which would otherwise be formidable, is controlled by an assistant grasping the chief vessels through the broad ligaments. The raw cavity is obliterated and permanent hæmostasis is secured by superimposed layers of sutures, and, lastly, the peritoneal flaps are united by a Lembert's suture. The chief anxiety is so to unite the tissues and to obliterate the cavity as to prevent the formation of a hæmatoma, which would probably prove very troublesome. Where this operation is successful, the uterus and its appendages are left in a functionally perfect state, and in careful hands its mortality does not exceed that of abdominal hysterectomy.

Removal of the Uterine Appendages. — This operation was devised by Lawson Tait, and was gladly welcomed by the profession at a time when the alternative was the clumsy and dangerous hysterectomy with the stump of the uterus clamped in the lower angle of the abdominal wound. Now that an improved technique has reduced the average mortality of abdominal hysterectomy to 10 or even 5 per cent, Tait's operation has fallen into disuse. Tait himself in his latter days admitted that it was useless in the soft, solitary fibroid; its effects, even when it checks hæmorrhage, are too slow in cases where pressure symptoms call for relief; and at the present time surgeons are anxious to spare one or both ovaries in order to save the patient the distressing symptoms of a premature menopause.

Ligation of the uterine arteries (per abdomen and per vaginam) has been suggested with the intention of lessening the blood-supply to the uterus and so starving its tumours. The operation may be easy, difficult, or impossible, according to the size and position of the fibroids. The most serious objection to it is the uncertainty of the results. It may do too much or too little. In some cases the collateral circulation develops so well that the tumour flourishes undisturbed, in other cases its vitality is so lowered that it is liable to degenerate. In no case can the ultimate result be known before operation, nor indeed for some time after.

Tuberculosis of the Uterus

1. Occurs as an extension of tuberculous disease from the Fallopian tubes.
2. As an infection from the vagina.
3. As part of a general tuberculosis.

(1) When it occurs as an extension from the Fallopian tubes the sequence appears to be first tuberculous disease of the intestine—the mucous membrane, muscular and peritoneal coats, suffering in succession. Infection of the general peritoneum follows. Thus the bacilli obtain access to the Fallopian tubes and through them to the uterus. In these cases the fundus and corpus are earliest and chiefly involved.

(2) In infection from the vagina the morbid process occurs in the cervix, and may be limited to it.

(3) In general tuberculosis the uterus is said to be invaded in about 66 per cent of the cases. Tuberculosis of the uterus has been recognised in four forms, of which three appear to be a sequence, or phases of one process: (*a*) miliary; (*b*) caseous; (*c*) fibroid, the usual life-history of tuberculous lesions; while (*d*) papillary tuberculosis seems to be a special development.

In the earliest stage of uterine tuberculosis the epithelium is intact and the glands normal, the tubercle being found in isolated specks scattered in the subjacent tissues where the network of vessels surrounds the extremities of the glands.

In a more advanced stage the uterine cavity is covered with a more or less necrotic caseous material, and below this there is tubercular tissue involving the superficial layer of the musculature and in which the typical bacilli and giant-cell formations may be found. At this stage there are sometimes multiple shallow ulcers with undermined edges in the mucous membrane. In the papillary form a definite growth occurs, usually in the cervix, growing from the mucous membrane of the arbor vitæ. It pushes aside the pavement epithelium of the portio vaginalis, attains a considerable size and to the naked eye resembles epithelioma of the cervix, for which no doubt it is frequently mistaken.

The symptoms of uterine tuberculosis are not pathognomonic. Menorrhagia, metrorrhagia, and discharge occur as in many other diseases, and pain is not common unless there are coexisting tubal and ovarian lesions with inflammatory adhesions.

Treatment.—The symptoms are likely to lead to curetting, and the possibility of the existence of uterine tubercle should insure microscopic examination of the scrapings. In cases where other treatment fails, removal of the involved organs is indicated.

Hydatids of the Uterus.—These parasites probably gain admission by penetrating the uterine tissues from the abdomen or neighbour-

ing organs. They may be found under the peritoneum, in the walls, or under the mucous membrane. They can easily pierce the peritoneum and develop fresh colonies in the pelvic or abdominal cavities; or, piercing the mucous membrane, they may be extruded into the cavity of the uterus and so escape per vaginam.

The symptoms vary with the position of the mass. If the cysts are large and develop towards the abdominal cavity, a smooth, elastic swelling may be felt through the anterior abdominal wall. This tumour must be diagnosed from ovarian and broad ligament cysts, from fibroids and from pregnancy. Pain may arise from pressure and from inflammation, the patient may also suffer from a sense of weight and uneasiness. Difficulties of micturition and of defæcation may occur, and the periods are often irregular. Where the tumour communicates with the uterine cavity there may be expulsion of variously sized cysts, also of fluid and of blood. The hæmorrhage is seldom severe; it usually occurs as a slight but persistent metrorrhagia. In some cases sympathetic mammary changes occur which complete the simulation of ordinary pregnancy, or of pregnancy with hydatidiform degeneration of the chorion. A certain diagnosis rests on the detection of hooklets and on the chemical characters of the fluid discharged. It contains no albumin, and therefore is not coagulated by the ordinary reagents, such as heat and nitric acid. It does contain chloride of sodium, and therefore becomes turbid on the addition of nitrate of silver. The diagnosis is specially important in the case of large masses simulating fibroids, ovarian and other cysts, because the entrance into the abdominal cavity of the hooklets and minute cysts must be carefully avoided.

Treatment.—When hydatids of the uterus simulate a vesicular mole, the cervix should be dilated and the uterus evacuated and curetted. If the tumour present unbroken at or near the os uteri, it should be incised, its contents cleared out, then it should be irrigated with 25 per cent solution of hydrogen peroxide, and, lastly, it should be packed with long strips of iodoform gauze. In the case where hydatids have burrowed deeply into the musculature of the uterus, hysterectomy may be needed.

ACTINOMYCOSIS OF THE UTERUS is very rare, and when it exists is probably only an extension from the Fallopian tubes. Even in these actinomycosis is said to be "an anatomical curiosity" (*see* vol. i. p. 51).

V. Malignant Tumours of the Uterus

INTRODUCTION 363
CANCER OF THE NECK OF UTERUS . . 364
Treatment 368
CANCER OF NECK IN PREGNANCY . . 369
CANCER OF THE BODY 369
Treatment 371
CANCER OF THE UTERUS AND FIBROIDS . 371
SARCOMA 372
HYSTERECTOMY 372
Abdominal 374
Vaginal 375

The term malignant, when used in relation to a tumour, signifies that it inevitably destroys life; and in the article "TUMOURS" (vol. x. p. 153) every care is taken to point out that malignant tumours occur in the connective tissue group, where they are called sarcomata, and in the epithelial group, where they are known as cancers or carcinomata. As the architecture of the uterus is such as to render it liable to carcinomata and sarcomata, it will be useful to briefly review the distribution of the connective tissue and the epithelial elements in this complex organ, and also to consider certain structures connected with the development of the embryo, which have acquired notoriety in relation to some destructive tumours of a peculiar type.

The uterus is structurally a hollow muscle of the unstriped variety, containing, as is usual with this kind of muscle, a central space lined with mucous membrane. In this instance the mucous membrane is called the endometrium, and presents such peculiar characters that it may, without exaggeration, be described as the most extraordinary mucous membrane in the body. If the Fallopian tube or a piece of small intestine be examined in transverse section, it is quite easy to distinguish its muscular coat and to separate it from the mucous membrane, a loose connective tissue track indicating the boundary line. In the uterus there is no such line of separation, and the walls are thick and resistant. A careful study of the morphology of the uterus, however, teaches that this thick wall represents an enormously developed muscularis mucosæ, and this metamorphosed area extends from the fundus to the vaginal opening (external os) of the uterus. The endometrium which lines the cavity of the uterus is distinguished from that belonging to the cervical canal by the fact that when the Fallopian tube, the uterus, or an ovarian follicle contains an oösperm (fertilised ovum), the superficial parts of the endometrium above the point known as the internal os become changed into a thick, soft membrane known as the decidua, which is sometimes shed as a membranous bag, or in shreds when pregnancy fails.

The epithelium in relation with the uterus exhibits some remarkable variations which need consideration. The endometrium of the cavity of the uterus is beset with a single layer of columnar ciliated epithelium directly continuous with that lining the Fallopian tubes. This epithelium dips into the endometrium very deeply, and forms long tubular glands; they run obliquely in the mucous membrane,

and sometimes bifurcate at the extremity (Fig. 1).

The glands which belong to the cervical endometrium are of the racemose kind, and possess cylindrical epithelium which differs from that belonging to the glands in the body of the uterus in that the cells are narrower (Fig. 2). The stroma in which the glands are embedded is denser than that in the body of the uterus, and the cells are spindle-formed. At the external orifice of the uterus the epithelium undergoes a sudden change, and instead of consisting of a single layer the surface cells stratify, and the whole of the cone-shaped portion of the cervix uteri which projects into the vagina is covered by squamous epithelium, directly continuous with and like that clothing the wall of the vagina (Fig. 3). In the normal uterus the transition from the single cell layer of the cervical endometrium to the multiple cell strata of the vaginal surface of the cervix occurs very abruptly at the margin of the external orifice of the uterus. It is necessary to be familiar with these cell modifications, because the epithelial elements of cancers of the uterus are determined according to the part of the organ in which they arise.

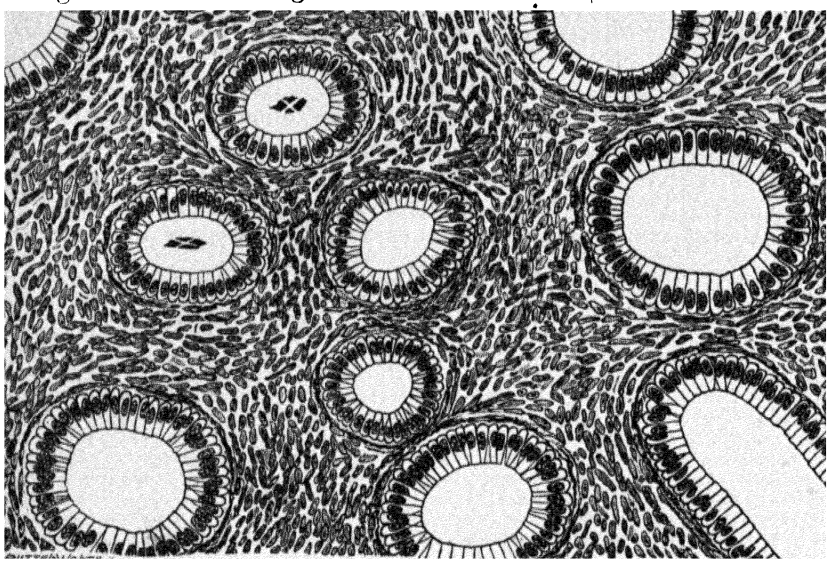


FIG. 1.—The tubular glands of the uterus in section. Magnified. (From the writer's book on *Tumours*.)

The distribution, microscopic characters, and peculiarities of uterine cancer are determined by epithelium. When describing the features of cancer in general (vol. x. p. 160) it was pointed out that this disease exhibits peculiarities in its mode of growth, infection of lymph glands, dissemination, and the way in which it destroys life according to the organ in which it arises. These features are illustrated in an extraordinary way in the uterus, for the frequency, course of the disease, clinical characters, and the way death is induced, vary widely according to whether the cancer arises in the epithelium of the cervix or in the body of the uterus. So that on pathological as well as on clinical grounds it is convenient to consider the subject in two sections, viz. cancer of the neck of the uterus, and cancer arising in the endometrium of the body of the uterus.

CANCER OF THE NECK OF THE UTERUS.—Careful statistical inquiry shows that the three organs most liable to cancer are the mamma (breast), the neck of the uterus, and the stomach. The same method of investigation also brings to light the important fact that cancer of the neck of the uterus is almost exclusively confined to women who have been pregnant. The writer found in one hundred consecutive cases of undoubted cancer of the cervix that ninety-nine patients had had one or more children; and the exceptional patient was a married woman, and in her case a miscarriage could not certainly be ex-

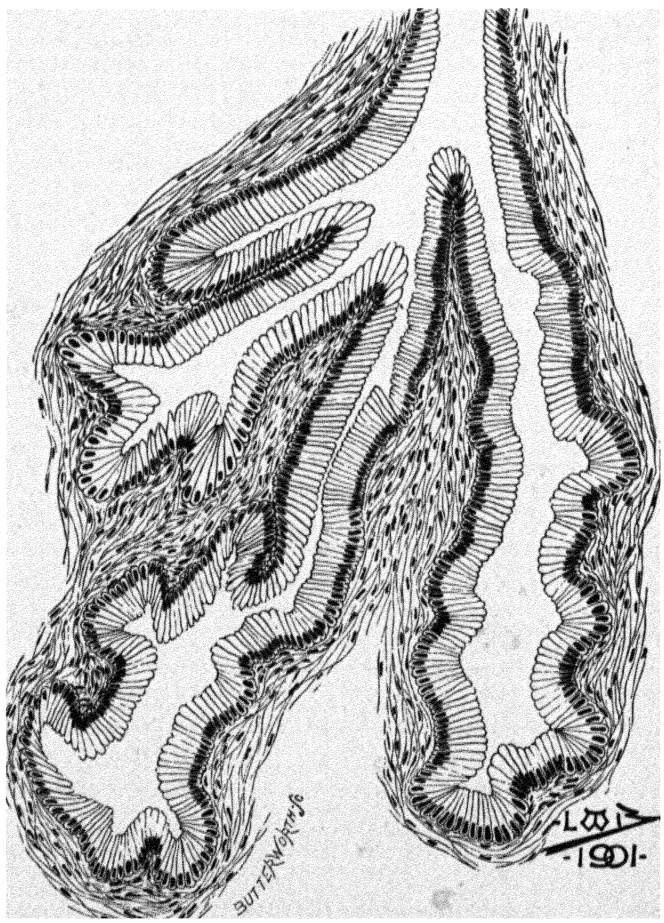


FIG. 2.—A gland from the cervical endometrium (magnified).

cluded. This disease is very common between the fortieth and the fiftieth years; many cases occur between the thirtieth and the fortieth years. Before the thirtieth year the disease is rare, but the writer has observed undoubted cases in women of twenty-three, twenty-five, and twenty-six years. Cancer of the neck of the uterus belongs especially to the latter part of the child-bearing period of life and the decade subsequent to the menopause. It is not easy to determine whether this predisposition depends on injury to the cervix due to delivery, or to traumatism associated with coitus. A remarkable observation bearing on this matter has been made by Czerwenka. A woman, thirty-five years of age, had a double vagina and "uterus bicornis bicollis." Coitus was practised on the left vagina. Panhysterectomy was performed because of cancer of the left cervix; the left uterus contained two fibroids, and the corresponding Fallopian tube contained pus and had its cœlomic ostium occluded. The relation of pregnancy to cancer of the neck of the uterus is shown in the table on p. 369.

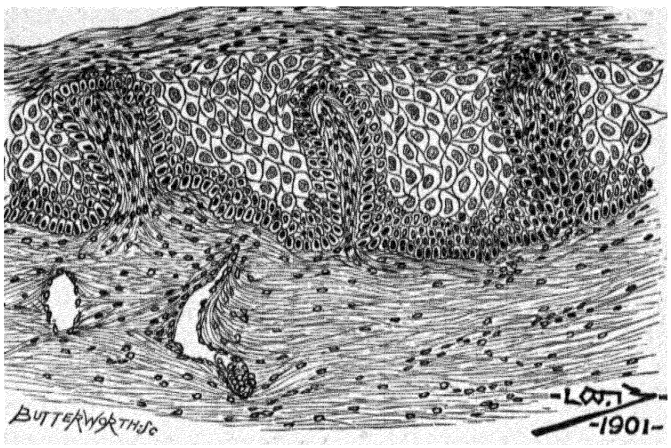


FIG. 3.—The microscopic characters and disposition of the epithelium on the vaginal aspect of the neck of the uterus.

Cancer may begin at any part of the cervix from the internal orifice of the cervical canal to its junction with the vaginal wall. The character of the epithelium determines the microscopic features of the cancer. Thus when the disease begins on any part of the vaginal aspect of the cervix, the cancer is of the squamous-celled (epithelioma) species; but when it arises from the cervical endometrium its distinguishing epithelium is of the columnar type (Fig. 4).

A great amount of careful labour has been expended on the microscopic examination of cancer of the neck of the uterus, with the hope of determining the relative frequency of squamous-celled and of glandular carcinoma. So far as the writer's efforts were concerned, his object was to decide if possible which species gave the best result to treatment. In a small proportion of cases, and especially those in the very early stages, it is a simple histologic exercise to determine the species, but unfortunately in the advanced cases it is not an easy, and certainly in some instances an impossible task to determine whether it be a squamous-celled or a gland-celled cancer. Some of the confusion in recent writings is due to the neglect of the fact, that when a cancer orginating in the epithelium of the cervical endometrium overruns the vaginal surface of the cervix it assumes the squamous-celled or stratified arrangement. This mutation of epithelium may be studied in the case of cancer of the rectum; when the disease involves the outlet of the bowel, the anal portion often has the structure of squamous-celled cancer, whereas the epithelium of the primary rectal tumour retains its columnar character. With our present means of investigation it is hazardous to attempt a prediction simply on the cell features of the cancer.

Mutation of epithelium may also be studied in a uterine polypus. For example, a polypus arising in the endometrium will be clothed with mucous membrane, covered with a single layer of columnar epithelium, but when the tumour protrudes into the vagina, the epithelium of the portion resting on the vagina stratifies and resembles the epithelial covering of the vagina. In a small pedunculated polypus of the cervical endometrium it is an interesting task to cut sections through the whole length of tumour, and study the transition in the epithelium. This peculiar change in the epithelium has been erroneously described as squamous-celled cancer arising in a uterine polypus.

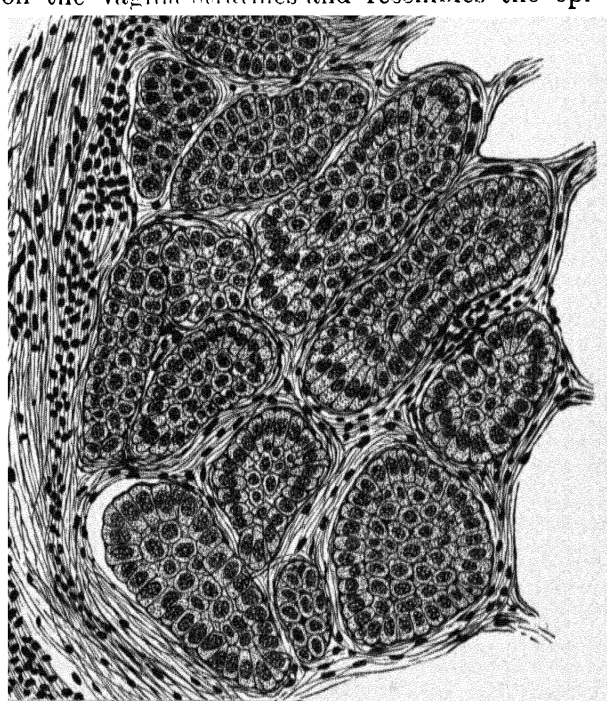


FIG. 4.—The microscopic characters of cancer beginning in the endometrium of the cervical canal. (From the writer's book on *Diseases of Women*.)

Although the ultimate results of cancer arising in the cervical endometrium or on the vaginal aspect of the cervix are the same, it will be advisable to discuss their pathologic features separately. It is necessary to mention that in the great majority of cases which come under observation, particularly in hospital practice, the disease has destroyed or eroded the neck of the uterus to such an extent that it is impossible to determine whether it arose in the cervical canal or in the epithelium of its vaginal aspect. Nevertheless, patients do occasionally come under observation at a sufficiently early stage to enable an exact localisation of the primary focus of the disease. In this way it has been determined that

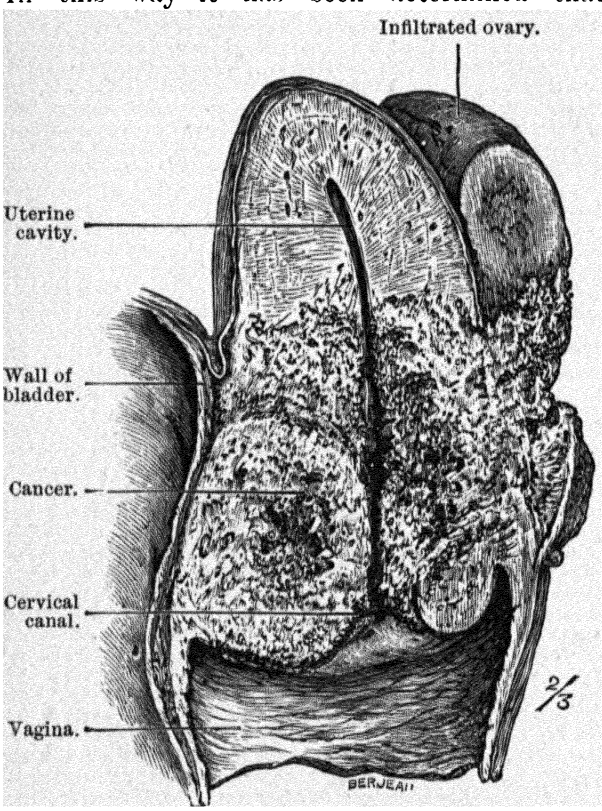


FIG. 5.—A cancerous uterus in sagittal section. The cancer arose in the cervical endometrium. (From the writer's book on *Diseases of Women*.)

squamous-celled carcinoma of the cervix begins in much the same way as on the lips or tongue. It may appear as a circular ulcer with raised and everted edges, or it may erode the tissues deeply at the outset; exceptionally it forms luxurious warty excrescences. The growth quickly infiltrates the cervix (Fig. 5), extends to and implicates the vaginal wall, and then involves the connective tissue of the mesometrium (broad ligament).

Cancer arises in the epithelium in any part of the cervical canal or its glands, but it appears to be more prone to arise in the lower than in the upper half of the canal. It begins either as a deeply eroding ulcer, or as a soft, fungating, vascular, cauliflower-like outgrowth. Commonly the cancer spreads, and after infiltrating the adjacent tissues of the cervix spreads into the mesometrium, and implicates the vaginal wall. It ulcerates early and destroys the cervix, and spreads into the body of the uterus, and in the late stages this organ may become hollowed out by ulceration, until nothing but a thin layer of muscle tissue covered by peritoneum remains (Fig. 6).

The microscopic features of cancer arising in the cervical epithelium consist of rounded spaces filled with columnar epithelium. This is due to the fact that the invasion of the tissues is due to columns and cylinders of epithelium, and in the microscopic sections these cell columns are cut at right angles.

Cancer of the cervix leads to infection of the lumbar lymph glands. Dissemination is also frequent, and secondary deposits form in the lung, liver, and other organs; but deposits do not occur in the bones with the same frequency as in cancer of the mamma. The worst features of cancer of the neck of the uterus do not arise from dissemination, but are due to the slow and insidious way in which the growth implicates adjacent structures; and this demands attentive consideration, because it is this aspect of the disease which imposes very marked limitations to its surgical treatment.

The neck of the uterus is in close relation in front with the bladder; posteriorly it is in contact with the rectum, and it has on each side of it a ureter. Another dangerous relation is the uterine artery which joins the cervix near the utero-vaginal junction. A careful study of the clinical facts connected with cancer of the neck of the uterus demonstrates that its more lethal character, considered apart from its frequency, as compared with cancer of the body of the uterus, is due to the secondary effects resulting from the implication particularly of the bladder, ureters, and uterine arteries (Fig. 7). The occurrence of the many serious complications of cancer of the cervix is mainly determined by the primary position of the lesion. For example, if the cancer begins on the posterior aspect of the vaginal surface it will, whilst it is deeply eroding the cervix, extend to the posterior vaginal wall, infiltrate the recto-vaginal septum, and either lead to a fæcal fistula or obstruct the bowel. On the other hand, it may start on the anterior surface and involve the vesico-vaginal wall, lead to cystitis, which may spread to the ureters and infect the renal pelvis, producing pyelitis, or in many instances render the patient extremely miserable by perforating the bladder, and producing a vesico-vaginal fistula.

When the disease begins in the cervical canal it exhibits a tendency, so marked in cancer of the intestine, to circumscribe the canal, and at the same time it infiltrates the tissues adjacent to the cervix, and in this way implicates the base of the bladder and one or both ureters. In

such cases it is not uncommon to find that segment of the ureter which traverses the mesometrium converted into a solid column. The liability of the bladder and the ureters to be implicated in cancer of the cervix explains the frequency with which uræmic symptoms become manifest in the terminal phases of this disease.

Cancer of the cervix tends to block the cervical canal, and in this way obstructs the free escape of the uterine secretion. The result is that the retained secretion becomes infected from the necrotic and decomposing tissue about the cervix, and becomes purulent and produces a condition known as pyometra. In some of the cases the cancerous tissue sloughs and allows the pent-up pus to escape, and irregular "gushes" of pus are not infrequent phenomena in the later stages of the disease. A pyometra becomes a source of danger when the infection extends to, and involves the Fallopian tubes. These structures then become thin-walled pus-sacs. Should the infective fluid contained in a sac of this kind leak into the belly through an unoccluded ostium, or should a perforation occur in the wall of the tube, death may follow in a few hours from septic peritonitis. Almost at any period in the course of the disease the uterine artery may be broached, and the patient suffers from severe and sudden bleeding. This in a certain proportion of cases terminates life, and when it falls short of this it places life in great jeopardy. Probably on the whole the patient's strength is more gradually exhausted by oft-repeated small bleedings, and thus death slowly supervenes, than that life is suddenly extinguished by a furious hæmorrhage of short duration. However, the two earliest, and the most constant signs of cancer of the neck of the uterus are frequent fitful losses of blood, and purulent and offensive discharges from the vagina. It is for one or other, and more frequently for both, of these signs that the patients seek advice, and in a small proportion of cases they also complain of pain.

It is the duty of every practitioner to examine carefully the uterus of women who complain of these troubles.

In the early stages the margin of the uterine orifice may be found hard and everted. Perhaps a soft mass protrudes from the canal and bleeds on the slightest touch. Occasionally a cauliflower-like mass will be found sprouting from the vaginal surface quite clear of margins of the "os." In some cases the conditions are such as to preclude a positive diagnosis, but bleeding, even on gentle manipulation, is always a suspicious sign, and especially when women complain that coitus is followed by slight bleeding. In cases of doubt an accurate diagnosis is of so great importance for the welfare of the patient, that a fragment of the suspected tissue should be removed and submitted to microscopic examination.

When the disease is well established its diagnosis is never difficult. The ulcerating, fungating, bleeding mass which protrudes from the cervical canal, or the crater-like depression which replaces it, with the hard, infiltrating ring of tissue which can be traced into the vaginal walls, are features easily recognised by the trained finger.

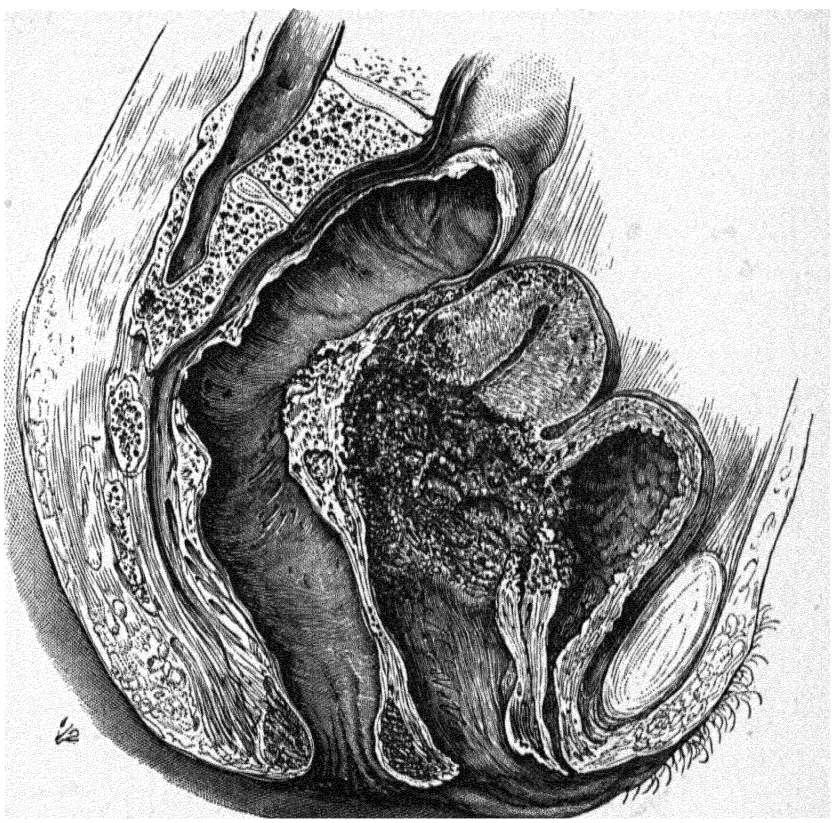


FIG. 6.—Pelvis and its viscera in section. From a case of cancer of the uterine cervix which invaded the bladder. (From the writer's book on *Tumours*.)

Cancer of the uterus terminates life in a variety of ways, which it will be useful to summarise :—

1. The uterine artery may be opened by ulceration, and fatal bleeding ensue.

2. Repeated bleeding from smaller arteries, and from the surface of the cancer, often leads to exhaustion and death.

3. Implication of the bladder, and of one or both ureters, causes cystitis, septic pyelitis, and uræmia.

4. Septic changes in the uterus extend to the Fallopian tubes, and septic matter may leak

through an unoccluded ostium and establish peritonitis.

5. Peritonitis may follow rupture of a pus-containing Fallopian tube.

6. Intestinal obstruction may follow the adhesion of a piece of intestine to the uterus, or direct extension of the cancer into the rectum.

7. Hydroperitoneum and hydrothorax may arise from the presence of secondary nodules on the peritoneum or pleura.

8. Secondary deposits in the brain are rare, but they occasionally induce fatal coma, and sometimes attacks of acute mania.

The only treatment available for cancer of the neck of the uterus is that adopted for cancer

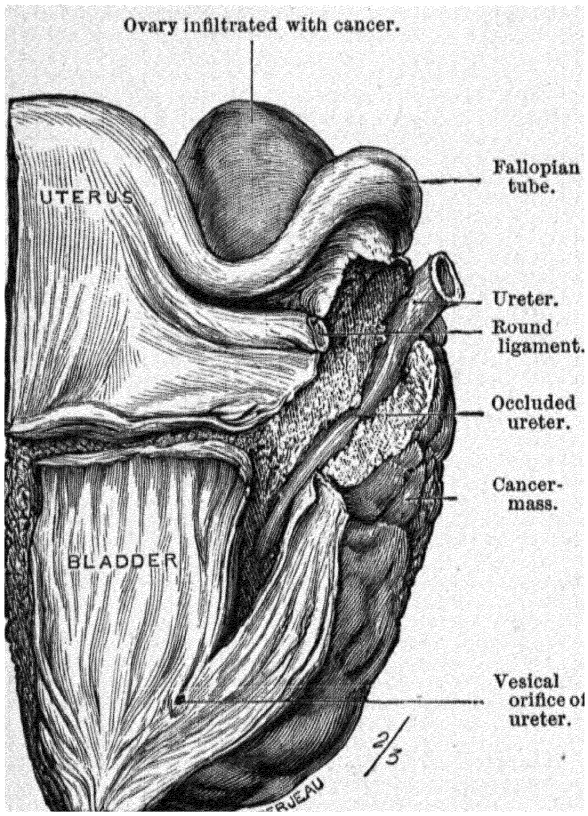


FIG. 7.—Cancer of the cervix uteri implicating the ureter and bladder. (From the writer's book on *Diseases of Women*.)

in other parts of the body, namely, thorough removal at the earliest possible moment; but, unfortunately, it is only practicable to carry this out in a very small proportion of patients. The first object in a radical operation for the relief of cancer in any part of the body is a wide removal of the infected part, and this is prevented in the case of the cervix by the proximity of the bladder, uterus, and rectum. When the patient comes early under observation, and especially when the cancer is limited to the cervical canal, the performance of vaginal hysterectomy is followed by satisfactory consequences immediate and remote; but when the disease has overrun the cervix and implicates the vaginal wall, it is impossible to make a free removal of infected tissue without imperilling the integrity of the bladder and uterus, and thus anticipating some of the most distressing effects of the disease. Quite apart from the limitations which the anatomical environment imposes on surgical efforts in the treatment of this disease, there is, unfortunately, the insidious character of the disease itself, and only a small percentage of patients seek advice when there is an opportunity of doing good by surgery. In recent years the mortality of vaginal hysterectomy of cancer of the cervix has reached the low point of 5 per cent, but the frequency and rapidity of recurrence has discouraged many surgeons. Now that the profession is awakening to the necessity of early diagnosis in this dreadful disease, and at the same time realising that the only hopeful cases are those where the operation is undertaken in the early stages, and that surgeons abstain from operating except in the promising cases, so will the results improve, and patients will be encouraged to seek surgical aid at the very outset.

In the majority of cases where no radical operation is possible, much may be done to make the patient's life not only tolerable but even comfortable. In a very large proportion of cases the patients are able to keep about, look after their homes, and lead useful lives often for many months until renal complications and anæmia, the joint result of repeated bleeding and purulent discharge, so weaken them that they are compelled to keep abed. At this stage careful nursing renders them fairly comfortable: the patients should be kept scrupulously clean by daily douches of warm water tinged with permanganate of potash. When the discharge is offensive a daily douche of perchloride of mercury (1 in 5000) will rarely fail to correct it. Pain may be alleviated by the judicious use of morphia administered subcutaneously. The constipating effects of this drug are best met by variations in diet, accompanied by ripe fruit and vegetables, aided by occasional enemata consisting of two to four drachms of turpentine, an ounce of castor oil, a small piece of soap, and a pint of warm water.

In cases unsuitable for operation life is rarely prolonged beyond a year and a half. Many die within a few months from the time they come under observation.

In order to emphasise the small proportion of cases of cancer of the uterus in which a radical operation can be carried out with success, the writer had a list prepared of fifty consecutive patients admitted into the Chelsea Hospital for Women, London, for the purpose of operation. The plan usually followed is to admit such patients with cancer which the junior officers think may prove suitable (the advanced cases are not admitted into the institution). The patient is then examined under an anæsthetic to ascertain the extent of the disease, and especially to determine if the cancer is limited to the uterus. The large proportion of rejec-

tions, even in cases which seem favourable without this critical examination, is well shown in the subjoined table. It is very important to exercise a rigid selection of cases for operation, otherwise vaginal hysterectomy for cancer runs a great risk of being discredited. The table was drawn up by Dr. Crewdson Thomas, the registrar: the patients were admitted during 1897, 1898, 1899, and 1900. During these years the average number of women who were operated upon for diseases of the genital organs was 500 yearly. The lists show the frequency of the disease, and also demonstrate that cancer of the cervix has an extremely significant relation to childbirth.

So far as this table is concerned, and indeed in very large collections of cases, cancer of the cervix is seen to be intimately associated with the later period of sexual life and the decade following the menopause. It is almost unknown before the twenty-fifth year, the great proportion of cases falling in the zone between the thirtieth and fifty-fifth years of life.

Table of 50 *Consecutive Cases of Cancer of the Neck of the Uterus*

No.	Age.	Children.	Miscarriages.	Treatment.	Remarks.
1.	21	1	...	Vag. hysterectomy	
2.	27	1	...	Inoperable	
3.	29	1	...	,,	
4.	29	2	...	Vag. hysterectomy	
5.	30	1	1	Inoperable	
6.	31	...	1	Vag. hysterectomy	
7.	32	2	...	Inoperable	
8.	33	3	...	,,	
9.	33	5	...	Ab. hysterectomy	8 months pregnant.
10.	34	5	1	Vag. ,,	
11.	35	3	...	Inoperable	
12.	36	4	...	,,	
13.	37	3	1	,,	
14.	37	1	...	,,	
15.	38	8	...	,,	3 months pregnant.
16.	38	1	...	Vag. hysterectomy	
17.	39	7	1	Inoperable	
18.	40	3	2	Vag. hysterectomy	
19.	41	1	...	Inoperable	
20.	41	4	...	,,	
21.	42	8	1	,,	
22.	42	7	6	,,	
23.	42	4	...	,,	
24.	43	4	...	Vag. hysterectomy	
25	43	1	...	Inoperable	
26.	45	6	4	,,	
27.	46	7	2	,,	
28.	47	2	...	Vag. hysterectomy	
29.	47	7	4	Inoperable	
30.	48	7	...	,,	
31.	49	6	2	,,	
32.	49	4	...	,,	
33.	50	1	...	,,	
34.	51	1	...	,,	
35.	52	2	...	,,	
36.	53	8	1	,,	
37.	53	6	2	,,	
38.	53	6	1	,,	
39.	55	8	...	,,	
40.	55	3	...	,,	
41.	55	1	...	,,	
42.	55	6	1	,,	
43.	55	1	..	Vag. hysterectomy	
44.	59	6	...	Inoperable	
45.	59	7	...	,,	
46.	59	3	...	,,	
47.	61	9	1	Vag. hysterectomy	
48.	66	5	...	Inoperable	
49.	68	5	...	,,	
50.	70	2	...	,,	

An interesting feature in relation to cancer of the neck of the uterus is its occurrence in women many years subsequent to double ovariotomy. Examples of this are recorded in the *Transactions of the Obstetrical Society, London*, vols. xxxvii. 213, and xliii. 214.

Cancer of the Neck of the Uterus and Pregnancy.—It may be stated without fear of contradiction that the most appalling complication of pregnancy is cancer of the cervix. It is somewhat difficult to understand how a woman with cancer of the neck of the uterus can conceive, but it is quite certain that it happens when the disease is well established. From a careful inquiry, the writer is persuaded that this complication is not uncommon, but that the cases in which cancer of the neck of the uterus obstructs labour are unusual, and this is due to two circumstances—(1) that the cancer prodisposes to abortion; and (2) that when it has advanced to such a stage as to fill the vagina with an obstructive mass, it has had such an effect upon the health of the mother that it endangers the life of the fœtus. This is a matter of some importance, because in considering the advisability of performing Cæsarean section in these circumstances, it is necessary to be certain that the fœtus is really alive. However, in very exceptional cases it has been found absolutely necessary to resort to this operation in order to deliver a dead and putrid fœtus.

The careful study of the literature relating to this complication shows clearly enough that, when a pregnant woman with early cancer of the uterus comes under observation in the early months, her best hope lies in vaginal hysterectomy. In the later stages (fourth to seventh months) very good consequences have followed the amputation of the cervix, and this operation has been carried out very successfully and without disturbing the pregnancy. In the latest stages the best consequences have followed the induction of labour and the immediate performance of vaginal hysterectomy, for, surprising as it may seem, the uterus, though enlarged from the pregnancy, can be safely extirpated through the vagina.

These methods of treatment only apply to cases where the cancer is in such a condition as to afford reasonable hope of a prolongation of life. When the disease is in an inoperable stage and the fœtus is dead, then after a little patient waiting abortion usually occurs. When there is reliable evidence that the fœtus is alive, then the pregnancy should be allowed to go to term; if the cancer affords an impassable barrier to the transit of the child, then Cæsarean section becomes a necessity.

Cancer of the Body of the Uterus.—This is much less frequent than cancer of the neck of the uterus, and it arises in the epithelium lining the cavity of the uterus and its tubular

glands. There is very little accurate knowledge regarding its early stages, and the writer has only had one opportunity of obtaining a cancerous uterus before it had extended to the muscular wall. The disease remains for a long time restricted to the body of the uterus, and may creep into the uterine sections of one or both Fallopian tubes: it rarely invades the cervix, and then only in the late stages of the disease. It is apt to perforate the wall of the uterus and infect the peritoneum (Fig. 8).

It is only in the last fifteen years that the importance of cancer of the body of the uterus has been clearly appreciated, and this was due to the fact that there were no means available for the proper examination of the interior of the organ, and as a result the description of diseases of the endometrium was disfigured or obscured

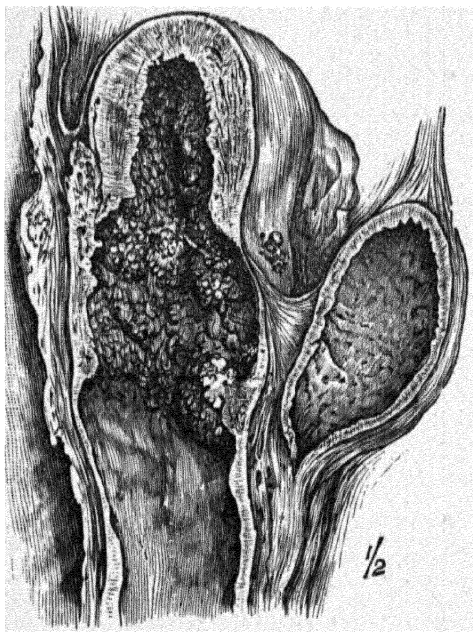


FIG. 8.—A cancerous uterus in sagittal section; a small process of cancer protruded through the uterus and infected the peritoneum, which contained thousands of secondary nodules. (From the writer's book on *Tumours*.)

by a crowd of terms, such as senile endometritis, malignant endometritis, villous endometritis, and so on.

When the plan of mechanically dilating the cervical canal was introduced, so that the endometrium could be examined and fragments obtained for the laboratory, then light began to shine and we are in possession of some accurate data.

As in other organs cancer of the body of the uterus consists of cell-columns, the cells being identical with the epithelial cells of the endometrium (Fig. 9). The disease assumes two distinct forms; thus it may appear as an eroding ulcer penetrating the muscular wall of the uterus, and sometimes even perforating the serous coat. In the common form it gives rise to luxuriant masses of soft succulent and vascular polypoid-like masses projecting into the cavity of the uterus, and this is the variety which used to be termed villous endometritis.

As the diagnosis of cancer of the body of the uterus is largely determined by the assistance of the microscope, it is essential for those who venture to give opinions on this point to be thoroughly familiar with the various abnormalities of the corporeal endometrium, and especially those which are known as glandular polypi.

Although in writings and in clinical work we treat very definitely of cancer of the cervical endometrium and cancer of the corporeal endometrium, it is well to understand that cases come to hand in which, after the uterus has been removed, it is extremely difficult on examining the organ to state positively whether the disease arose in the body of the organ or in the upper segment of the cervical canal (Fig. 10).

The writer is also convinced that there are

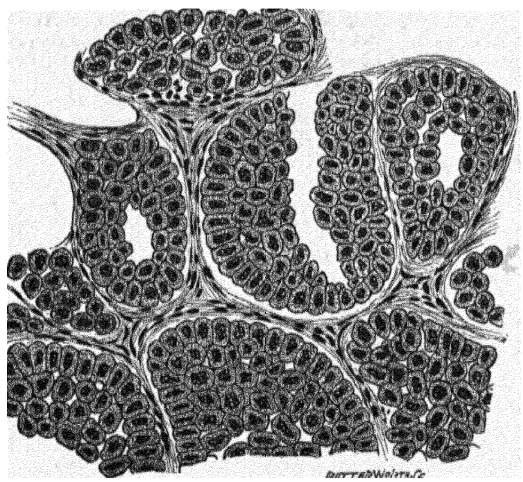


FIG. 9.—Microscopic characters of cancer of the corporeal endometrium: from a scraping. (From the writer's work on *Diseases of Women*.)

two species of cancer arising in the corporeal endometrium, and that these species are not only distinct in their histologic features, but also in their gross characters and clinical aspects (Fig. 11).

The commoner species resembles, and indeed is very similar to that which attacks the cervical endometrium, except in the more perfect columnar type of the epithelium composing the cancer columns. The rarer species is that which has been described as "malignant adenoma" by more than one writer, and this is due to the fact that the normal features of the uterine glands are less distorted than in the common kind of cancer (Fig. 12). It is remarkable in forming large, yellowish-white masses in the endometrium, and causing great enlargement of the uterus.

Cancer of the body of the uterus is unusual before the forty-fifth year; it is most frequent at or subsequent to the menopause. The majority of the cases occur after the fiftieth

year. The majority of the patients are multiparous.

The signs which attract attention are fitful

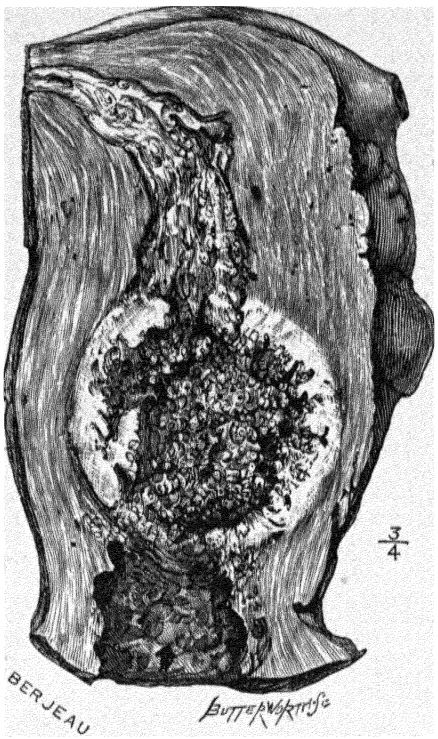


FIG. 10.—Cancerous uterus in sagittal section. A process of the cancer has crept into the right Fallopian tube. It was impossible to determine whether the cancer arose in the lower part of the body or the upper part of the neck of the uterus

vaginal hæmorrhages after the menopause, followed by profuse, offensive, and often blood-stained discharges. The uterus on examination may seem scarcely enlarged; sometimes, however, it may be much bigger than usual. In the majority of cases the diagnosis cannot be established with certainty until the cervical canal is dilated and a fragment of the suspected cancer extracted and submitted to microscopic scrutiny. It often happens that when the cervical canal is dilated in this way the disease may be so advanced that there is no reasonable doubt in regard to the diagnosis, and in such circumstances it is sometimes to the best interest of the patient to complete the treatment by at once removing the cancerous organ. Even should it be decided to await a microscopic examination of the tissue, it is a useful measure to carefully remove with a blunt curette all the exuberant cancerous material, as this checks bleeding and limits decomposition of the necrotic portions of the cancer.

The only successful treatment available for cancer of the body of the uterus is hysterectomy. This operation may be carried out either by the vaginal route or by cœliotomy. The writer has carried out a series of operations by both methods, and has satisfied himself that though the immediate risks of the operation are the same by both methods, the remote consequences are much better when the uterus is removed by the abdomen. It must be remembered that in cases where the cancerous growth is very luxurious the uterus may be very big, and its removal entire through the vagina becomes an impossibility, for it is a matter of the first importance to remove a cancerous uterus entire and without undue handling and squeezing, because the one great danger which women run in the operative treatment of this disease is cancer-infection. When the peritoneum is fouled with cancer material the cells possess so much power of independent growth that large masses of cancer spring up on the pelvic peritoneum in a few weeks and quickly destroy the patient.

When the uterus is removed by the abdominal route the operator is not only able to completely extirpate the uterus, but he can remove the Fallopian tubes, ovaries, and adjacent segments of the broad ligaments. It has also this advantage, that it enables the operator to satisfy himself as to the extent of the disease, and also he can assure himself as to the absence of lymph-gland infection and dissemination. Should either of these conditions be present, then it would be useless to perform hysterectomy. It is a fortunate matter that the prospects of a patient after hysterectomy for cancer of the body of the uterus are infinitely better than in cancer of the cervix, and a number of cases can be brought together where women have had immunity from recurrence for five and even ten years. The great secret of success is early recognition of the disease and prompt treatment.

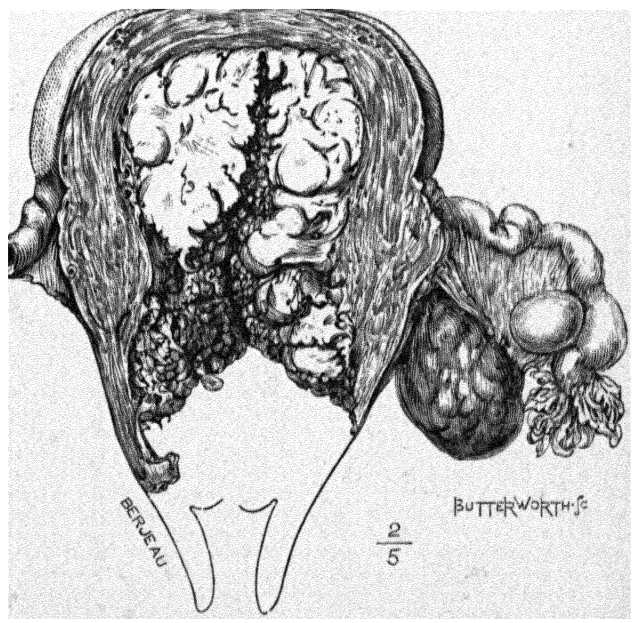


FIG. 11.—Uterus in coronal section: it is occupied with a cancerous mass, portions of which projected as polypoid processes with rounded ends into the cavity of the uterus.

CANCER OF THE UTERUS AND FIBROIDS.—Uterine fibroids are very common, so is cancer of the uterus, and as the maximum of frequency in relation to age is very near in the two dis-

eases it is not a matter of surprise that the two conditions should frequently coexist. In some cases the two diseases may be seen in close proximity without interfering with each other, but when the capsule of a fibroid is invaded by contiguous cancer the tumour ulcerates and sloughs with great rapidity. Cancer of the body of the uterus is more frequently complicated with fibroids than cancer of the cervix, and it is a fact important to bear in mind that a woman who has attained her menopause, and is known to have fibroids, and especially if she be sterile, begins to suffer from irregular "issues of blood"; this may be due to cancer of the endometrium (Fig. 13). It is always a suspicious sign and demands the most careful investigation. The matter may be put in the following aphoristic form:—*When a woman with uterine fibroids, having passed the menopause, begins to have irregular profuse uterine hæmorrhages, it is extremely probable that she has cancer of the body of the uterus.*

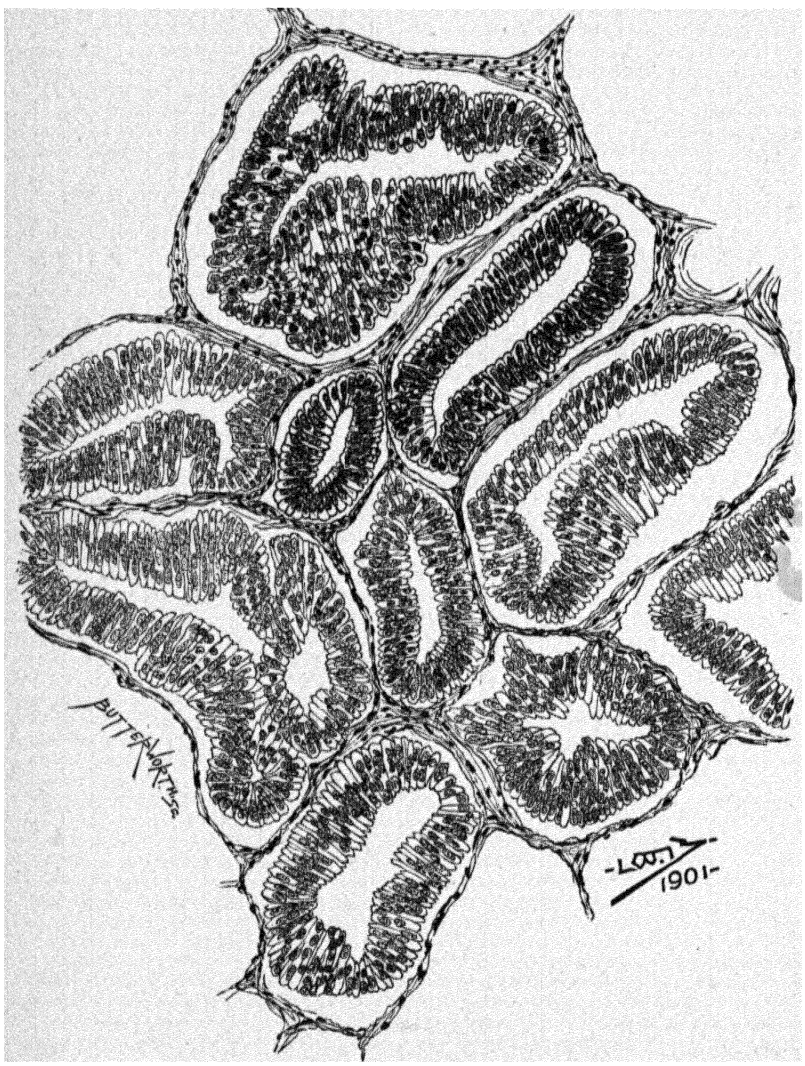


FIG. 12.—Microscopic characters of the "tubular" cancer shown in Fig. 11.

SARCOMA OF THE UTERUS.—The uterus, like other hollow muscular organs, is liable to sarcomata, but it would be difficult, indeed impossible, to state whether these tumours arose among its muscular elements or in the connective tissue of the endometrium. Before 1889 very few accurate facts were available in regard to uterine sarcomata, because it had become customary to describe all malignant tumours of the uterus as cancerous; but at that date Sanger and Pfeiffer independently described a remarkable variety of malignant tumour arising in connection with the parturient uterus, which has led pathologists to give very careful attention to all forms of malignant disease attacking the uterus.

Quite apart from the tumours described by Sanger and Pfeiffer, and which are considered in another article under the heading "Deciduoma" (*see* vol. viii. p. 380), tumours with the characters of round-celled and spindle-celled sarcomata do arise in the uterus. They are, however, very exceptional. In one remarkable case described by Pernice a tumour grew from the neck of the uterus, and had the structure of a spindle-celled sarcoma; many of the spindles presented the transverse striation so characteristic of the tumours known as myosarcomata. The tumour recurred after removal, and the recurrent growth exhibited the ordinary spindle cells common to sarcomata. The disease in this case was rapidly fatal. In the few examples which have come under the writer's observation the tumours arose from the neck of the uterus by a somewhat broad stalk, and then formed dendritic masses in the vagina. In one case the tumour had the bulk of a cocoa-nut. These sarcomata are generally of the spindle-celled species, and the most marked feature is the rapidity with which they recur after removal. The patients are generally women between twenty and thirty-five years of age. This kind of sarcoma growing from the cervix is probably very rare, because the peculiar mass which fills the vagina, the narrow base from which it springs, the extraordinary rapidity with which it recurs after removal, constitute a clinical picture not likely to be mistaken; and they differ so much from the ordinary genera of tumours which attack the cervix that they would be sure to attract attention.

HYSTERECTOMY

Hysterectomy, as the surgical operation for the removal of the uterus is called, can be effected by two distinct methods. In one

access is obtained to the uterus through an incision in the lower part of the belly wall—this is known as *abdominal hysterectomy*; in the other the uterus is extirpated through the vagina, and is termed on this account *vaginal hysterectomy*. It is somewhat remarkable that although the object of both operations is the same, namely, removal of the uterus, in the abdominal operation the surgeon takes every care to thoroughly close the peritoneum over the vaginal opening, but in the vaginal method the gap in the peritoneum, left after the removal of the uterus, is usually left open, and its edges merely fall into apposition.

The results of both methods in the hands of operators experienced in this class of work are among the most remarkable in surgery, and to ensure success the surgeon must exert himself to obtain a *rigid asepsis and perfect hæmostasis*; for this purpose it is essential to be extremely careful in the preparation of the patient for operation, as well as the materials and instruments used during the operation; the surgeon requires, above all things, to be scrupulously clean in regard to his own hands.

The choice of operation, as well as the details of the methods, are those which the writer adopts at the present time, and they are the outcome of several hundred hysterectomies performed for a variety of conditions.

Choice of Operation.—Cancer of the cervix suitable for operation is best removed through the vagina; even when it complicates pregnancy which has advanced to the seventh month, the fœtus and placenta can be extracted, and the uterus successfully extirpated. Fibroids coexisting with cervical cancer, unless they are very large, are not a bar to the vaginal hysterectomy, for they can be enucleated and extracted separately.

Cancer of the body of the uterus sometimes leads to great enlargement of the organ, then it is best removed by the abdominal route, but in a fair proportion of cases it is easily dealt with through the vagina. When cancer of the body of the uterus is complicated with a large fibroid, then abdominal hysterectomy is the only judicious course.

There are conditions in which vaginal hysterectomy is a comparatively simple operation, for example, a small uterus and a capacious vagina; with the converse conditions the operation is often difficult, and it requires experience to enable the surgeon to judge when the relation between the size of the uterus and vagina are favourable or the reverse for the purpose of vaginal hysterectomy. It occasionally happens that an attempt is made to remove the uterus by the vagina, but in the course of the operation it is found impossible to extract the uterus, and the surgeon has to complete the work through an abdominal incision. Under such conditions some surgeons recommend removing the uterus and tumour piecemeal with the aid of the knife and scissors; this is termed *morcellement*. It is unwise to adopt this method with a cancerous uterus for fear of infecting the adjacent parts, and especially the peritoneum, with cancer.

It is better to remove the uterus by the vaginal method when the cancer is in the cervix, because a freer removal of the diseased parts is possible. When the disease is in the body of the uterus, abdominal hysterectomy affords opportunity for a wider removal of the parts than the vaginal method. These are

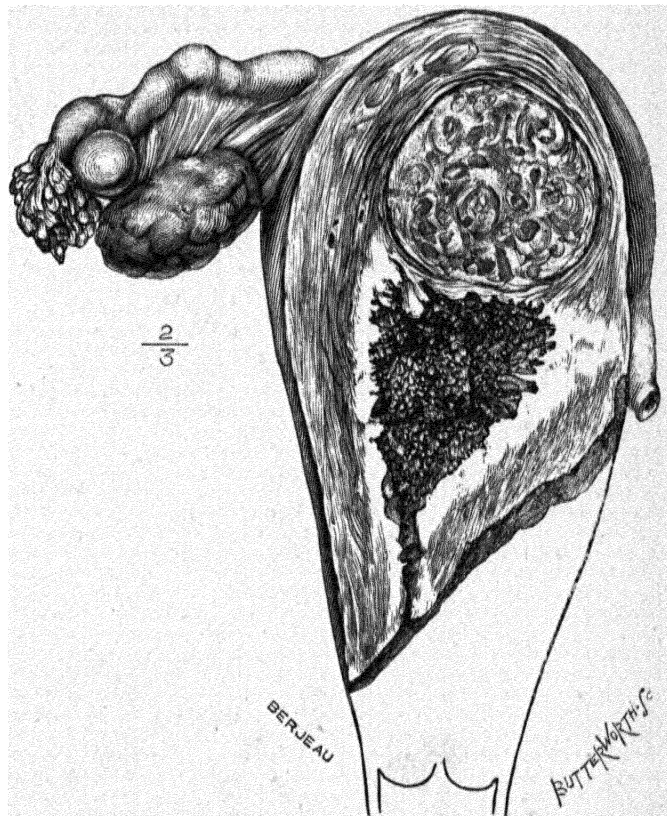


FIG. 13.—Cancer of the uterus complicated with a submucous fibroid. The uterus is shown in coronal section. Removed by abdominal hysterectomy from a sterile spinster.

good working rules, but the latter is the one which is open to the greatest number of exceptions.

In operations of this kind a great deal depends upon the preliminary treatment of the patient, and for this purpose it is essential to have the assistance of a good nurse specially trained in this class of work. Such a nurse understands the methods of washing and disinfecting the vagina and pudenda, is apt at passing the catheter, and without fuss arranges and prepares the patient. It is good practice to have such a nurse in attendance for at least forty-eight hours before the operation, so that she may shave the hair from the pubes and labia, and, after thoroughly washing them with soap and warm water, renders the cutaneous

parts as aseptic as possible with such antiseptic solutions as the operator is accustomed to use. The writer finds a solution of perchloride of mercury (1 in 5000) a very efficient antiseptic agent for the skin. The bowels should be very thoroughly emptied by enemata, and the patient should abstain from food or drink for at least six hours preceding the operation, and the bladder is emptied by a glass catheter immediately before the patient lies on the table. In regard to instruments they should be of metal throughout, as this facilitates the sterilisation, whether by boiling or the application of dry heat.

The ligature material in common use is silk; it can be obtained of any thickness and length, and will stand boiling for half an hour or even longer without impairing its strength. The same is true of silkworm gut. Catgut cannot be boiled, and should be discarded as a ligature material. For dabs the writer employs squares of Gamgee tissue carefully hemmed; they may be cut of any size or pattern, and should be boiled for one hour immediately before use. This material is cheap, capable of easy and efficient sterilisation, and has usurped animal sponges in surgical practice. They are burnt after each operation.

It is unnecessary to give minutely every detail in the preparation and sterilisation of material and instruments, for it may be assumed that no one would undertake such a serious operation as the removal of the uterus without some previous experience in operative surgery; most surgeons have had opportunities of witnessing the operation, or, what is infinitely better, assisting a skilful and experienced surgeon in the performance of hysterectomy.

Abdominal Hysterectomy.—The preparation of the patient is described in the preceding remarks. For the satisfactory performance of this operation the Trendelenberg position is indispensable.

Instruments.—A scalpel; twelve hæmostatic forceps; dissecting forceps; scissors; needles, straight and curved; silks of various sizes; volsella; six dabs of Gamgee tissue; two sponge-holders; silkworm gut. These instruments are necessary, and are quite sufficient for good operators. The instruments should be immersed in warm sterilised water, and the dabs should be washed in water (at 100° F.) during the operation.

The anæsthetic depends on choice; some surgeons insist on chloroform. Ether administered by a skilful anæsthetist is the safest agent yet discovered for prolonged anæsthesia. Artificial temporary paraplegia from subdural injections of cocaine or its allies is yet in the experimental stage.

It is important when the patient is in the Trendelenberg position to ensure that the arms lie parallel with the trunk. This avoids pressure-palsy of the limbs, and prevents the hands and fingers being nipped when the table is restored to its horizontal position.

Steps of the Operation.—When the patient is completely unconscious, and the nightdress and other simple covering are isolated with sterilised towels, the surgeon freely incises the wall of the abdomen in the middle line midway between the umbilicus and the pubes. The actual length of the cut varies with the condition of the patient and the size of the uterus. Fat patients require long incisions, and it is easier to gain access to the uterus with a short incision when the parietes are thin and lax in women who have had children than in those that are barren. The length of the incision has no influence on the result of the operation, but a free incision greatly facilitates the necessary manipulations.

As soon as the uterus is exposed it is isolated from the intestines and omentum by dabs, and the uterus can be seized at the fundus by a volsella and drawn into the wound. Each mesometrium (broad ligament) is then transfixed and ligatured with silk in order to secure the ovarian vessels, or they may be seized with forceps and ligatured at a subsequent stage. When the surgeon wishes to leave the ovaries he secures the mesometrium between the uterus and the ovary; but when he decides to remove the ovaries as well as the uterus, and this is the preferable practice in cases of cancer, the mesometrium is ligatured near the brim of the pelvis on the outer side of the ovary. As soon as the mesometria are divided the uterus can easily be drawn out of the pelvis, and the uterine arteries are usually detected running upwards in the vascular tract on each side of the organ; these should be secured with forceps. A peritoneal flap is then turned down from the anterior surface of the uterus, care being taken not to injure the bladder. A similar flap is fashioned from the posterior surface of the uterus, and then the cervix is carefully enucleated from the upper end of the vagina. In favourable cases it often happens that when the peritoneal connections of the uterus are divided and the organ well drawn up, the mucous membrane attachment of the cervix will invaginate like the finger of a glove; the vaginal mucous membrane is then divided, and the surgeon is able to pass his finger into the vagina. The various bleeding points are secured with forceps, and any free blood or clot occupying the pelvis is removed. The uterine arteries are carefully secured with silk; the ovarian pedicles are transfixed and ligatured with silk as in the operation of ovariotomy. The anterior and posterior peritoneal flaps are brought into apposition with sutures, care being taken not to puncture the bladder. Any oozing points are seized and tied; then the edges of the peritoneal flap are finally secured with a continuous suture of thin silk, which extends from

the ovarian pedicle on one side to that of the other; thus the vagina is completely isolated from the general cavity of the peritoneum, and any serious oozing which may occur finds a ready means of exit through the vagina.

The pelvis is then cleared of clot and dabs; the intestines are allowed to return into the pelvis. The forceps and dabs are carefully counted to insure that none is left in the abdomen. The incision is then closed in triple or single layers, according to the practice of the surgeon. The writer always closes incisions below the umbilicus in three layers: (1) The peritoneum with a continuous suture of thin silk; (2) the fascia with interrupted suture of fine silkworm gut; and (3) the skin with a continuous suture of thin silk. When it is deemed advisable to drain the pelvis, a thin strip of sterilised gauze is introduced into the pelvis and brought out at the lower angle of the incision.

The wound is dressed with a layer of sterilised gauze, and Gamgee tissue maintained in position by means of a manytail of flannel.

The patient is then restored to the horizontal position; all blood and clot is carefully removed from the vagina, and a thin strip of gauze is introduced to conduct away any blood or serum that may subsequently ooze into the vagina. A catheter is then introduced into the bladder, and any urine it contains is withdrawn. The patient is then returned to bed.

After-Treatment.—This is conducted on the same lines as after ovariotomy, and as a rule the convalescence is a quick one.

Vaginal Hysterectomy.—The preparation of the patient has been already described. Instruments: Clover's crutch, duckbill speculum or Auvard's modification of this useful instrument, uterine sound, vesical sound, two volsellæ, scalpel, six hæmostatic forceps, dissecting forceps, three or four long-handled forceps with fenestrated blades, silks of various thickness, needles in handles and curved needles of various sizes, six Gamgee tissue dabs, glass catheter.

Steps of the Operation.—The patient is anæsthetised and secured in the lithotomy position by means of Clover's crutch, and arranged so that the perineum faces a good light. The vagina is then exposed by the duckbill speculum, and the cavity thoroughly irrigated with a solution of perchloride of mercury (1 in 1000), or something equally efficacious.

The operator, seated at a convenient level, passes the beak of the speculum into the vagina and seizes the cervix with a volsella. If there be much sprouting growth it is sometimes useful to detach it with the handle of a knife or a curette, and then thoroughly mop or reirrigate the parts. The assistant empties the bladder with a glass catheter, and then introduces the sound and keeps the surgeon informed of the relation of the bladder to the cervix throughout the first stage of the operation.

Stage 1.—The cervix is drawn down with a volsella, then with the scalpel the mucous membrane on its anterior aspect is transversely divided along a line well above the cancer, but sufficiently low to avoid wounding the bladder, which is then cautiously separated from the cervix with the forefinger or the handle of the knife. When the lower limit of the utero-vesical pouch is reached the peritoneum is divided with scissors and the fingers enter the peritoneal cavity. Throughout this stage the operator constantly informs himself of the exact position of the bladder by manipulating the sound.

Stage 2.—The incision in the mucous membrane is now carried round the cervix by means of scissors, and the recto-vaginal pouch is opened.

Stage 3.—The mesometria are dealt with in the following manner: A needle in handle armed with silk is made to transfix the connective tract close to the side of the cervix in order to avoid the ureter. The object of this ligature is to secure the uterine artery. Frequently the artery is distinctly visible, and is then picked up with forceps and tied by itself. As a rule, when the artery on each side is ligatured and the tissues adjacent to them cut with scissors the uterus can be drawn into the vagina and its fundus brought outside. The remaining upper segments of the mesometria are transfixed with silk and securely ligatured, and the uterus detached. These upper ligatures control the ovarian vessels and surround the Fallopian tubes. Should the ovaries or tubes be diseased, they can at this stage be removed by drawing them into the vagina and securing the pedicles by transfixion with silk. If all the lateral ligatures have been securely applied and hold, there should be no bleeding from the sides, but there is usually some oozing from the anterior and posterior folds. Any vessels which are freely bleeding should be secured with forceps and tied, but in regard to the posterior flap it is an advantage to seize it with a pair of fenestrated forceps and leave them *in situ* for twelve or even twenty-four hours.

The parts are then gently irrigated with warm sterilised water; bleeding points are secured with forceps or ligature, the whole cavity is gently mopped out, and a thin gauze drain inserted to allow any serum or blood to drain away; the bladder is then tested with a sound, the crutch removed, and the patient returned to bed.

The above method does not find favour with all operators; many prefer to compress the mesometria with specially constructed forceps and then cut the uterus away. The forceps

are allowed to remain *in situ* forty-eight hours, and are then carefully removed. Each method has its advocates, and there are advantages and disadvantages associated with both. The employment of clamps materially shortens the operation. The writer, after a careful trial of both methods, prefers to ligature the main vessels and employ the clamps or forceps for the posterior septal flap.

After the operation the anterior and posterior flaps fall into apposition and unite in a few hours, so that it is not necessary to suture the flaps, which is moreover a dangerous practice, for it obstructs the escape of serum and blood, which may ooze even after the most carefully executed operation.

After-Treatment.—This is carried out on the same lines as those adopted for ovariotomy: the vagina needs to be considered in addition; for instance, the gauze may be changed in thirty-six or forty-eight hours. When forceps or clamps are used they require to be taken away with the following precautions:—The patient's thighs are slightly raised and the knees gently separated, then the handles of the forceps are gently unlocked and the blades detached by a gentle twisting movement. Should bleeding occur in attempting to remove a clamp, it should be relocked and left for a few hours longer.

The temperature after vaginal hysterectomy often rises in consequence of the sloughing of the ligatured or compressed tissue: it may rise as high as 103° Fahr. and the discharge become offensive. When this happens the vagina should be gently mopped out with small dabs of wool steeped in an antiseptic solution. By the ninth day some of the ligatures come away, and as a rule most of them are detached by the twentieth day. The patient is usually able to leave her bed about the end of the second week.

The Risks of Abdominal Hysterectomy.—The dangers are those common to all pelvic operations, such as shock, bleeding due to the slipping of badly applied ligatures, injury to the intestines, and particularly the bladder and ureters. Peritonitis may arise from failure in the preparations or contamination from the septic contents of the uterus, or dabs and instruments left in the belly at the time of the operation.

Later complications are suppuration, either in connection with the abdominal wound or from the bed of the cervix; thrombosis of the pelvic veins leading to œdema of the lower limb is a troublesome complication, and embolism has been a fairly frequent cause of death.

Cystitis may arise from two causes: the careless use of the catheter, or suppuration around a ligature involving the bladder, and occasionally actually penetrating this viscus.

Intestinal obstruction may follow this operation, due either to adhesion of the bowel to the stump in the pelvis or to the line of the abdominal incision, but it is fortunately rare.

The Risks of Vaginal Hysterectomy.—The chief of those are injury to the bladder or the ureters, injury to the rectum, sigmoid, cæcum, or small intestine, bleeding due to an artery slipping out of a ligature or the grasp of the forceps and retreating in the tissues.

The Sequelæ.—The more important are vesical and ureteral fistulæ, sepsis, and fatal peritonitis, death from intermediate and secondary bleeding or from suppression of urine due to accidental ligature of the ureters, thrombosis of the pelvic veins and œdema of the limb, embolism, this is rare, and intestinal obstruction due to adhesions, but this is very unusual after vaginal hysterectomy.

Mortality.—The life risks of hysterectomy vary with the skill of the operator and the character of the case. In skilled hands the risk of abdominal hysterectomy for cancer varies from 5 to 10 per cent, in unskilled hands it may reach to 30 per cent.

In vaginal hysterectomy for cancer the life risk in experienced hands varies from 2 to 5 per cent. In unskilled hands the risk to life is not much greater, but the chances of damage to bladder, ureters, and bowel are much increased.

VI. Uterine and Ovarian States, Insanity associated with

There certainly exists a definite relationship between the healthy growth, development, and function of the reproductive organs and the healthy development and discharge of the cerebral and mental functions in both men and women. But in women one can go still further, and say that in many cases full physical and mental development is not complete until the reproductive organs have discharged their function in the reproduction of the race. There is an acknowledged difference between the mental characteristics of the old maid and the mother of a family. It is by no means so certain, however, that there are such diseases as uterine insanity and ovarian insanity. The terms uterine insanity and ovarian insanity do not convey to one any definite clinical picture, as, for instance, the terms mania or melancholia do convey. I do not believe that the most skilful alienist, after an exhaustive examination of the women patients in all the asylums in Great Britain, could classify one of them with any certainty as a case of ovarian or uterine insanity.

There are well-recognised mental symptoms which are regarded as dependent upon certain changes in the uterus and ovaries, and for purposes of reference these conditions can be most concisely mentioned under the following divisions:—

1. Mental symptoms liable to occur during the period of physical development.

2. Mental symptoms liable to occur during the period of full functional activity of the uterus and ovaries.

3. Mental symptoms frequently coincident with the period of the climacteric.

4. Mental symptoms the result of surgical operations (ovariotomy, hysterectomy).

1. *Mental Symptoms liable to occur during the Period of Physical Development.*—During the period of life in which the reproductive capacity in women is coming to maturity (13-25 years) there is a tendency for women with a predisposition to the neuroses to suffer from morbid loss of self-control in speech, habits, and conduct. The various symptoms classified as hysteria and moral perversions are legion, and may range from the slightest cases of exaggerated self-consciousness to symptoms so marked and unnatural as almost to constitute insanity. The marked outstanding symptom most characteristic of this period is impulsiveness, and may present itself in the motor equivalent of hystero-epileptic seizures. None of these mental abnormalities necessarily mean insanity, but they are danger signals indicating a lowered nutritive activity, and may pass into insanity under unfavourable conditions. During attacks of adolescent mania the delusions, actions, speech, and general conduct are often markedly sexual in character, and more especially during the menstrual periods. During such attacks of mania, amenorrhœa is quite common, but this is probably a result, not a cause of the mental disease, as according to Dr. Cardew amenorrhœa is common amongst the senior classes in girls' schools without being accompanied by abnormal mental symptoms.

2. *Mental Symptoms liable to occur during the Period of full Functional Activity of the Uterus and Ovaries.*—Amongst women of a nervous temperament the monthly ovulation tends to produce regularly recurring mental symptoms which, although not sufficiently definite to be regarded as a distinct disease, are classified as the hysteropsychosis. When in addition to the natural strain of ovulation in such cases there is added some organic obstruction to the menstrual flow, misplacement or structural disease of the uterus or ovaries, one would expect to find and does find an exaggeration of all these symptoms. Many of these cases border very closely upon actual insanity, having at each menstrual period transient attacks of depression or morbid suspicions, and in some cases actual delusions and hallucinations, especially hallucinations of smell. The special sense of smell and the sexual functions in both men and women are very closely connected. In some instances this monthly neurosis imprints upon the brain habits which persist after the menopause. I have seen women, long past the climacteric, suffering from regularly occurring monthly attacks of dipsomania, each attack lasting for a few days. The history in each case being that these patients had been accustomed to relieve their menstrual pains with alcoholic stimulants. There are undoubtedly cases of insanity directly due to such menstrual pain; but, firstly, one must remember that there was almost certainly in each case a predisposition to mental instability, and the menstrual pain was merely the last link in the chain of adverse causes which precipitated the attack; and, secondly, such cases of insanity present no special symptoms which would enable a psychologist to diagnose them as being due to uterine or ovarian disease. In what percentage of the women patients in asylums does actual uterine or ovarian disease occur? Does operative treatment benefit cases so affected? These are questions at present unanswerable from statistics in this country. In the asylums in Great Britain and Ireland no systematic examinations of women patients have been made. In the United States of America and Canada, however, it appears to be in some asylums a routine practice to examine the women patients. Hobbs of Ontario states that out of 800 insane women patients 220 were examined, and 85 per cent of these had distinct lesions of the pelvic organs; 173 of these women were operated upon, 42 per cent recovered mentally, 24 per cent were relieved, 32 per cent were not improved. He believes that the introduction of gynæcological surgery into asylums as part of the regular treatment has raised the recovery rate from 33 to 51 per cent.

Ernest Hall states that out of 75 cases of insanity in women in private practice only four were found to be free from uterine or ovarian disease. Out of 38 of these cases operated upon 6 recovered. Dr. Robe states that out of 100 women examined, 40 had lesions of the sexual organs, and he strongly believes in early operative treatment.

The question as to whether operative treatment is justifiable in insane women is often debated with considerable warmth. As a woman suffering from mental disease does not differ structurally from her sane sister, it is reasonable to suppose that treatment considered desirable and beneficial for the one cannot be injurious to the other. The consensus of opinion now is that operative procedure, if required, should be performed just as readily in the insane as in the sane woman, only in the former the consent of the patient herself, if possible, and certainly that of the friends, guardian, or curator, should be obtained in writing. From time to time isolated and striking recoveries are recorded amongst insane women as the result of a gynæcological operation, and the recorder of the case may draw too sanguine conclusions from the example brought prominently under his notice. In connection with such cases it should be remembered that many insane patients recover just as marvellously after attacks of

typhoid, erysipelas, boils, etc., and it is probable in many of these cases recovering after operation, that the recovery was due as much to the reaction following the shock of the operation as to the actual benefit accruing from the surgical interference.

Just at the end of the period of functional activity, and before the actual climacteric falls due, one occasionally meets cases of insanity known as "ovarian" or "old maid's insanity." Dr. Clouston says: "There is really no definite proof that the ovaries are either disturbed in function or diseased in structure in these cases. . . . The disease usually occurs in unprepossessing old maids, often of a religious life, who have been severely virtuous in thought, word, and deed, and on whom nature, just before the climacteric, takes revenge for a too absolute repression of all the manifestations of sex by arousing a grotesque and baseless passion for some casual acquaintance of the other sex, whom the victim believes to be deeply in love with her. . . . Usually her clergyman is the subject of this false belief. Out of several such cases which I can recall, seven have had clergymen as their supposed wooers or seducers. In no case was there the very slightest possible ground for the notion."

3. *Mental Symptoms frequently coincident with the Period of the Climacteric.* — Climacteric melancholia is the classical form of mental disease to which predisposed and neurotic women are liable at the period of arrest of sexual function and uterine involution. Short of actual insanity, there also exist at this period many mental symptoms closely allied to the psychoneurosis of adolescence and the period of fully developed functional activity, with this difference, that whereas the neuroses of adolescence and adult life are not invariably of a depressing nature, the neuroses of the climacteric are depressing and are characterised by depression of spirits, deficient motor power, loss of the love of life, weakening of the social instincts, jealousies, and perverted sexual ideas. The delusion known as "pseudocyesis," or false pregnancy, is said to be common at this period of life in women. As a delusion among the insane I have seen it just as frequently in men as in women, and Dr. Clouston tells me he has very rarely seen it in private practice.

4. *Mental Symptoms the Result of Operations (Ovariotomy, Hysterectomy).* — Evidence as to the effect of gynæcological operations upon the mental condition of patients so operated upon is very conflicting. In nine years I have only seen two cases of acute mania which were apparently due to removal of the appendages on both sides. Both women were nervous subjects of below thirty years of age, and both recovered.

It is recorded that out of 483 women operated upon by Dr. Savage of Birmingham, in whom he removed the appendages on both sides, four became insane. Dr. Keith, apparently, is of the opinion that removal of the ovaries alone does not produce any mental change, but removal of the uterus as well as both ovaries was followed by insanity in 6 cases out of 64.

Lawson Tait stated that he had never seen a case of insanity follow hysterectomy, but, on the other hand, he had actually seen insanity cured and relief from neurotic symptoms follow upon operative procedure for diseases of the reproductive organs.

Utricle or Utriculus.—The uterus masculinus, sinus pocularis, or prostatic utricle (utriculus prostaticus) is the little cavity (Lat. *utriculus*, a little leather bottle) which communicates with the prostatic part of the urethra and lies in the substance of the prostate gland; it represents the posterior ends of the Mullerian ducts fused together; also, a part of the membranous labyrinth of the internal ear.

Uva Ursi.—Bearberry leaves or the dried leaves of *Arctostaphylos uva-ursi* (*Uvæ Ursi Folia*), act as a diuretic medicine and have also an astringent and disinfectant effect on the urinary tract, which is of value in cystitis, especially the gonorrhœal variety; they contain arbutin ($C_{24}H_{32}O_{14}$), a glucoside which yields hydroquinone and glucose, ericolin, urson, and tannic and gallic acids; they must not be prescribed with iron, salts of silver and lead, with alkaloids, or with gelatin; there is one official preparation, the *infusum uvæ ursi* (dose, ½ to 1 fl. oz.). *See* Diuretics; Pharmacology; Prescribing; etc.

Uvea.—The choroid coat of the eye, or the iris, ciliary body, and choroid membrane, taken together; *uveitis* is usually regarded as synonymous with iritis. *See* Choroid, Diseases of (*Irido-Choroiditis*); Iris and Ciliary Bodies (*Congenital Abnormalities of the Iris, Ectropion of the Uveal Pigment*).

Uvula, Diseases of.

Malformations 378
Uvulitis, Acute 379
„ *Chronic* 379
Elongation of Uvula 379
New Growths 379
Paralysis 379

See also Ear, Middle, Chronic Suppuration (*Sequelæ, Facial Paralysis with Deflection of the Uvula*); Facial Nerve, Paralysis of (*Symptoms, Palate*); Nose, Examination of (*Posterior Rhinoscopic View, Uvula*); Pharynx, Examination of the; Pharynx, Chronic Pharyngitis.

Malformations of the uvula are not uncommon. Occasionally it is congenitally absent, though more frequently its absence is due to destruction from ulceration. More or less completely bifid uvula is very common; or two distinct

uvulas may be present, either side by side or one in front of the other. Even a case of triple uvula has been described; a normal uvula in the centre with a rudimentary one at each side.

Acute uvulitis is usually only a part of an acute pharyngitis, though it may occur independently. It arises from exposure to cold, from septic infection, or from traumatism. Most commonly it is seen along with acute tonsillitis and peritonsillar abscess, or in association with tertiary specific or tubercular ulceration. The uvula may become greatly swollen and œdematous, and attain such length and thickness as to cause extreme distress. The writer can recall a case in which the uvula attained the length of the little finger and the patient had sat up all night with his uvula lying on his tongue, as any attempt to lie down brought on a severe laryngeal spasm.

An acutely inflamed uvula may be relieved by sucking ice, or by an application of cocaine (10 per cent to 20 per cent); but if there be much œdema, scarification or amputation gives the most rapid relief.

Chronic uvulitis is practically always associated with chronic pharyngitis or naso-pharyngitis. The uvula is congested and relaxed, or slightly elongated and thickened. Any symptoms complained of in such cases will be found to depend on the accompanying pharyngitis and not on the slight changes in the uvula.

Elongated uvula is frequently only an exaggerated form of chronic uvulitis, in which the congested and hypertrophied uvula has attained abnormal length. In another form there is no congestion or inflammatory change, but merely extreme length of the uvula as a whole, or great prolongation of the mucous membrane beyond the muscular tissue.

The causes of elongation of the uvula are the same as produce chronic pharyngitis. Lennox Browne believes it is specially common in persons who use the voice professionally during catarrhal attacks. The non-inflammatory form is in most instances primarily congenital in Bosworth's opinion.

In the great majority of cases an elongated uvula causes no symptoms, and in any case there is no correspondence between the length of the uvula and the amount of discomfort it may cause. This will depend rather on the irritability of the pharynx and the temperament of the individual. In one patient we see a greatly elongated uvula and yet there is no complaint, while in another a uvula only moderately elongated gives rise to much cough and irritation, which disappear on its partial amputation. It is therefore sometimes difficult to determine how far the uvula is the cause of the symptoms complained of, and we should first seek for, and remove, other sources of irritation before shortening the uvula. No doubt the uvula is frequently blamed for symptoms arising from granular pharyngitis or catarrh of the naso-pharynx, and its removal, being as simple as cutting the end off a piece of string, adopted without justification.

In a small proportion of cases an elongated uvula does produce well-marked and even distressing symptoms. Among these are persistent cough, made worse by lying down; the feeling of a foreign body, giving rise to frequent effort at "clearing" the throat; retching, or even vomiting, on getting up in the morning and after meals; and in rare cases, especially among those who smoke and drink to excess, laryngeal spasm. A few extreme cases are on record where the persistent cough and the emaciation caused by the frequent vomiting of food have led to a diagnosis of phthisis being erroneously made.

Treatment.—Where the uvula is only moderately elongated and the symptoms slight, astringent paints, lozenges, or gargles may be employed. If, however, the symptoms are distressing, and we are convinced the uvula is the cause, it is better to remove the redundant tissue at once. The best method of doing this is by means of the forceps and scissors devised for the purpose.

After applying a 20 per cent solution of cocaine to the parts, the tongue should be well depressed so as to obtain a good view, and the tongue-depressor then given to the patient to hold. Seizing the tip of the uvula with the forceps held in the left hand, it should be gently drawn forwards but not pulled upon, while with one cut of the scissors the excess of tissue is removed. In this way the cut, running from before backwards and upwards, leaves the wounded surface posteriorly. The whole uvula should never be removed, else it may be followed by persistent pain, difficulty in swallowing, and loss of vocal power. Our object should be to leave a uvula of normal length.

The amount of pain which follows this small operation varies greatly in different cases, and may be very severe for some days. All food should be soft and cold, and the patient should talk as little as possible. The sucking of ice is the best means of relieving the pain.

Though the writer has never met with it, quite a large number of cases of severe primary and secondary hæmorrhage after uvulotomy are on record.

New growths of the uvula are rare, with the exception of papilloma, which may occur either as a sessile or pedunculated growth. Other growths which have been met with include angioma, mucous polypus, and primary carcinoma.

Paralysis of the uvula is common after diphtheria, and may occur alone or along with paralysis of the palate.

Uvulaptosis.—A pendulous state of the uvula. *See* UVULA, DISEASES OF.

Uvulatomy or Uvulotomy.—Excision of the uvula, the instrument used for this purpose being a *uvulatome*. *See* UVULA, DISEASES OF.

Uvulitis.—Inflammation of the uvula. *See* UVULA, DISEASES OF.

Vaccination.

INTRODUCTION 380
IMPORTANCE OF SMALLPOX IN RECENT CENTURIES 380
VACCINATION—
History 384
Results and Statistics . . . 388
OBJECTIONS TO VACCINATION . . . 395
INJURIES FROM VACCINATION . . . 395
VACCINATION ACT, 1898 397
Results of 397
SUGGESTED ALTERNATIVES . . . 398
SUGGESTED AMENDMENTS 398

See also CONJUNCTIVA, DISEASES OF (*Exanthematous Conjunctivitis*); PSORIASIS (*Etiology*); SMALLPOX (*Treatment*); SYPHILIS (*Acquired in Children*).

VACCINATION.—The value of vaccination as a protective against smallpox cannot be correctly estimated without an acquaintance with the severity and prevalence of smallpox in the pre-vaccination era. If the evil was a trifling one, legislation in support of its prevention cannot be defended. But if the evil was great, and if experience proves that vaccination protects from smallpox, legislation may properly enforce it on a population of which a very large proportion can never estimate correctly the importance of the danger from which it is intended to save them.

What, then, is the record of history as to the effects of smallpox before it was modified by vaccination? It is unnecessary for our purpose to enter upon this subject from an antiquarian standpoint. The point we have to consider is this, Was smallpox a grave social danger in the centuries immediately preceding the introduction of Vaccination? History proves without doubt that it was. In communities which were in touch with each other, smallpox was essentially a disease of infancy, not because the infection was only taken by children, but because the majority of the adult population were protected by having had the disease in childhood. Of the four great plagues of infancy, measles, whooping-cough, scarlatina, and smallpox, the last was the most common and the most dreaded. It attacked almost all exposed to the infection; it killed at least one in seven of those attacked, in many epidemics as many as one in three.

The type of the disease, then as now, varied much in severity. In all but mild cases it was an illness of the gravest suffering, many of its victims were blinded for life, and a large majority were terribly disfigured. The one ameliorative fact was that the disease was in large measure self-protective. Children who struggled through it did not, as a rule, have it again. They were protected by their early sufferings; so that where smallpox returned at short intervals in a community, as it did in large towns, the adult population was, for the most part, immune, most of them having had it in childhood. The case was different in isolated country districts and out-of-the-way corners of the country, where the greater part of the adult population had never before been exposed to the infection. If in such a locality smallpox was by chance introduced, it was not limited to the children of the community, it attacked all ages equally, in proportion to their exposure to infection. Infection, then as now, depended upon the co-existence of the two factors, the contagious poison and the contact with it of unprotected people. Adults who had not gained immunity by going through the disease in childhood were almost sure, if they moved about the world at all, to meet the contagion at some time and to take it. One of Horace Walpole's correspondents says: "Poetry is as universally contagious as smallpox; every one catches it once in a lifetime at least, and the sooner the better." The immense effect of a large part of the adult population being immune to a second attack of the disease is seen if we compare even severe smallpox epidemics in England in the seventeenth or eighteenth centuries with those which occurred when the disease was introduced into new places where no one had had it previously, and where, therefore, no one was immune. Prescott, in his *History of Mexico*, describes the ravages of the disease in 1520. He says:—

"Maxixca, the old lord of Kascala, had fallen a victim to that terrible epidemic—the smallpox, which was now sweeping over the land like fire over the prairies, smiting down prince and peasant, and adding another to the long train of woes that followed the march of the white men. It was imported into the country, it is said, by a negro slave. . . . The poor natives sought relief in their usual practice of bathing in cold water, which greatly aggravated their trouble. It spread rapidly over the country to the Astec capital where Montezuma's successor fell one of its first victims. Thence it swept down towards the borders of the Pacific, leaving its path strewn with the dead bodies of the natives who, in the strong language of a contemporary, perished in heaps like cattle stricken with the murrain. . . . So great was the number of those who died of this disease that there was no possibility of burying them, and in Mexico the bodies were thrown into the canals then

filled with water, till the air was poisoned with the stench of putrid bodies."

Catlin, in his *History of the North American Indians*, mentions that of a population of 12,000,000, 6,000,000 fell victims to smallpox. In another place he says: "Each tribe has had this exotic disease in turn, and in a few months it has lost one-half or more of its numbers."[1] In 1707 an epidemic in Iceland is said, from records furnished by the Government of Denmark, to have killed 18,000 out of 50,000 of the inhabitants, *i.e.* 36 per cent. In Greenland in 1734 an epidemic is believed by Sir John Simon to have destroyed two-thirds of the whole population. In Mexico and Greenland there is no history of any earlier epidemic; in Iceland there had been none for a number of years, probably not since 1616. Therefore in all three cases no considerable section of the population would be protected by a previous attack. How severe a scourge smallpox was towards the end of the seventeenth century is plainly seen in the pages of Evelyn and Pepys. From 1684 to 1695 Evelyn's Diary abounds in references to the disease and to its terrible results.

"*December* 1684.—The smallpox very prevalent and mortal.

"*7th March* 1685.—My daughter Mary was taken with smallpox, and there soon was found no hope of her recovery. A great affliction to me."

A few days later there is mention of her death:—

"Oh, dear sweet child! how shall I part with all this goodness and virtue without the bitterness of sorrow and reluctancy of a tender parent? . . . Never can I say enough; oh dear, my dear child, whose memory is so precious to me. . . . Thus lived and died. . . . The joy of my life."

Three months later it is noted that —

"Mr. Hussey, who made love to my late dear child, died now of the same cruel disease.

"*27th August* 1685.—My daughter Elizabeth died of the smallpox soon after her marriage.

"*15th April* 1686.—The Archbishop of York now died of smallpox.

"*December* 1694.—The smallpox increased exceedingly, and was very mortal. The Queen died of it on the 28th.

"*13th January* 1695.—The deaths in London by smallpox increased to 500 more than in the preceding week."

This was for London only. The epidemic in London, in the winter of 1901-1902, with a population of 5,000,000, killed, from August 1901 to February 1902, 600 people; but in Evelyn's time the increased mortality from one week to the next was said by him to be 500, and this in a population of probably less than one-twelfth of the present number in London.

Pepys's Diary tells the same sad story. The history of the deaths caused by smallpox among the royal families of Europe is a further confirmation of its great prevalence and severity. Mention has been made of the death of Queen Mary in her thirty-third year. The king also lost from the same cause both his parents, an uncle, and two cousins. He had it himself very severely, and recovered with a constitution damaged for life. In Austria it killed Joseph I., and in the eighteenth century two empresses, six other members of the imperial family, an Elector of Saxony, and the last Elector of Bavaria. Also a Dauphin (1711) and a King (1774) of France, a Queen (1741) of Sweden, a Queen of Naples, and an Emperor (1727) of Russia. In fact, the high mortality in royal and aristocratic families illustrates the danger of trying to keep children away from the contagion in their youth. The risk of infection in later life was greatly increased by the care exercised to preserve the children of great families in early life.

Horace Walpole's letters are full of references both to smallpox and inoculation.

"Lord Dalkeith is dead of the smallpox in three days. It is so dreadfully fatal in his family, that besides several uncles and aunts, his eldest boy died of it last year, and his only brother, who was ill but two days. . . ."—Vol. ii. p. 203.

"Lord Waldegrave is just dead of the smallpox. . . . His brother and sister were inoculated, but it was early in the practice of that great preservative, which was then devoutly opposed; he was eldest son, and weakly."—Vol. iv. p. 65.

"A very curious list might be compiled of the heads of great families left without heirs-male by the smallpox; the two most remarkable are the familiar friends Queen Anne and the Duchess of Marlborough."

In vol. vi. Walpole comments on the death of Louis XV. from smallpox, and on the devotion of his daughters who tended him, and who both caught it. On page 95 he notes, "The whole Blood Royal of France is now recovered from the smallpox." In an earlier volume he says, "Prince George (afterwards George III.) is ill of smallpox"; and on page 327, "Lord Beauchamp, only son of the last Duke of Somerset of that branch, died of smallpox after a four days' illness.

Ben Jonson's epigram may also be quoted:—

> Envious and foule disease, can there not be
> One Beautie in an age and free from thee?

Macaulay, in his essay on Sir William Temple and Dorothy Osborne, says:—

"Poor Mistress Osborne fell ill with smallpox, and though she escaped with her life, lost all her beauty. To this most severe trial the affection and honour of the lovers of that age was not unfrequently subjected. Our readers probably remember what Mrs. Hutchinson tells

[1] Vol. ii. p. 150, revised edition.

us of herself . . . how her beloved colonel 'married her as soon as she was able to quit the chamber, when the priest and all that saw her were affrighted to look on her.'"

If, leaving these general statements as to the prevalence of smallpox, we try to ascertain the actual percentage of the population of London or any other town that died of the disease before the vaccination era, we are met by the fact that there was in London no registration of births till early in the nineteenth century; and that the only record of deaths up till the year 1838 were the bills of mortality or lists of burials kept for all the parochial burial-grounds. The London bills of mortality were first compiled by order of Thomas Cromwell about 1538, and the systematic keeping of them was begun by the Company of Parish Clerks in 1593. Each parish clerk was directed to bring "to the Clerks' Hall, weekly, a note of all christenings and burials." Obviously, calculations on such a basis cannot pretend to have any minute accuracy. The records were, however, carefully studied by Sir John Simon, and the conclusion he arrived at was that from 1660 to 1679 smallpox in London had killed anually 4170 per million of inhabitants, and that from 1746 to 1755 the rate was about 3000 per million. To estimate correctly the smallpox mortality rate for all England is still more difficult. It would certainly be lower than in London, and in some isolated parts of the country it might be entirely absent for a long term of years. Some of the provincial towns were in advance of London in the matter of registration; and here and there it is possible to study the mortality caused by smallpox towards the end of the eighteenth century with considerable precision. In Glasgow it was 4700 per million; in Manchester, 4300; in Liverpool, 6400; in Chester, "a place of incredible healthiness," 3300; in Boston, 2700: all per million of population. Occasionally we can get the exact record of a given year. At Leeds, for instance, in 1798 more than one-third of the total mortality was caused by smallpox. The numbers are 272 out of 779.

In 1801, when the first census was taken, London, with less than three-quarters of a million of inhabitants, had a smallpox mortality of over 3000. A census exists of the small town of Ware, taken soon after an epidemic in 1722, and with the special view of recording the facts as concerning smallpox—in itself good evidence of the importance of the disease. The total number of inhabitants was 2515. Of these, 1601 had had the smallpox before the recent epidemic; 612 had just had it; 72 had died; and the rest of the entire population (302) are described as "those who have to have their smallpox." That accounts for the entire population of Ware. Evidence abounds pointing to the almost universal liability to the disease. Advertisements for servants mention that they must have had the smallpox. Sir Gilbert Blane told a Committee of the House of Commons that at the end of the eighteenth century there was scarcely an adult person to be met who had not had the smallpox. It even came to be considered a grave disadvantage not to have had it in childhood. Sir Gilbert Blane spoke in this sense to a Committee of the House of Commons. He said:—

"Such as have not had the smallpox in the early period of life are not only rendered unhappy, but likewise in great measure unfit for sustaining many of the most useful and important offices. Few people would choose even to hire a servant who had not had the smallpox, far less purchase a slave who had the chance of dying of this disease. How could a physician or surgeon who had never had the smallpox himself attend others under that malady? How deplorable is the situation of females who arrive at mature age without having had the smallpox! . . . How often is the affectionate mother forced to leave her home and abandon her children at the very time when her care is most necessary! Yet should parental affection get the better of her fears the consequences would often prove fatal."

In the Final Report of the Vaccination Commission we read, on page 13:—

"Perhaps the most striking evidence in favour of the conclusion as to the great prevalence and high mortality of smallpox in Western Europe in the eighteenth century and earlier, is to be seen in the fact that the chance of taking the disease and of dying from it was made the subject of mathematical treatment by distinguished mathematicians of the time. Thus Daniel Bernouilli, writing in 1760-65, takes as one of the bases of his calculation the datum that smallpox carries off the $\frac{1}{13}$th or $\frac{1}{14}$th part of each generation."

We may compare this estimated rate of the eighteenth century's smallpox mortality with that of scarlet fever at the present time. For twenty years, from 1875 to 1894, the average annual mortality from scarlet fever in England and Wales was 430 per million, which is about one-ninth of the smallpox mortality in the pre-vaccination century as estimated by Dr. Farr. It must always be remembered, moreover, that those who escaped with their lives from a bad attack of smallpox had gone through a time of great suffering. Few illnesses are as painful as smallpox of even moderate severity. No doubt epidemics varied very greatly then, as they do now, in the severity of their type of the disease; but it is apparently well within the mark to say that in England generally smallpox killed over 2000 per million in each year, and that in London and other large towns the rate was often over 4000 per million. Let us see now what vaccination has done to reduce this terrible mortality.

The first attempt to do anything systematic in England against smallpox was made in 1720. It had long been known in China that a much milder form of the disease could be produced by putting some of the variolous matter under the skin of the person to be infected. This process was known as "buying the smallpox." Dr. M'Vail has found the same expression in Wales and in various parts of Western Europe, and he thinks it likely that the practice of inoculation had been introduced from the East into scattered parts of Western Europe, and had by degrees been forgotten and disused. It is obvious that the practice was likely, in careless and ignorant hands, to do at least as much harm as good. The credit of the reintroduction of inoculation into Europe belongs to Lady Mary Wortley Montagu, whose husband was at that time ambassador at Constantinople. She heard of the method, and after considerable inquiry into its results she made it known in England. Smallpox being then one of the most certain sources of every general practitioner's income, she thought the medical profession would be her most vigorous opponents. Nowadays we often hear that doctors support vaccination because it adds to their income; it is forgotten that the earnings from this source are insignificant compared to those obtained from attending patients through the illness of smallpox. Lady Mary writes thus to a friend:—

"I should not fail to write to some of our doctors about it if I knew any of them that I thought had virtue enough to destroy such a considerable branch of their revenue for the good of mankind. But that distemper is too beneficial to them not to expose to all their resentment the hardy wight that should undertake to put an end to it. Perhaps, if I live to return, I may, however, have courage to war with them."

Lady Mary had her little daughter inoculated in England, and the practice was slowly brought into fashion. But the early results were not very satisfactory. Up to 1728 only 897 cases were inoculated in England, and of these 17 died, so that the induced disease was evidently not always a slight affair. This was disappointing, as safety had been promised. During the immaturity of a new idea or practice there is always a crowd of timid folk who condemn every novelty that does not come before the world in its final and perfected form. Few people have knowledge or insight enough to be patient with that which is new and in its developmental stage. Inoculation was quite out of fashion from 1728 to 1740, when it was again revived. Two things were obviously against its general usefulness. The inoculated disease was not always mild, and it was contagious. Gradually, as time went on, it was found possible to guard against these two disadvantages. By taking the variolous lymph earlier in the disease, by introducing it under the skin in a better way, and by careful regulation of the patient's life before the operation, the illness was made exceedingly light. Inoculating for smallpox became a special part of the medical art, and men practised it almost to the exclusion of everything else. About 1763, for instance, two brothers named Sutton enjoyed immense popularity as safe inoculators. They boasted of having had upwards of 10,000 cases with no deaths. The other disadvantage of inoculation was met by isolating patients with the inoculated form of the disease so carefully that persons not immune from having had smallpox had nothing to do with them. With an adult population who were largely protected from the disease by having had it already, the necessary quarantine was not impossible, but it required to be accepted and enforced in a thorough-going way. Children and young people who were inoculated had to be segregated and surrounded by immune adults for several weeks. The Scotch people, with their excellent sense, and with the attention to detail which has conduced so much to their reputation in professional and commercial life, as gardeners and cooks, etc., seem to have been able to get all the good that was possible out of inoculation then, as for the most part they do now out of vaccination. Dr. M'Vail brought under the notice of the Commission an old book, Sir John Sinclair's *Statistical Account of Scotland*, which extended from 1790 to 1799. Sir John had asked the parish ministers of Scotland for information on a number of details about their parishes. He did not ask about smallpox, but many of his correspondents mention inoculation as a valuable means of protecting the population from the disease. People who are opposed to vaccination often assert that the prevalence of smallpox at the end of the eighteenth century was due to its spread by inoculation. In careless hands this might be the result, but Sinclair's book proves that it was not the rule in Scotland. Among the many statements in favour of inoculation a few may be quoted.

"The former virulence and ravages of smallpox are much abated in this parish (New Abbey, Kirkcudbright) owing to the ministers performing the operation of inoculation to a considerable extent, and with the greatest success." At Portingal in Perthshire "it may be added that fewer children die in the Highlands than almost anywhere, especially since inoculation has been so universally practised, which it has been for a good many years back, to the saving of many lives." At Kilmuir, Skye, "in former times the smallpox frequently prevailed to a very great height, and sometimes almost depopulated the country. The people in general are now so well convinced of the propriety of inoculation that it is become the practice universally, and many useful lives are saved by it." At Durness,

Sutherlandshire, "the smallpox used formerly to cut off great numbers of the children, but inoculation was introduced here more than thirty years ago . . . and it has been attended with the greatest success."

In one or two places permanent increase of population was attributed by the writers to the practice of inoculation. At Largo, Fifeshire, it is stated, "Few children are now cut off by smallpox, as inoculation is generally introduced with remarkable success. During twenty years' practice our surgeon has not lost one patient." These statements were made by parish ministers relating the impression the facts had made on their minds. They wrote independently of each other, and their testimony is worthy of attention.

Horace Walpole[1] may again be quoted: "Oh, your poor young Queen of Naples who has got the smallpox and will lose her beauty, if not her life! How much stronger superstition and prejudice are than maternal love, when all these deaths cannot open the Empress Queen's eyes (Maria Theresa) in favour of inoculation! But she has escaped herself, and that will close them faster than ever." And again, "I wonder all the princes of Europe are not frightened *into* their wits—why they die every day! and might avoid it, most of them, by being inoculated. Mr. Sutton would ensure them at 12 pence a head. He inoculates whole counties, and it does not cause the least interruption in their business. They work in the fields, or go up to their middles in water, as usual. It is silly to die of such an old-fashioned distemper!" In another letter Walpole speaks of "his zeal for the cause of inoculation." George's III.'s eighth son, Octavius, died in a convulsion on the sixth day after inoculation. Walpole says that even though it may be true that the death was due to the operation, "is one child lost an argument against millions preserved? Did not another child die, also from convulsions, two years ago, who had not been inoculated?" The era of inoculation, however, was not a long one. It was destined to be superseded by vaccination.

In the year 1769 Edward Jenner, a medical student from Gloucestershire, came to London and lived with John Hunter as one of his house pupils. Hunter was always questioning Nature in one direction or another. He had a genius for observing facts, and for putting them into their true relation with each other. To this questioning and observing man Jenner reported that in the midst of an epidemic of smallpox a dairymaid had said confidently to him, "I shall not take smallpox; I have had cowpox." What did this mean? Hunter said in answer, "Do not think, try; be patient, be accurate"; meaning, no doubt, be sure of your facts before you theorise upon them, and when you are sure test them experimentally. We know now that the tradition which Jenner heard from the dairymaid was common not only in Gloucestershire, but in many other parts of England, Ireland, and the Continent. It has been traced in eighteen English counties. The practice of inoculation was practically an experiment on a large scale testing the truth of the tradition. Whenever smallpox was introduced into a dairy or cheese-making district, hundreds of people would seek the protection of inoculation. Every practitioner would look carefully into the causes of failure, when he had inoculated without success. He heard in many cases that the subject of the operation had had sores on her hands, contracted from the cow, and that the dairy people believed these sores protected against smallpox. If this happened again and again, and the dairymaids who could not be inoculated did not get smallpox when exposed to it, the practitioners would gradually begin to respect the traditional belief. So strongly was this tradition held that we now know of a farmer named Jesty who actually took lymph from a cow and inoculated his wife and children with it to protect them from smallpox, and that this was done before Jenner's memoir appeared. Dr. M'Vail quotes from Pearson's *Inquiry into the History of Cowpox*, much evidence of the wide diffusion and strength of the tradition about cowpox.

"Mr. Rolph says there is not a medical practitioner of even little experience in Gloucestershire, or scarce a dairy-farmer, who does not know from his own experience and that of others that persons who have suffered the cowpox are exempted from the agency of the variolous poison. . . . So, too, Mr. Bragge, many years before, had inoculated over fifty people, of whom three had had cowpox, and these he therefore charged with abundance of matter, but to no purpose."

Dr. Seaton, in his Handbook, refers to a German newspaper dated 1769 in which mention is made of cowpox being often seen about Göttingen, of milkers being affected by it, and of "their being protected thereby against smallpox." From the time Jenner heard of the tradition he pondered over it. He lived the life of a busy country doctor for thirty years. But about midway in this time, in 1780, his pondering led him to a new idea in connection with cowpox, and it is upon this idea that his fame is based. It flashed upon his mind that if the tradition referred to was true, the protection from smallpox might possibly be transferred directly from one person to another, and then that every one might be made immune to the poison of smallpox. Can we not imagine the delight that such a conception as this would bring to one who knew, as every doctor did then, the misery and suffering caused by smallpox? Jenner was afraid to mention his idea publicly; but one day, riding with his intimate friend Gardiner,

[1] *Horace Walpole's Letters*, vol. v. p. 71.

from Gloucester to Newport, he confided it to him. Gardiner said that Jenner did so "with deep and anxious emotion," and that he said, "Gardiner, I have entrusted to you a most important matter which I firmly believe may prove to be of inestimable value to the human race." He added, "Say nothing of this; if it fails, all would laugh at me, and something untoward may happen." Year by year he brooded over the idea, and watched for an opportunity of testing its truth. Finally, sixteen years later, his chance came. In May 1796 he was able to take vaccine lymph from a person who had contracted cowpox, and with it to inoculate a boy who had not had smallpox. Vaccine vesicles resulted. Six weeks later he tried to inoculate the boy with smallpox lymph, and it failed. He wrote to Gardiner to tell him the result of the experiment, and added, "But now comes the most delightful part of my story. On July 1st the boy was inoculated for smallpox, but without any result." After this Jenner went on passing the vaccine lymph through a succession of people, and then inoculating them for smallpox, always with the same result; those in whom he had produced vaccine vesicles did not respond to the smallpox inoculation—they were immune to the contagion. Two years were thus occupied; then he came to London and published his *Inquiry into the Causes and Effects of the Variolæ Vaccinæ: a disease discovered in some of the western counties of England, particularly Gloucestershire, and known by the name of Cowpox.* It is worth noting that Jenner boldly called cowpox smallpox of the cow; also that he did not claim to have had any share in its discovery. In the hundred and more years since the *Inquiry* appeared, after endless controversies on the subject, Jenner's name for cowpox has held its place. We believe, as he did, that the two diseases, cowpox and smallpox, are essentially from one stock, and that either in cowpox the virus has been attenuated by passing through the constitution of the cow, or that it has become much more virulent by passing through the system of the human. With the exception of a few cranks, the whole of the civilised world accepted the boon Jenner had been able to offer it with enthusiasm and joy. Every one knew then what smallpox was; almost every one had lost relatives from it; a still larger number had seen the fair young faces around them disfigured for life by its ravages. Honours poured in upon Jenner. Almost every crowned head in Europe did him honour. We hear of his being asked to appeal to Napoleon on behalf of a Gloucestershire man taken prisoner in the wars. As Napoleon wrote the order of release he said, "I can refuse nothing to *that* man." The great majority of mankind, when Jenner had established his facts, welcomed with enthusiasm the deliverance he had brought them. Much, no doubt, remained to be learnt about vaccination, and on one important point Jenner himself went wrong. He thought the protection acquired through the agency of vaccine lymph would prove to be as lasting as that conferred by having had the smallpox itself. The early observers had to find out when to take the lymph from the vaccine vesicle, how soon after vaccination its protective influence would be felt, if one vesicle was as efficacious as three or four, and if the modified disease was ever in itself a source of danger. Probably the importance of each of these points was only gradually appreciated. The attention of every one was fixed on the point of greatest moment, viz.: "Have we in vaccination a possible mode of escape from the demon of smallpox?" They would not be able at first to think of much more than this.

One of the most interesting episodes in the early history of vaccination was the method adopted to convey the newly found boon to the Spanish-American dominions. The ravages due to smallpox at the time of the conquest of Mexico were still remembered. Other severe epidemics had frequently occurred. The difficulty of conveying the lymph across the Atlantic was considerable. It was not the age of swift steamers, cold chambers, or capillary tubes. Ingenuity had to find a way. Children who had not had the smallpox were sent to Spanish America, one or two being vaccinated each week during the voyage, and in this way the lymph arrived at its destination in a fresh and active condition. The supplement to the *Madrid Gazette* dated 14th October 1806 contains the following account of the expedition which had started from Corunna on 20th November 1803: "On Sunday, 7th September last, Dr. Francis Xavier Balmis, surgeon extraordinary to the king, had the honour of kissing his Majesty's hand on the occasion of his return from a voyage round the world, executed with the sole object of carrying to all the possessions of the Crown of Spain situated beyond the seas the inestimable gift of vaccine inoculation."

"The expedition consisted of three frigates, with several physicians and twenty-two children who had not had the smallpox. . . . The children, many of whom were very small, were placed under the care of a matron, and the greatest attention was paid to their cleanliness and comfort. At New Spain they made a fresh start with twenty-six more children. Material for vaccination was lavishly distributed through the northern part of Spanish America, and in each capital a central society was formed. Half of the expedition went to the Philippine Islands, Macao, and Canton, and the other half to Peru. This section was wrecked, but happily the subdirector, his three physicians, and the children came to no harm. The expedition was often publicly received by the bishops, military

governors, and persons of the greatest distinction, who took into their arms the little children who were to carry the cowpox to the indigenous Americans and the Malays of the Philippine Islands, and returned thanks to God for having been the witnesses of so happy an event."

If we now inquire what was the immediate effect of vaccination in the early years of the nineteenth century, we must remember that throughout the civilised world there were two influences which aided vaccination then more powerfully than they have ever done since. Every adult person at that time knew from personal or family experience what smallpox was. Every one had lost relatives by it; he had seen with his own eyes what it involved. There is no such lesson as to the value of vaccination as an experience of smallpox; it was a lesson which all had learnt. Moreover, the adult population was in a very large measure, as has already been said, protected by having had the disease in childhood. It was not therefore necessary to vaccinate every one. If the children were vaccinated, a large proportion of the entire population was protected. These two influences worked powerfully in support of vaccination in the early years of the last century. There seem to have been few or no vaccination laws. Fear of smallpox influenced the majority of parents; careless and indolent people were successfully controlled by public opinion and by indirect pressure. In Copenhagen, for instance, in the twelve years before vaccination was practised, an average of 458 persons each year had died of smallpox. From 1802 to 1818 the average mortality dropped from 450 to 9·8 in spite of the absence of direct compulsion in support of vaccination. No child was admitted into the public schools, no one could be confirmed or apprenticed to a trade, no one could be married, without proving that he had either been vaccinated or had had the smallpox. Auspach, in Bavaria, furnishes a similar example. Here the annual average mortality from smallpox had been 500. In 1800 there was a severe epidemic which killed 1609, and which no doubt also protected from future attacks some 5000 or 6000 persons who had survived the illness. In the next eleven years, by the aid of infantile vaccination, there was a total smallpox mortality of five only. Much the same thing occurred in many English towns. Parental anxiety to escape an evil so familiar and so justly dreaded, led to a large amount of infant vaccination, and the adult population was in considerable measure immune. At Norwich, for example, from 1810 to 1813, there was scarcely any smallpox, not a single case from 1813 to 1818. Gradually the knowledge and memory of smallpox died out, the anxiety about it disappeared, the public appreciation of the immense value of infant vaccination diminished, the children vaccinated ten or fifteen years earlier were outgrowing their protection, the adults who had had smallpox in their youth were passing away, and as soon as the contagion was introduced a large number of unprotected people were prepared to receive it. The Norwich epidemic of 1819 was a severe one. Cross estimates that one-thirteenth of the whole population suffered; there were 530 deaths.

This brings us to the question, How far is the protection of vaccination absolute and lasting?

It is a familiar fact about almost all the contagious fevers, that, as a rule, one attack protects against subsequent ones. How this immunity is gained no one even now knows with precision. A few years ago it was supposed that in the course of each of these illnesses something was removed from the body, in the absence of which the contagious germs could not again develop. The more modern view is that in each of the self-protective contagious illnesses an antidote is formed in the course of the development of the tens of millions of germs which cause the illness, and that the antidote remains, and is the protective agent against future attacks (*vide* art. "IMMUNITY," vol. iv.). The antidote has in fact been produced by the development of the germs, and it is owing to its presence that the illness in question comes to an end. The antidote is probably chemical in nature. It is certain that the poisons of many contagious diseases can be grown of varying strengths; they can by one method be made more virulent, and by another less so. There is a large amount of evidence in support of Jenner's original view that smallpox and cowpox both come from the same poison; human smallpox being probably a very virulent development of a milder disease common to several animals. Several good observers have succeeded, though not without difficulty, in inoculating calves with human smallpox, and in getting, after a few removes, vesicles which yield lymph that protects from smallpox. The opponents of vaccination constantly dwell upon the supposed absurdity of preventing one disease by the induction of another, and a quite different one. Their argument falls to the ground if an essential and close relationship has been proved to exist between human smallpox, cowpox, and its analogue in the equine race known as horse-grease. In the early efforts to prove, experimentally, the pathological identity of these three diseases, the great majority of the results were entirely negative. Considering how much commoner cowpox was a century ago than it is now, it is likely that the animals upon which the variolous inoculation was attempted were often immune, from having already had cowpox. Basil Thiele of Kasan twice, in 1836 and in 1838, however, succeeded in inoculating a cow with variolous matter, and in producing cowpox; Ceely in

1839 did the same; also Badcock in 1840. In 1852 the American physicians Drs. Adams and Putnam again succeeded, and the vaccine lymph thus obtained was extensively used in Boston and its neighbourhood. In 1865 a Commission, appointed by the Society of Medical Sciences at Lyons, and presided over by Chauveau, seemed to arrive at results at variance with those of the earlier observers. In their experiments the inoculation of cows with variolous matter produced not a vesicle but a papule; this papule could not be produced in an animal which had had cowpox, either naturally or artificially; also in an animal on which a papule from variolous matter had been developed cowpox could not be induced. The lymph from the Lyons papule, when used on the human subject, induced not cowpox but smallpox, and this result was seen even in a second remove. Somewhat earlier similar results were obtained by Martin of Boston, U.S.A., as a large number of persons "vaccinated" by him from vesicles raised on a cow's udder by inoculation of variolous lymph were nearly all attacked with smallpox, and three died.

In 1885, and again in 1892, Dr. Simpson produced lymph by inoculating calves with variolous lymph. It appears that success much more often follows when young calves are used rather than mature animals, and when the variolous lymph was taken not later than the fifth day of the rash.

In several cases the tendency of the lymph to produce a generalised eruption was seen only in the earlier removes from the human, and disappeared after passing the lymph several times through calves. "Surgeon-Major King in 1889, being then stationed at Madras, inoculated a young bull-calf with fifth-day smallpox lymph obtained from a child suffering from the disease. On the eighth day after the inoculation a crop of vesicles made their appearance not at points of insertion, but some little distance away; from the lymph obtained from these vesicles another calf was then inoculated. In this calf, again, secondary vesicles also formed at each point of insertion of the lymph. The lymph stock was carried on through three more generations in calves, the vesicular eruption being now in each instance strictly localised to the points of insertion. With lymph obtained from the fifth calf of the series a number of children were vaccinated successfully."[1] It appears probable that the apparently negative results in many of the earlier experiments were less negative than were then supposed to be. The vaccine vesicle is not as a rule seen the first time variolous matter is introduced into a calf. In many instances it has appeared only in the fifth or sixth passage through calves. Dr. Copeman's own experiments go to confirm this view. Even in the calves which showed no vesicles, subsequent vaccination with vaccine lymph produced no effect, and the earlier calves in each series from direct variolous inoculation had papules without definite vesiculation. Dr. Copeman says:—

"I have thus far obtained an undoubtedly successful result in one series only out of four attempts; but I have at any rate been able to satisfy myself that it is possible to variolate the calf, and, further, that the result obtained in the first instance may become greatly modified in the course of successive removes; and, again, that animals which have been thus treated are no longer susceptible to vaccination."

He adds (p. 64):—"If it can, as I believe, be conclusively proved that smallpox lymph, by passing through the system of the calf, is so altered in character as to become deprived of its power of causing a generalised eruption, while inducing at the site of inoculation a vesicle indistinguishable from a typical vaccine vesicle; and, more important still, if it be shown that when transferred again to man it has by such treatment completely lost its former power to produce a general disease, it may be fairly asserted that cowpox—or, rather, that the artificially inoculated form of the disease which we call vaccinia—is nothing more nor less than smallpox modified by transmission through the bovine animal."

It seems, therefore, to be all but proved that cowpox is only much attenuated smallpox, and that the protection it gives is due to the manufacture within the system of the vaccinated person of the same antidote, the presence of which, after an attack of smallpox, brings the disease to an end and prevents, in the majority of persons, a second attack. It is, however, well known that in a small minority even the protection afforded by a previous attack of smallpox fails, and the disease is again taken. Louis XV. died of a second attack at the age of sixty-four, having had his first attack of the disease fifty years earlier. The German Vaccination Commission in 1884 considered that the proportion of second to first attacks was about 1 in 636 cases. It is, however, certain that the protection derived from vaccination does not last as long as the protection from the survived disease, the amount of antidote formed being even in efficient vaccination much less than in smallpox itself. The antidote may vary, too, in more than one way; it may be poor in quality and deficient in quantity. It is probable that it is formed round each vaccine vesicle, and, if so, there would be four times as much from four vesicles as there would be from one, and much more from a large vesicle than from a small one. The degree of protection from vaccination would, therefore, vary with the number and size of the vesicles, and with the length of time which has elapsed since the vaccination. It is highly probable, too, that

[1] Copeman, *Vaccination*, p. 55.

even the chemical value, as we may call it, of the antidote is by no means uniform. We must not expect mathematical precision in what is fundamentally a vital process. The child after vaccination grows rapidly, and the amount of antidote which secured him complete protection at first may be insufficient when his body-weight has changed from ten or twelve pounds to four stone or more. Presently a time comes when he can again take the disease, though he is so far protected that it is not severe enough even to disfigure, much less to kill him. Later on he is still less protected, and presently he has practically used up the influence of his infant vaccination. The store of the antidote should be renewed by revaccination. It is impossible to say exactly when these stages from safety to danger are reached. The strength of the assailing poison varies no less than the strength of its antidote. Smallpox has always varied much in its degree of severity. Even in unvaccinated people it is sometimes quite a mild disease. In the Leicester epidemic 1892-93 the type of the disease for the most part was very mild. Of 357 cases 261 were quite mild; among them there was no death; there were 89 confluent cases, with 14 deaths; and only 7 malignant cases, with 7 deaths.

Jenner was mistaken when he said vaccination would be as lasting a protection against smallpox as a previous attack; and much of the discredit which has fallen upon vaccination is due to this error on his part. It is unfortunate that he did not realise that he was not in a position to know how long the protective influence of vaccination would last. It needed only twenty years to show that after even efficient vaccination a slow progress from safety towards danger is inevitable, and that revaccination at least once after childhood is necessary if protection is to be maintained. In spite, however, of the almost universal oblivion of this fact, the effect of vaccination during the past century has been very striking. If we ask for exact figures, each country must be studied separately; we must know what the law is as to vaccination, how the law is administered, and how far it is actually enforced or evaded. The conditions vary in each country.

England. — The first vaccination law in England was passed in 1840. It recommended vaccination, but left the parent free to adopt or to decline it. In 1853 vaccination was nominally made compulsory, and penalties were imposed for non-compliance with the law. Practically, however, these provisions were found to be very imperfect, and the obligation to be vaccinated was little more than nominal till paid vaccination officers were appointed. In 1867 power was given to Unions to appoint such officers, but in many Unions the power was not exercised. In 1870 nearly half of the Unions were reported not to have appointed vaccination officers. In 1871 the Act of 1867 was amended, by making the appointment of paid vaccination officers compulsory in all Unions, and by improving the methods of registering vaccinations. In 1898 "the tremendous experiment" (as Lord Lister called it in the debate in the House of Lords) was made of practically leaving infant vaccination to the discretion of the father.

The whole effect of the Act of 1898 will be considered later. Omitting it for the moment, it may be said that the number of deaths from smallpox in England steadily declined with each of the improvements in the law enforcing vaccination. Instead of 2000 to 4000 per million as in the eighteenth century we had, during optional vaccination (1840-53), an average death-rate of 417 per million. With enjoined vaccination from 1854 to 1871, there was a rate of 154 per million; and with enforced vaccination the rate was again reduced to 99. During the ten years 1889 to 1898 the mortality rate per million averaged 10 only. In London, Dr. Farr's estimate of the annual smallpox mortality at the end of the eighteenth century was 5020 per million; under enforced vaccination this has been reduced to an average of 178 per million for the eighteen years ending 1890.

In considering these figures we must bear in mind that though a compulsory law does much, it fails to compel in a minority of cases, and that when we speak of compulsion it is likely that in every community there will be a considerable number of people whose parents have succeeded in evading the law as to infant vaccination. Moreover, in England there is no law enforcing revaccination, and the large majority of adults have not been revaccinated, so that in the presence of an epidemic many of those vaccinated in infancy prove to be again liable to take the infection. The number of persons in this position will vary with the amount of public feeling for or against vaccination, and with the care taken to devise methods by which indirect pressure shall be brought to bear in favour of vaccination. It is also important to remember that even the low rate of mortality in England in recent years represents a considerable annual aggregate of deaths, and about seven times as many cases of illness; a death-rate of only 10 per million sounds low, but it comes to over 300 deaths a year, and it implies about 2000 cases of illness which might be prevented. It will be seen in studying the facts as to vaccination in the German Empire how far England is from the highest measure of success in the prevention of smallpox.

In tabular form we have:—

For all England.	Deaths.
18th century rate, a minimum of	2000 per million.
1801, Vaccination introduced and largely adopted . .	No precise figures.

For all England.	Deaths.
1840 to 1853, Optional vaccination, average	417 per million.
1854, Enjoined vaccination, average	154 ,, ,,
1871, Compulsory vaccination, average	99 ,, ,,
1889 to 1899, Better administration of the Vaccination Act, aided by isolation	10 ,, ,,

For London alone we have Dr. Farr's estimate of the smallpox mortality at the end of the eighteenth century as 5020 per million. Under enforced vaccination, in spite of many evasions of the law, and of the great epidemic of 1871-72, the average mortality of the eighteen years ending in 1890 was 178 per million. The gradual steps of the decline are thus given by Dr. Farr :—

1838-53, Average mortality	514 per million.
1854-71, ,, ,,	388 ,, ,,
1872-90, ,, ,,	178 ,, ,,

In Scotland the rate of mortality from smallpox in the last eighteen years of the eighteenth century is believed to have been at least 4700 per million of population. From 1853 to 1864 vaccination was optional, the mortality rate was 340 per million. From 1865 to 1889 it was compulsory, and the rate came down to 80 per million. For the five years 1893-97 it has averaged 12. In Ireland during the same five years the rate has averaged 10 per million :—

18th century rate	4700 per million.
1853-64, Vaccination optional	340 ,, ,,
1865-89, ,, compulsory	80 ,, ,,
1893-97, Better administration of Vaccination Act	12 ,, ,,
Ireland, 1893-97	10 ,, ,,

In France there was, up to February 1902, no compulsory vaccination law, but vaccination was indirectly encouraged by the Government. No official post could be taken without producing a certificate of vaccination. Vaccination has been enforced for the French army since the year 1888. No statistics exist as to the total mortality from smallpox in the whole of France. The smallpox mortality of the large towns is known, and in these, with a population in 1892 of 8,000,000, 5670 lives were lost in the five years 1889-98, while in the same years Germany lost only 572 out of a population of 50,000,000. There have been no means of ascertaining the mortality from smallpox in the rural districts of France. The women and children and all defective male persons excused from service in the army have not been protected by any legal enactments in support of vaccination. Smallpox has been continually present in France, especially in the southern Departments. It is likely that the loss of life from smallpox among women and children has been an important factor in the absence of growth of population in France. In February 1902, however, France adopted compulsory vaccination in infancy, and revaccination in the eleventh and twenty-first years of life. If this law is properly administered, the condition of France with regard to smallpox ought speedily to be much improved.

In Sweden, where registration of deaths dates from 1774, instead of as in England from 1837, the average for twenty-seven years before 1800 was 2008 deaths per million. With permissive vaccination the fifteen years' average from 1801 to 1815 was 631. With compulsory vaccination from 1816 to 1885 the seventy years' average was 173 per million; with increased care in enforcing the law the rate during the four years 1893-96 was reduced to an average of 2·1.

In tabular form :—

18th century, Smallpox mortality	2008 per million.
1801-15, Permissive vaccination	631 ,, ,,
1816-85, Compulsory vaccination	173 ,, ,,
1893-96, Improvement of administration of vaccination law	2·1 ,, ,,

Denmark. — At Copenhagen the population and the smallpox deaths have been recorded year by year since 1750. In the years 1750 to 1800 the average smallpox mortality rate was 3567 per million. Vaccination was introduced in 1801, and it was extensively practised. It was permissive only, but the mortality rate fell to 660. The law for compulsory vaccination came into effect in 1810, and the average death-rate for the next sixty years was 130. For the years 1893-97 the average mortality from smallpox has been ·5 per million for the whole of Denmark.

In Prussia vaccination has been compulsory upon all recruits for the army since 1831. There was no law for the compulsory vaccination of infants till the year 1874. It was then enacted that every child should be vaccinated before the end of the year next after the year of its birth, so that in many cases it happened that the child was nearly two years old before it was vaccinated. Revaccination was at the same time made compulsory upon all children of school age; and on joining the army the recruits were again revaccinated. Therefore, to argue that Prussia or any other kingdom of Germany had compulsory vaccination generally enforced before 1874 is worse than stupid; it is a deliberate untruth. But the statement is still made from time to time in this country. From the date of the Act of 1874 smallpox has been extremely rare in the Prussian army. The smallpox mortality in the Prussian nation from 1865 to the end of 1874 averaged 755·4 per million; after the law of 1874 the average for the next ten years was 22·2. The great epidemic of 1871-72 no doubt brought up the mortality very considerably in the earlier decade, but even in non-epidemic years it was high, *e.g.* in 1865, 1866, 1867 it was respectively 438, 620, 432; while in 1877, 1878, 1879 it was reduced

to 3, 7, and 13 respectively. There was a slight increase in 1880, 1881, and 1882, when for two years the rate was 36, but how small is this compared with the rates of the previous decade, when twice only was it under 150. The exact figures are shown in the following table:—

Prussia—Before the Law of 1874.

Year.	Deaths per Million of Population.
1865	438
1866	620
1867	432
1868	188
1869	194
1870	175
1871	2432
1872	2624
1873	356

After the Law of 1874.

Year	Deaths per Million of Population.
1874	95
1875	36
1876	31
1877	3
1878	7
1879	13
1880	26
1881	36
1882	36
1883	20
1884	14

The next decade of Prussian statistics shows a still further diminution in the mortality rate. Their splendid vaccination system, with twenty-five State Institutions for supplying calf-lymph, was not brought to its present high standard at once after the law was passed; it had to be created and to be gradually developed. The ten years 1885 to 1894 show these rates:—

Prussia.

Year.	Mortality per Million of Population.
1885	14
1886	5
1887	5
1888	3
1889	5
1890	1·2
1891	1·2
1892	3
1893	4·4
1894	2·5

In the Prussian army there have been but two deaths from smallpox since the year 1875: one in 1884, one in 1898. The first was that of a reservist who had been twice unsuccessfully vaccinated in the army.[1]

Statistics for all Germany do not begin till 1886. Steady improvement is shown in the smallpox mortality rates per million, year by year, from 1886 to 1899, *i.e.* for fourteen years.

[1] V. r. *Smallpox in England and other Countries*, p. 35. E. J. Edwardes, M.D., etc.

Year.	Smallpox Mortality Rate per Million.	Year.	Smallpox Mortality Rate per Million.
1886	4·2	1893	3·1
1887	3·5	1894	1·7
1888	2·3	1895	0·5
1889	4·1	1896	0·2
1890	1·2	1897	0·1
1891	1	1898	0·3
1892	2·1	1899	0·5

In the entire German Empire the average rate of smallpox mortality in the ten years 1890 to 1899 was 1·07 per million, *i.e.* about one-tenth of the English rate in the same years, and this in a population surrounded with more or less unvaccinated neighbours.

Is it possible to doubt, after studying these figures, the value of a strict law both for vaccination and revaccination, and of ceaseless endeavour after perfection in the administration of the law?

Attempts are made by the opponents of vaccination to attribute the remarkable results of the law of 1874, and of the administrative improvements which have been gradually made in Prussia since that date, to stricter isolation, which they say began in 1883. No reasonable person doubts the value of the isolation of smallpox patients. Professor Koch spoke highly of it before the German Vaccination Commission of 1884, but the official Report of the Commission, issued in 1885, mentions no new law of isolation, nor does the volume on vaccination issued by the Imperial Health Office, Berlin, in 1896. It is, however, possible that some administrative improvements were made in favour of stricter isolation without any change in the law. Of this, however, no evidence can be quoted. When Germans are asked how isolation is carried out in their country in cases of smallpox, the answer is to the effect that they do not know, as there is no smallpox in Germany. This is not strictly correct, but it shows that in Germany epidemics are not so much arrested by isolation as prevented by efficient revaccination.

The Report of the German Commission on Vaccination gives the following table, showing the relative mortality from smallpox per 100,000 population over nine years, between Dresden with compulsory vaccination and revaccination, and Prague with no compulsory vaccination:—

Year.	Dresden.	Prague.
1875	2·5	11
1876	0·5	78
1877	0·9	395
1878	0·0	86
1879	1·8	84
1880	3 6	290
1881	2·6	64
1882	1·3	57
1883	0·8	224

In Austria vaccination is recommended, but it is not compulsory. Since 1891 sharp supervision has been exercised in its support, especially as to vaccination or revaccination at school age. The average mortality for the five years 1877 to 1881 was 619 per million; for the four years

1893 to 1897, with increased pressure in favour of vaccination, the average mortality was reduced to 99 per million, not good in itself, but much better than before. To reduce the mortality to the lowest possible, the vaccination law should be compulsory, the methods of its supervision carefully organised, and revaccination in early adult life made obligatory.

In Switzerland, some of the cantons have no compulsory infant vaccination. The towns of Basle and Zurich are in these cantons. Much indirect pressure in favour of vaccination is, however, given in the absence of a compulsory law, and in some districts from 60 to 78 per cent of the children are vaccinated. There is nowhere in Switzerland any compulsory revaccination. Grouping the cantons in which infant vaccination is the law into one group, and those in which it is not the law into another, the small pox death-rates *per million* living are as follows:—

Year.	Cantons with Compulsory Vaccination.	Cantons without Compulsory Vaccination.	Year.	Cantons with Compulsory Vaccination.	Cantons without Compulsory Vaccination.
1880	1·5	35	1887	0	12
1881	4·5	23	1888	0	15
1882	0	16	1889	0	13
1883	0	13	1890	0	29
1884	1·4	15	1891	0	15
1885	105·6	165	1892	8·9	89
1886	0	14			

In Italy there is now compulsory infant vaccination and revaccination at school age. The laws date from 1888 and 1891. The actual number of smallpox deaths in 1889, before the new law had taken effect, was 13,416; in 1898 it had fallen to 420. This is in a population of about 30 millions. The mortality rate per annum for the five years 1893 to 1897 is much higher, amounting, where averaged, to nearly 73 per million. The actual deaths from 1893 to 1898 were these:—

Year.	Actual Deaths.	Year.	Actual Deaths.
1893	2638	1896	2033
1894	2606	1897	1003
1895	2998	1898	420

The Italian figures supply a further commentary on the statement that success as to vaccination laws must depend to an enormous extent on the degree of perfection attained in their administration. The mortality in Italy even after the laws of 1888 and 1891 was very high; gradual improvements in administration have brought it down, under the same law, to 420.

In Hungary the vaccination law dates from 1887, in which year there was a smallpox mortality of 18,063. In 1898 this was reduced to 1656. For the five years 1893-97 the average rate per million was 134 = 2412 deaths each year. There is still evidently great room for administrative improvements in carrying out the law in Hungary.

In the Netherlands there is no compulsory infant vaccination, but vaccination certificates are required from all pupils and teachers in schools. A considerable proportion of the infant population is vaccinated every year, in spite of the absence of a compulsory law. The smallpox mortality rate per million in the five years 1893-97 was 38·7.

In Spain there is no law enforcing vaccination, and for the four years 1893-96 the mortality rate per million was 563·4.

In Russia there is also no law enforcing vaccination, and for the five years 1893-97 the mortality rate in a population (including Asiatic Russia) of 119 millions was 463·2; *i.e.* in each year the deaths from smallpox are believed to have been over 55,000.

The following tables show at a glance the varying rates of smallpox mortality per million of population under varying conditions as to vaccination and revaccination. The effect of legal pressure in support of vaccination is seen by taking the smallpox mortality in various countries and decades, and contrasting it in each case with the mortality in Sweden before the introduction of vaccination, which is accurately known:—

Before Vaccination, per Million of Population.	Vaccination not Obligatory by Law, but Encouraged.		Vaccination (nominally) Obligatory by Law.		Vaccination Obligatory under Penalties Charged only.	Revaccination of all School Children.	Thrice Vaccination.
Sweden, 1792-1801.	Prussia, 1860-69.	Austria, 1881-90.	Sweden, 1859-68.	England, 1857-66.	England, 1889-98.	Germany, 1890-99.	Prussian Army, 1875-99.
880	190	814	388	206	·8	1·2	
944	302	948	183	335	0·6	1·0	
1758	211	592	49	197	1·7	2·1	Two deaths in
2956	338	508	37	140	14·7	3·1	twenty-five years.
1963	463	601	76	66	49·3	1·7	One death refers
751	438	382	182	81	27·3	0·5	to a reservist
585	620	447	325	293	7·3	0·2	twice unsuccess-
1609	432	645	293	373	17·6	0·1	fully vaccinated.
5126	188	557	253	309	0·8	0·3	
2566	194	249	342	144	8·1	0·5	
Average in the 10 years 1914	337	574	213	214	13	1	

In studying this table it must be remembered that Germany is surrounded by populations (Russia, Holland, Austria, France) which have had till lately no compulsory general vaccination law, and that most of the cases in Germany occur on the frontiers.

Further evidence is obtained from the facts tabulated thus:—

Recent Death-rates from Smallpox per Million Living, for the Ten Years 1890-99, *in Four Countries.*

	Sweden.	Scotland.	Belgium.	Netherlands.
1890	0·2	0	105	not given
1891	0·2	0	211	2
1892	0·8	3	408	10
1893	5·0	17	336	41
1894	3·5	32	85	130
1895	0	11	46	16
1896	0	0·4	20	7
1897	0	2	21	not given.
1898	0 2	0·4	24	1
1899	0	0 2	38	not given.

Average Deaths from Smallpox in various Places in the same Five Years, 1893-97, *per Million of Population.*

	French Towns.	Germany.	Netherlands.	Austria.	Russia.
1893-97	90	1·1	39	99	463 [1]

Germany, Hungary, Italy, Roumania, Japan, and, quite recently, France, all now have compulsory revaccination. Time and patient effort to improve administration will be required everywhere to reach results equal to those seen in Germany.

In the survey now made of the effect of legal compulsion as regards vaccination and revaccination in European countries the evidence all points the same way, viz. that efficient vaccination and revaccination greatly diminish the number of deaths from smallpox, and that a compulsory law for vaccination and revaccination is necessary if a nation is to be free from a large smallpox mortality. It is, however, necessary to bear well in mind that it is not the existence of a law in favour of vaccination that protects, but the fact that the individual members of the community have obeyed the law. It is misleading to speak of places as being well vaccinated. The place is not the unit: every person in each place is protected or not according to his own vaccination and revaccination. Where there is a minority of people who are not protected, they are liable to take the contagion of smallpox *if exposed to it*, no matter how well protected their neighbours may be. They are, of course, much more liable to meet the contagion if they are surrounded by people who are able to receive and to communicate it, but this is all they gain by the protected condition of their neighbours. If and when they do meet the contagion they are pretty certain to take it.

One of the most important proofs of the value of vaccination has been the change of the age at which smallpox deaths mostly occur now as compared with the age at which they chiefly occurred in pre-Jennerian times.

It is certain that before the practice of vaccination, smallpox was the most dreaded and the most fatal of all the diseases of children. In the seventeenth and eighteenth centuries it was said by some authorities to account for half the total mortality of children under ten years of age. Among other proofs which might be quoted in support of this statement, the following may be given. In Chester in 1774 all the 202 deaths in an epidemic were those of children under ten, and 50 of the deaths were in children under one year of age. In Warrington in 1778 all the deaths were those of young children. In Kilmarnock, of 622 deaths which occurred in several epidemics, the ages of 9 are not given, but all the rest were under ten years of age. The burial registers of St Cuthbert's and Buccleuch Street, Edinburgh, show that during the years 1764-83, of every 1000 deaths from smallpox 993 were those of children under ten. The smallpox deaths at the Hague for fifteen years numbered 7473; of these, 6045 were of children under five.

In the Final Report the Commissioners say that "in all records of epidemics where the ages are given, the mortality was mainly among infants." In Berlin from 1758 to 1774 there were 6705 smallpox deaths; of these, 5876 were of children under five (= 87 per cent), and of children under ten there were 6618 (= 99 per cent). In Manchester, 1769-74, of 589 smallpox deaths, 559 (= 94 per cent) were of children under five. In 1796, in Berlin, of 817 deaths only one was over sixteen years of age.

The opponents of vaccination are fond of saying that smallpox was a trifling illness, "a mere disease of children." The records now quoted scarcely justify the "mere" or the "trifling." The truth is, it was a fearful scourge, and a terror to all parents.

The explanation of the very great mortality of young children as compared with adults is to be found in the rapidity with which epidemics of smallpox followed each other. In each epidemic those who died were probably about one-seventh of those attacked. The six-sevenths recovered and were immune for many years, often for life. They had purchased immunity

[1] The actual number of deaths from smallpox in the Russian empire, including Asiatic Russia, in the five years quoted was 275,502.

by suffering, possibly by loss of sight, almost always by permanent disfigurement. In isolated places and where epidemics came at long intervals many adults died, because there were in this case fewer adults who were protected by having had the disease in childhood. Since vaccination has been employed, and since it has been practised mainly in early infancy, the mortality rates, as between infancy and adult life, have completely changed. Children are now the protected class, and their protection is stronger the younger they are, because they are then nearer to their vaccination. It would be difficult to even suggest any possible cause other than vaccination for this striking change in the age incidence of the mortality from smallpox.

The Sheffield epidemic 1887-88 afforded a good illustration of the value of vaccination in early life. There were in Sheffield 33,393 vaccinated children under five years of age. Of these, 121 (= 0·36 per cent) took smallpox, and one child died from it (= 0·003 per cent of the total number). Of unvaccinated children under five there were 1981, of whom 128 (6·5 per cent) took the disease, and 66 died of it (= 3·3 per cent of the whole number of unvaccinated children, and 51 per cent of the children attacked). The table of the Sheffield mortality in age classes is instructive:—

rate," in the two classes, and at the varying ages.

The Sheffield figures are often quoted by the opponents of vaccination. They say Sheffield was a well-vaccinated place, and yet smallpox caused 474 deaths, of which 200 were of vaccinated people. When analysed, the figures show clearly how great the protection of vaccination is in early life, and how necessary revaccination is to renew the protection for later life.

The study of the proportion of deaths between the ages 0 to 10 at different places also gives evidence which is worth considering, vaccination being much more practised in some places than in others. Taking six places where recent epidemics have been carefully studied, we find that the percentage of mortality in cases under ten years of age varies very much, and that where vaccination has been to a great extent in abeyance the infant mortality from smallpox is the highest. The Commission gives the following table in their Final Report, p. 50:—

	Percentage of the Total Smallpox deaths borne by those between the age 0-10.
Warrington	22·5
Sheffield	25·6
London	36·8
Dewsbury	51·8
Gloucester	64·5
Leicester	66·6

Sheffield Epidemic—Age Classes

1	2	3	4	5	6	7	8
Ages.[1]	Population at that Age.	Cases.	Per Cent.	Deaths.	Per Cent.	Fatality, *i.e.*, Rate of Death to Attack.	Deaths per 100,000 in each Age Class.
			Vaccinated.				
Under 5	33,393	121	0·36	1	0·003	0·8	3
5 to 10	34,843	232	0·67	5	0·014	2	14
10 to 15	32,965	629	1·9	11	0·03	1·7	30
15 to 20	27,111	979	3·6	19	0·07	2	70
20 to 30	44,788	1267	2·8	69	0·15	5·4	150
Above 30	92,041	899	0·97	95	0·1	10·4	100
			Unvaccinated.				
Under 5	1,981	128	6·5	66	3·3	51·	3,300
5 to 10	278	100	36	34	12·2	34·	12,200
10 to 15	235	91	38·7	32	13·6	35·	13,600
15 to 20	282	84	29·8	53	18·8	63·	18,800
20 to 30	884	98	10·6	61	6·9	62·	6,900
Above 30	2,028	49	2·4	28	1·4	57·	1,400

[1] Excluding children under three months, the age at which they should be vaccinated.

The fourth column in this table shows the difference of liability in the two classes to take the infection at varying ages. The fifth column gives the actual number of deaths at each age. It shows how the protection of vaccination wears out progressively with age. The sixth column shows the percentage of deaths to the number of inhabitants; and the seventh the percentage of deaths to attacks, or the "fatality

In London there had been a considerable falling off in the amount of vaccination for some time before the epidemic of 1892. In Warrington and Sheffield the law had been fairly well obeyed. At Dewsbury vaccination had been greatly neglected. At Leicester vaccination had been largely abandoned for some time before the epidemic. At Gloucester vaccination fell into almost complete disuse for

some years before its epidemic. There is here no question as to whether children were vaccinated or not vaccinated. The total deaths of children under ten were three times as many where vaccination had been in abeyance as it was in places where the law was for the most part complied with.

The Commissioners conclude by saying:—"Apart from the difference in the extent of vaccination, no cause has been suggested at all adequate to account for the variations in the age incidence of fatal smallpox upon which we have been dwelling. . . . If improved sanitation were the cause of the diminished mortality of children in proportion to that borne by those of older years, it is quite impossible to understand how its effect should have varied so greatly in these different towns, and why in Gloucester and Leicester the mortality from the disease should have been so largely among children, approaching in that respect the experience of the epoch preceding vaccination." In the same six towns the fatality in the vaccinated was 2·8 per cent., while in unvaccinated children it was 30·3 per cent.

The incidence of smallpox mortality on different classes of the community also furnishes proof of the protective influence of vaccination. Doctors, nurses, hospital attendants, all show similar results. But for vaccination doctors would certainly be far more likely to contract smallpox than other people. As, however, they almost invariably protect themselves by vaccination and revaccination, their mortality rate is only as 1 to 5·6 in the whole community. The risk they run of taking contagious fevers, for which no protection is possible, is shown by the fact that their mortality from this cause is 3·68 times as high as that of the general public. It has always to be remembered that vaccination takes a certain time to develop its protective influence. The incubation period of smallpox is usually twelve days, occasionally more. The initial illness begins twelve or more days after the poison has been received into the system. Vaccination takes nine days to develop its influence. The attack of smallpox is not prevented unless the antidote created by the vaccine vesicles has had its necessary nine days before the fever of smallpox begins. Dr. Gayton reported that at the smallpox hospital in 1871-77 out of 366 people employed all but one were revaccinated. The one unvaccinated person took it, and at once. The same thing was repeated later. Dr. Marson, in thirty-six years, had no nurse or servant attacked. Probably some of the nurses and servants in smallpox hospitals have usually had the disease itself, and are protected by their previous illness, but this only refers to a small proportion of the whole staff, and it does not destroy the weight of evidence afforded in support of vaccination. In the recent epidemic (London, 1902) there were in the Mile End Infirmary forty-three nurses or attendants when small-pox was accidentally brought into the wards. Thirty-one of the number had been revaccinated at various dates up to two years before the outbreak. Four were revaccinated, two unsuccessfully. None of these thirty-five contracted smallpox. Of the remaining eight, one was away on sick leave, leaving seven, who all suffered from smallpox. Of these three were not revaccinated, and four all sickened with smallpox before their vaccination had had time to protect them, viz. two on the second, one on the third, and one on the sixth day after vaccination. None after nine days. Thus these four cases were incubating smallpox at the time they were vaccinated, and though they might be classed as vaccinated, they were not really under its influence. It is certain that none of the nurses who were revaccinated before exposure to infection contracted the disease, and that all who suffered were either unvaccinated or revaccinated after they had received the contagion. Dr. Hope in his Report upon the epidemic of smallpox in Liverpool (1902) says: "Nurses, doctors, laundrymaids, and servants at the smallpox hospital in Liverpool are rendered immune from smallpox by efficient vaccination. No one can doubt that if the general public were as efficiently vaccinated as the hospital staff they would be equally immune from smallpox."[1]

Experience of this kind could be quoted from every smallpox hospital. Where vaccination has been evaded, or unsuccessful, or postponed even for a few days, the infection is taken; not where it has been efficiently performed before exposure to infection.

Among the Post-Office staff in London, averaging, at that time, 10,504 persons, there was not a single death from smallpox in the ten years 1871 to 1880, though this included the great epidemic of 1871. During the ten years 1891 to 1900, the Post-Office service of the United Kingdom employed 692,852 persons. Among them, in the ten years there were two deaths from smallpox.[2] In the recent epidemic (1902) one Post-Office servant died of smallpox at Bury St. Edmunds. He had refused to be revaccinated on his appointment, and by some administrative defect he had been allowed to evade the regulation.

In the Navy revaccination has been the rule since 1864. From 1895 to 1900 inclusive there have been in all nine deaths from smallpox; six of these have occurred off China, one in India, and two in the irregular forces. Probably all were due to evasion of the regulations.

In the Army revaccination has been the rule

[1] Report of the Medical Officer of Health, Liverpool, 1902.

[2] Official letter from Postmaster-General to Dr. Edwardes.

since 1858. In 1899 the total Force was 201,004: (India, 67,697; Home, 99,832; Egypt, 3959). There were two smallpox deaths, one in India, and one in the non-European troops in West Africa. In India, Egypt, and West Africa there is always a large amount of smallpox in the native populations. But for vaccination and revaccination probably no European country could hold India, the loss of life in the Army from smallpox would be so enormous. In the Home Force for 21 years up to 1899 there was an annual death-rate from smallpox of ·076 per 10,000. In the colonies the rate was ·038 per 10,000, in India it was nearly ·7 per 10,000; in Egypt there had been twenty-five deaths in the British troops in the course of thirteen years.

The evidence in the support of vaccination being so overpoweringly strong the questions arise: How can any one refuse to accept it? What is the secret of the resistance to vaccination? The first point to notice, in trying to answer these questions, is that the number of people practically acquainted with smallpox who do not believe in the protective influence of vaccination is so small as to be in effect non-existent. No one has ever heard of an anti-vaccinist who had held a post at a smallpox hospital, or had had large experience of smallpox in any other way. The anti-vaccinist as a rule has only studied smallpox from afar, in the safe seclusion of the British Museum Reading Room or of his own study. In the second line of the party stands the impenetrable body of cranks, the people who cannot accept evidence, and who cannot distinguish between small evils and great ones. To them a sore arm for a few days seems as important an evil as smallpox itself. Behind the cranks there is the great mass of uneducated people who are against vaccination because it gives them a little trouble, some restless nights, and because they do not realise what smallpox really is. They have heard of or seen cases in which vaccination has done harm. It is easy to sympathise with this group of objectors. It must be admitted that a large part of the opposition to smallpox is based upon evil results which have sometimes followed the operation, and which have been due for the most part not to the compulsory law but to its faulty administration. Many of the deaths ascribed to vaccination have probably had nothing whatever to do with it; but on the other hand we have to recognise that infants of three months old are very sensitive creatures, and that as an undigested mass of casein or other food in the stomach may cause a convulsion in one child, an inflamed arm may possibly do the same in another, and that in a third both conditions may exist, and that they may combine to do the mischief.

Eruptions and Injuries after Vaccination.—Vaccination is occasionally followed by eruptions. Some of these follow a pure vaccine inoculation, and they may occur immediately after vaccination or soon after the development of the vesicles. Many of them are probably due to reflex nervous influences, others to secondary local inoculations, or to absorption of virus from the developed vesicles. Bearing in mind the powerful influence that vaccination exerts over the organism for a series of years, it is not remarkable that it should occasionally disturb the nutrition of the skin and other organs, and interfere for a time with the vital chemistry of the individual. On the other hand, many cases have been recorded in which the constitutional change started by vaccination has appeared to be beneficial, as for instance in the sudden disappearance of an extensive eczema after vaccination. Few of the eruptions due to a pure vaccine inoculation need be regarded with anxiety. They are rare, and for the most part trivial in character. Other eruptions are due to a mixed infection either at the time of vaccination or subsequent to it, but through the wounds. Much may be done to prevent eruptions of this class by guarding the vaccination wounds with care, and by using only lymph from which extraneous germs have been removed. Even as regards vesicles which may be called normal, considerable variety in the rate of healing and in the degree of the surrounding inflammation of the skin may be seen. This variety probably depends more on the vital condition of the patient than upon anything else.

Vaccination vesicles should be well and soundly healed before the end of the third week, but the power and rate of healing vary much in different individuals. Generalised vaccinia usually results from auto-inoculation. Dr. Acland has recorded a remarkable case which resulted in death. It may depend upon an abnormal absence of vaccinal immunity. "It is certain that during the first week after vaccination a person may be readily revaccinated or may contract smallpox, for it does not appear that any considerable degree of immunity is established till after the pocks have reached maturity. Immunity probably reaches its maximum about the fourth week after vaccination, but the standard of resistance varies in each individual, and probably also with the dose and activity of the virus."[1] In the fatal case above alluded to pocks continued to form for thirty days or more, showing the absence of immunity. The same condition may have been the explanation of the case in the Gloucester epidemic, in which a child developed fatal smallpox twelve days after being successfully vaccinated. The problems of infection and immunity are vital, not mathematical, and they manifest from time to time the variations proper to all vital processes. Another case illustrating the same truth has been re-

[1] *Vaccinia in Man*, J. D. Acland, M.D., p. 29.

corded by Dr. Clifford Allbutt, in which a woman under forty years of age had had small-pox in a mild form three times, and had also been three times successfully vaccinated. Vaccinia hæmorrhagica and vaccinia gangrenosa are two conditions which apparently depend upon constitutional defects in the individual rather than upon the presence of any pathogenic organism in the vaccine lymph. Eczema and other inflammations of the skin are extremely common in early infancy, and the slight irritation resulting from even normal vaccination may be sufficient in some children to produce the condition which underlies the skin affection. With regard to congenital syphilis it should be borne in mind that vaccination may be quite normal in a child who will presently show indubitable signs of inherited taint; it is possible, too, that in such a child the temporary disturbance of health due to vaccination may precipitate the manifestations of inherited disease. In such a case careful inquiry into the history of the symptoms, their order and chronology, will almost always show the real relationship between the two sets of facts. It is obviously of the greatest importance to distinguish between syphilis which has been communicated by vaccination and the results of vaccination in a syphilitic child. It should be remembered that, both in England and Scotland, inherited syphilis kills a large proportion of its victims before the age at which vaccination is enjoined; also that when vaccination was practically in abeyance in Leicester the number of deaths from infantile syphilis showed an increase of 69 per cent as compared with an increase of 24·7 per cent from the same cause over the whole of England and Wales, where vaccination was much more common. It would, of course, be absurd to suggest that vaccination prevents the development of inherited disease, but these figures show that such disease is as common and as fatal in the unvaccinated as in the vaccinated portions of the community. It is true that a very small number of cases have occurred in which the disease has been conveyed through vaccination. The fact that they were infinitely rare did not carry much weight in the minds of anxious mothers. The other fact, that syphilis thus inoculated into the skin of a previously healthy child would run a course entirely unlike the inherited disease, the lay public were not able to appreciate, and the tendency was to connect every manifestation of inherited syphilis with the vaccination which had perhaps preceded it by a week or two. It is easy to understand how ready the father or mother would be to throw the blame of such disease on the vaccination, and also how willing sympathetic and courteous neighbours would be to accept this explanation. It would be unreasonable to expect in parents or neighbours the knowledge as to the prevalence of inherited specific disease that is learnt in the out-patient department of every hospital where children are received. The truth is that vaccino-syphilis is and has ever been almost infinitely rare; that the inherited disease is extremely common; and that, as a rule, this form of disease is plainly to be recognised before the child arrives at the vaccination age. Similarly with regard to deaths from erysipelas, the infant mortality from this cause in Leicester during the years 1883-1887, when there was very little vaccination, showed an increase of 41·5 per cent as contrasted with a diminution of 16·7 per cent over the whole of England and Wales. Had vaccination really been the cause of many cases of erysipelas, Leicester would surely have been able to show a better result than this.

The same considerations apply to lupus and tubercular infection as supposed results of vaccination. The fact that lupus has in a few cases begun in a vaccination wound or scar does not prove that the virus was introduced at the time of vaccination. Lupus is not very uncommon, and it is often started in a trivial wound of the skin with no possible connection with vaccination. Unvaccinated Leicester was no more free from deaths due to tubercular disease of the brain and abdominal organs than was fairly well-vaccinated England. So also as to the supposed connection between vaccination and leprosy or cancer. In the case of leprosy, where the virus is located mainly in the skin, it may possibly be that it could be communicated through vaccination if this was carefully attempted, and if lymph were taken from vesicles raised on a portion of skin already infected with leprosy. Needless to say, this would in practice never be done, nor would lymph be taken from an individual who showed signs of leprosy in even very distant parts of the skin.

The really grave complications of vaccination arise in almost every instance from inflammatory or septic affections common to all wounds, unless most carefully guarded against. Serious injury from vaccination is almost unknown in well-cared-for infants. With cleanliness, suitable food, and care, any abnormal result from vaccination is exceptionally rare. Taking all classes together, the mortality resulting from vaccination accidents before the Act of 1898 was in England 1 in 14,000, in Scotland 1 in 38,000, and in Germany 1 in 100,000. Every vaccination ought to involve four wounds of the skin. Till within a very short time a large number of vaccinators left these wounds quite unprotected. The wonder is not that some of the wounds were poisoned, but that so few were. The Act of 1898 did good by insisting upon much more care being taken of vaccination wounds, and by enjoining regulations for keeping them aseptic. How far these regulations can be observed in the crowded and often dirty homes of the poor may be questioned; but it is

something to have urged the need of greater care in the direction of asepsis upon the vaccinators themselves, and through them upon the mothers. It may be hoped that the lesson will in time be taken to heart even more than has been done at present, and that a very large reduction in the number of vaccination accidents will be the result. What can be done in Germany is not impossible in England.

THE EFFECT OF THE VACCINATION ACT OF 1898.—The Act of 1898 did great service to vaccination by introducing the use of glycerinated calf-lymph instead of humanised lymph. As calves cannot take the syphilitic poison there is absolutely no possibility of its being conveyed from them, and this particular danger, rare as it was, no longer exists when calf-lymph is used. The risk of tuberculosis is not quite so completely removed. Calves can be tuberculous, though they are less prone to the disease in early life than they are later. But every calf used for the manufacture of calf-lymph is put to death and thoroughly examined before the lymph is distributed. In the rare cases in which any evidence of tubercular infection is found the lymph from that source is immediately destroyed. But even if this were not always done, by the thorough and intimate admixture of glycerine with the lymph, and by storing the mixture for a considerable time under conditions which prevent the access of light and air, Dr. Copeman believes that all foreign or extraneous organisms in the lymph, including the tubercle bacillus, are gradually destroyed, and the vaccine organism only left.

The Act of 1898 further abolished the vaccination stations, and ordered the public vaccinator to vaccinate the child at home and to inspect the result of the operation there, on the eighth day. It permitted the father to obtain a certificate of exemption for his child, on application to a magistrate, as a "conscientious objector" to vaccination.

How far has the Act of 1898 been on the whole a success or the reverse? The "conscientious objector" clause was passed in the interests of vaccination. The framers of the Act believed that the final result of allowing parents to ask for exemption from the duty of having their children vaccinated would be to diminish resistance of the law. They required the father to appear before a magistrate and to declare that he "conscientiously disapproved" of vaccination. This was intended to involve a certain amount of trouble. While permitting conscientious objectors to get leave to disregard the law, many important practical improvements were made in the administration of vaccination. The use of glycerinated calf-lymph, the abolition of vaccinating stations, and the much greater care to keep the wounds aseptic, all told in support of vaccination; and it seems probable that these influences are producing an effect. Certainly, in almost every union-county in England the number of children vaccinated in 1901 greatly exceeded the number in 1898. With but one exception—the North Riding of York—all the union-counties in the following table show increased numbers vaccinated in 1901; and in many cases, *e.g.* in Bedford, Chester, Derby, Essex, Leicester, Lancaster, Nottingham, Stafford, and Warwick, the increase is very considerable—

Union-Counties of England.	Certificates of Successful Vaccination.			
	1898.	1899.	1900.	1901.
Bedford	325	1,830	1,456	1,579
Berks	4,121	4,864	4,397	5,325
Buckingham	1,896	3,904	3,029	3,179
Cambridge	3,332	4,598	4,298	4,385
Chester	16,891	19,518	19,382	18,430
Cumberland	4,601	6,094	7,333	6,986
Derby	4,477	9,459	7,903	8,027
Devon	11,012	11,008 – 4	13,413	12,835
Dorset	2,826	3,845	3,932	3,933
Durham	26,720	29,270	29,902	32,146
Essex	11,834	18,699	20,775	22,877
Gloucester	5,477	10,816	10,206	10,162
Hertford	2,950	4,333	4,601	4,770
Huntingdon	925	1,383	979	1,037
Kent	14,174	18,961	19,840	20,349
Lancaster	74,951	101,768	103,402	97,556
Leicester	769	3,150	3,478	3,265
Lincoln	5,780	9,253	9,939	9,925
London	63,326	86,514	86,591	111,958
Middlesex	9,283	15,003	15,953	20,624
Monmouth	6,092	7,595	7,661	8,144
Norfolk	5,963	8,277	9,161	8,934
Northampton	1,718	3,755	3,510	3,454
Northumberland	10,538	12,598	14,088	14,559
Nottingham	6,384	11,289	11,927	12,252
Oxford	2,907	4,303	4,055	3,479
Salop	4,826	6,089	6,006	6,100
Somerset	5,780	9,127	7,953	7,944
Southampton	12,940	15,204	16,313	16,416
Stafford	19,184	31,857	29,232	30,169
Suffolk	5,830	7,166	7,562	7,566
Surrey	9,233	11,395	12,246	16,836
Sussex	7,388	9,584	10,018	10,600
Warwick	15,111	20,257	19,926	20,181
Westmoreland	1,233	1,440	1,348	1,276
Wilts	2,586	4,379	4,143	4,132
Worcester	8,757	10,481	11,053	11,527
York—East Riding	8,792	13,672	10,734	10,791
West Riding	46,390	60,703	58,816	58,027
North Riding	8,443	8,249 – 194	8,448	8,123

Comparing the number of successful vaccinations in 1898 and in 1901, a considerable increase is seen, and the increase is maintained year by year. In 1898 the successful primary vaccinations were 500,314; in 1901, they were 710,785.

That a considerable epidemic of smallpox has been present in London and in some parts of England and Scotland for the last two years does not prove that the Act of 1898 was a failure. Vaccination had been greatly neglected for many years, and no legislation could possibly do away with the inevitable results of this

neglect. All that can fairly be asked is whether the last Act is helping the cause of vaccination or the reverse, and if by any further change it could be made to help it more? On the whole, we think the evidence is that the Act of 1898 is doing good, but that it might do much more if certain amendments were incorporated into it.

During the year 1898, between the date of the passing of the Act and the 31st December, 230,147 children were exempted, by their parents appearing as "conscientious objectors." In 1899 this number was increased by 32,357. In 1898 there was a large number of children who belonged properly to earlier years; their parents had ignored or resisted the law, and when exemption was permitted they claimed it. If the exemptions in 1899 prove to be about the average, it will mean that something like 3·48 per cent of all children born get exemption through the direct action of their parents. But in the year 1899 it was not 3·48 per cent, but 27·9 per cent of the children born in the year that were not vaccinated, so that after deducting the conscientious objectors, there remained in addition nearly one-fourth of all children born who, from one reason or another, were not vaccinated, and whose patients did not claim exemption. Some of these children were perhaps vaccinated later, and many probably died before the legal age for vaccination. Still, the percentage who escaped by the conscience clause was small compared with the number who escaped through defects in the administration of the Act. On the other hand, the percentage of successful primary vaccinations at all ages in London, which in 1898 was only 47·7 per cent of the number of births, became in 1901 as high as 85·3 per cent, a result due mainly to the educating effect of the presence of smallpox. It is far more important to devise means of improving the administrative machine so that 20 or 25 per cent of all children born may not evade the law, than it is to concentrate attention on the comparatively small number of conscientious objectors. In large towns a good many children probably escape registration, and they would be likely to be unnoticed by the vaccination officers, and would not even be included in the list of defaulters.

Alternatives to Vaccination.—The opponents of vaccination have tried to formulate an alternative policy, and they have done so in the two words "sanitation" and "isolation." But as to the value of these watchwords they are not unanimous. Dr. Creighton frankly admits that there is no evidence in support of the view that smallpox can be evaded or modified by sanitation. Others in his party throw "isolation" over, and speak of smallpox hospitals as "the culminating mistake in the social treatment of the disease." So that at this moment the majority of anti-vaccinators are opposed to one of their leaders about sanitation, and to another about isolation.

The truth about sanitation is that there is not a scrap of real evidence that smallpox is even considerably influenced by defective sanitary conditions, except that, if a house is very full of people, more will be exposed to the contagion than would be the case in a sparsely populated house. The disease itself is not affected, but more people are brought into contact with its infection. There is not a shred of evidence that it is in any sense a "filth disease" as to its origin. It is no more accurate to call it a "filth disease" than it would be to say so of whooping-cough. Whether insanitary conditions of any kind can increase the malignity of the type of smallpox once it has been imported, it is impossible to say. But even if this could be proved to be true of smallpox, as it is probably true of scarlet fever and influenza, it would still be incorrect to say that the disease was due to the conditions which increased its virulence. If smallpox were bred of filth, it would be chronically present in half the villages and farmhouses in England, which it certainly is not. The other safeguard proposed in the place of vaccination is "isolation," or the separation of the sick from the healthy. Isolation aided by vaccination is no doubt of great value, but it could not be employed in an unvaccinated community. Smallpox patients cannot wait upon themselves; and if the people round them in the hospital were not protected by vaccination and revaccination, the disease would spread in the hospital exactly as it spreads outside. The whole value of taking patients out of their homes lies in the power of surrounding them with attendants who are protected by vaccination, and thus stopping the infection from spreading. There is very little treatment possible in bad cases of smallpox, and for the patient's own sake the long journey to the hospital-ship or shelter would be much better avoided. The one advantage is that at the ship it is possible to prevent contact with unvaccinated people. Another point to be considered in connection with any large scheme of isolation is its cost. London is rich, and it can afford to do everything well, but the cost of transporting 70 or 80 patients a day to the ships, and of keeping 1400 or 1500 there, with the necessary service for the living, the dying, and the dead, must be enormous. The recent epidemic in London (March 1902) was estimated as having cost £10,000 per week. Revaccination would have prevented all this waste, and also an untold amount of suffering.

Amendments wanted in the Vaccination Act of 1898.—As Dr. Bond has well said, "What has been chiefly wanted to make the vaccination mills grind is motive power. That power can only come from two sources—the

central or the local authorities."[1] The problem is how to get the greatest amount of motive power from each of the two, with the smallest amount of waste through friction. Perhaps it would conduce to success to leave to the central authority the duty of dealing with all the determined opponents of vaccination, of deciding when to prosecute or not, and to limit the work of the local authority to the duty of making the provisions of the Vaccination Act known to the large part of the population who are prepared to obey the law when it is authoritatively presented to them. What is aimed at is not the punishment, or even the better education of the determined enemies of vaccination, but the prevention of epidemic smallpox. The question to be asked is, Can the vaccination law and its administration be so far improved that epidemics of smallpox shall not occur in the United Kingdom in spite of frequent reintroduction of the contagion from other countries? It is absolutely certain that such reintroduction of contagion will frequently occur. In Liverpool, for instance, from 6th December 1901 to 19th February 1902, 30 cases of smallpox were imported by sea or land. In the first ten months of 1901, 8 separate cases of smallpox reached Liverpool by sea, and 1 by land. Glasgow is in a similar position. Importation of the disease is sure to be very frequent in London. How can the population at the ports and in large centres such as London be made as immune to infection as the German population is in the frontier districts adjacent to badly vaccinated countries, *e.g.* Russia and, up to the present time, France?

Unless the presence of contagion can be avoided, the only way to prevent an epidemic is to have the population that is exposed to the contagion immune at the time of exposure. It is not enough to try to make all exposed persons immune some days after the risk of infection has been run. If revaccination were the rule here, as it is in the German Empire, we could face the continual risk of the reintroduction of smallpox from other countries without any fear of epidemics. Can the vaccination law or its administration be still further improved towards this end?

The Act of 1898 could, we think, be materially improved in four ways.

1. The administration of the Vaccination Act should be put into the hands of the local authority responsible for Public Health. At present it is worked by the Poor Law Guardians, who are not rarely elected expressly to defy the Act. They have been able till lately to appoint vaccination officers pledged to their employers not to carry out the Act. Liskeard, for instance, appointed as vaccination officer a man who had refused to have his own children vaccinated. The Local Government Board, however, did not sanction the appointment. At Leicester the Guardians declined to have a vaccination officer at all, but it also had in the end to submit to the Board. It is a misfortune that the administration of the vaccination law is in the hands of the Guardians. It belongs properly not to poor relief but to public health. At present the vaccination officer is selected by the Guardians; but his duty is to carry out the law to the satisfaction, not of his immediate employers, but of the Local Government Board. Even when the Guardians wish him to do as little as possible, it is his duty to put pressure upon defaulting or negligent parents to obey the law and to have their children vaccinated. He may do that which his employers prefer, or he may prosecute defaulters and obtain legal assistance and send in the costs to the Guardians. It is not likely that, under such an arrangement, many vaccination officers will be as active as they might be. If the administration of the Vaccination Act were put under the authority responsible for Public Health the difficulty in the way of administering the Act would be much diminished. The control of smallpox (apart from vaccination) belongs now to the sanitary authority. It has to meet all the difficulties which an epidemic of smallpox entails; no other body has the same direct interest in preventing an epidemic, and yet the one thing which can prevent it, viz., vaccination, is worked by another body, the members of which are sometimes pledged to do all they can to stultify the law. Even in a community such as Liverpool, where the Guardians do their utmost in support of vaccination, and where, in a population of nearly 700,000 people, only 26 certificates were given in 1900 to conscientious objectors, the present system works badly. The medical officer of health, Dr. Hope, shows in his report for 1901, that delay often occurs precisely at the moment when time is of the utmost value, by the health officer being unable himself to make arrangements for the immediate revaccination of "contacts" in the presence of a case of smallpox. He has to communicate with the officers of another body, and much precious time is lost.

2. Revaccination at school age should be made obligatory, subject to the conscientious objector clause as in primary vaccination. It is to systematic revaccination at about twelve years of age that Germany owes her freedom from epidemic smallpox, and there can be no security against the frequent recurrence of such epidemics in Great Britain and Ireland till revaccination is made the rule by legal enactment.

3. An adequate supply of trustworthy lymph should be provided from Government laboratories, and private practitioners should be able to buy it. The provision of enough lymph would be greatly aided by systematic revaccination at school age. A definite amount of lymph would be wanted month by month, very little

[1] *National Review*, March 1902,—"Smallpox." Dr. Francis T. Bond, M.D. (Lond.).

panic revaccination would take place, and there would be no waste by providing much more lymph for possible emergencies than could, in non-epidemic years, be used while it was still active. In the German Empire twenty-four State Institutions for the manufacture of trustworthy glycerinated calf-lymph are kept at work to supply the lymph for about fifty-three millions of people. The United Kingdom, with about forty millions of inhabitants, has but one Government Laboratory, from which no private medical practitioner can obtain lymph.

4. "Efficient vaccination" should be legally defined, and every certificate of vaccination should show if the vaccination had been efficient or the reverse, and its date.

The cost of isolating smallpox patients should be borne as far as possible by the head of the family in which each case occurs. "Objectors" might be required to show that they had made, through benefit or insurance societies, arrangements to meet this liability before exemption certificates were given them. As at present arranged the enormous cost of a smallpox epidemic is not felt by the people who cause it, but by the overburdened rate- and tax-payers generally.

In addition to the improvements now suggested in the vaccination law, indirect social pressure should be applied in every direction in support of obedience to the law. All who realise the amount of suffering, waste of valuable lives, and loss of money and energy in fighting smallpox epidemics ought to do all in their power to assist the vaccination laws by informal and mild compulsion applied in their support. The certificate of efficient revaccination should be asked for as a preliminary for every kind of employment. Domestic and outdoor servants, shop assistants, clerks in offices and banks, school teachers, parish workers, servants in hospitals, tenants of flats and chambers, the employés of dressmakers, tailors, bootmakers, builders, and carpenters, men and women in factories, stokers, sailors, and officers in steamers, would-be life-insurers, proposers for election into social clubs, should all be required to show their precious Certificate of Revaccination. No one ever hears of a man refusing a good post in the Police, or Post Office, because he will not be revaccinated; and if the certificate were continually in demand as a step to employment, it would be obtained and valued. It is for the medical profession to aim at making vaccination efficient, and a perfectly safe and painless operation. Till these results are achieved there will continue to be opposition and prejudice in the minds of the ignorant. The Act of 1898 did great good by enforcing more care in the performance of vaccination. Every medical practitioner should realise the evil effect of "vaccination accidents," even when not fatal, in the formation of public opinion against vaccination. It is only by the creation of a sound opinion as to the immense value of vaccination that legal enactments in its support can be so administered as to overcome the natural inertia of the less instructed classes of the community.

The summary of all available evidence as to the value of vaccination cannot be better stated than in the words of the Royal Commissioners in their final report, dated August 1896. They say: "The conclusions as to the value of vaccination arrived at by the Commission are as follows:—We think—

1. That it diminishes the liability to be attacked by the disease.

2. That it modifies the character of the disease, and renders it (*a*) less fatal, or (*b*) of a milder and less severe type.

3. That the protection it affords against attacks of the disease is greatest during the years immediately succeeding the operation of vaccination. It is impossible to fix with precision the length of this period of highest protection. Though not in all cases the same, if the period is to be fixed it might, we think, fairly be said to cover in general a period of nine or ten years.

4. That after the lapse of the highest protective potency, the efficacy of vaccination to protect against attack rapidly diminishes, but that it is still considerable in the next quinquennium, and possibly never altogether ceases.

5. That its power to modify the character of the disease is also greatest in the period in which its power to protect from attack is greatest, but that its power thus to modify the disease does not diminish as rapidly as its protective influence against attacks, and its efficacy during the later periods of life to modify the disease is still very considerable.

6. That revaccination restores the protection which lapse of time has diminished, but the evidence shows that this protection again diminishes, and that to insure the highest protection which vaccination can give the operation should be at intervals repeated.

7. That the beneficial effects of vaccination are most experienced by those in whose case it has been most thorough. We think it may fairly be concluded that where the vaccine matter is inserted in three or four places it is more effectual than when introduced into one or two places only, and that if the vaccination marks are of an area of half a square inch, they indicate a better protection than if their area be at all considerably below this."

Vaccine.—The virus of cowpox used in the performance of vaccination for the prevention of smallpox; it may be got from the calf (calf-lymph) or from the human subject (humanised lymph); also the virus of any specific disease which is introduced into the body by inoculation. *See* VACCINATION.

Vaccinia.—Cowpox. *See* VACCINATION; CONJUNCTIVA, DISEASES OF (*Exanthematous Conjunctivitis*).

Vaccinophobia.—Morbid fear of vaccination.

Vaccinosyphilis. — Syphilis conveyed by the use of impure vaccine virus. *See* VACCINATION.

Vacuolation.—The formation of small spaces in a cell or tissue. *See* MUSCLES, DISEASES OF (*Forms of Muscular Degeneration, Vacuolation*).

Vagabond's Disease. — Pigmentation of the skin due to long-continued irritation from pediculi aud lack of cleanliness. *See* ADRENAL GLANDS, ADDISON'S DISEASE (*Diagnosis*); SKIN, PIGMENTARY AFFECTIONS OF (*Vagabond's Disease or Phtheiriasis*).

Vagina, Disorders of the.

ANATOMY (*see* "GENERATIVE ORGANS," vol. iii. p. 440).
DEVELOPMENT (*see* vol. iii. p. 448).
MALFORMATIONS (*see* p. 447 *et seq.*).
CHANGES IN POSITION (*see* "PELVIO-PERINEUM AND PELVIC FLOOR," vol. vii. p. 379).
VAGINAL SECRETION 401
INFLAMMATORY AFFECTIONS:—
1. CATARRHAL VAGINITIS . . . 403
Granular Vaginitis . . 403
Warty Vaginitis 403
Adhesive Vaginitis . . . 403
Gonorrhœal Vaginitis . . . 404
Mycotic Vaginitis 404
Emphysematous Vaginitis . . 404
Exfoliative Vaginitis . . . 404
Vulvo-vaginitis of Children . . 404
2. PSEUDO-MEMBRANOUS VAGINITIS . 405
3. ULCERATIVE VAGINITIS . . . 405
4. SPECIFIC INFLAMMATIONS AND ULCERS OF THE VAGINA . . . 406
5. PARA-VAGINITIS 407
VAGINISMUS 407
NEW FORMATIONS 408
Cysts 408
Myoma and Fibroma 408
Sarcoma 408
Carcinoma 408
INJURIES OF THE VAGINA . . . 410
Wounds 410
Hœmatoma 410
FOREIGN BODIES 410
ACQUIRED STENOSIS AND ATRESIA . . 411
FISTULÆ 411
Urinary Fistulæ 411
Fœcal Fistulæ 413

See also ABORTION (*Symptoms, Treatment*); ASEPTIC TREATMENT OF WOUNDS (*Before Vaginal Operations*); FALLOPIAN TUBES (*Vaginal Hysterectomy for Diseases of*); GENERATION, FEMALE ORGANS OF (*Vagina*); GENERATION, FEMALE ORGANS OF, ARRESTED DEVELOPMENT (*Vagina*); GONORRHŒAL INFECTION; HYSTERIA (*Genital Organs, Vaginismus*); LABOUR, PHYSIOLOGY OF (*Factors, Passages*); LABOUR, STAGES AND DURATION (*Second Stage*); LABOUR, DIAGNOSIS AND MECHANISM; LABOUR, MANAGEMENT OF; LABOUR, PRECIPITATE AND PROLONGED; LABOUR, ACCIDENTAL COMPLICATIONS; LABOUR, INJURIES TO GENERATIVE ORGANS; LABOUR, OBSTETRIC OPERATIONS; MENSTRUATION AND ITS DISORDERS (*Menorrhagia*); PELVIS, PERINEUM AND PELVIC FLOOR; PERITONEUM, ACUTE PERITONITIS (*Examination*); PREGNANCY, PHYSIOLOGY OF (*Vaginal Changes*); PREGNANCY, MANAGEMENT OF; PREGNANCY, AFFECTIONS OF GENERATIVE ORGANS (*Malformations of Vagina*); PREGNANCY, HÆMORRHAGE; PUERPERIUM, PHYSIOLOGY OF; PUERPERIUM, PATHOLOGY (*Puerperal Infection*); STERILITY; SYPHILIS (*Acquired, Primary*); UTERUS, MALFORMATIONS OF; VENEREAL DISEASE.

VAGINAL SECRETION.—Among the functions of the vagina that of excreting the products of the internal genital organs is one of the most important. In the interior of the canal are thus found constantly, in greater or less quantity, menstrual blood or various other discharges from the body and cervix of the uterus. The vagina itself is lined by a stratified squamous epithelium, and its mucous membrane is normally unprovided with glands. Doubts have, therefore, arisen as to whether the vagina itself secretes, or whether the materials found in its lumen do not all come from above. It is observed, however, that after complete removal of the uterus the normal vagina continues to have a moist surface; and that in those severe cases of prolapse, where only the lower half inch or so of the posterior wall remains *in situ*, the exposed part of the vagina becomes dry and horny, while the part of the lumen which is preserved remains moist as usual. Hence it appears that the vagina does actually secrete, though under normal circumstances the amount of the secretion is small.

The normal secretion is a more or less viscid, tenacious fluid like thick starch mucilage. It has an acid reaction and chemically contains albumen but no mucin. The opaque whiteness of the secretion is due to desquamation of the superficial layers of the epithelium, and to the presence of a small number of leucocytes. The fluid part of the secretion probably consists of serum, which has transuded from the vessels of the papillæ in the sub-epithelial layer of the vaginal wall. The acid reaction is probably caused by the presence of fatty acids; these are due to the growth of the bacillus vaginæ, which appears to set up decomposition in the secretion immediately after it reaches the vaginal lumen.

Another important function of the vagina is to protect the internal generative organs against the entry of germs; a function the performance of which is rendered difficult because the means that prevent the entry of living germs must not interfere with the life of spermatozoa. In secretion taken from the vagina cocci and bacilli of many different varieties are found, and among them chain and cluster cocci, which are believed by one set of observers to be pyogenic organisms in a state of diminished virulence, and by another to be simple saprophytes. All these are derived from the vulva, and it is certain that along with other varieties septic and pathogenic germs must from time to time enter. It has been shown, however, by experiment that the vagina can actually free itself of a living culture of certain organisms in the course of from a few hours to two or three days. The healthy vagina thus appears to have some means of ridding itself of living disease germs that may gain an entrance, but as regards the nature of this bactericidal influence little is known. The acid reaction, the antagonism of different varieties of germs, phagocytosis, mechanical removal, and the absence of oxygen, have all been considered, but none of them offers a satisfactory explanation of what appears to be a certain fact, namely, that the vaginal secretion is a strong antiseptic and disinfectant. In pregnancy the bactericidal power of the secretion appears to be even more marked than in the non-pregnant condition.

While thus the healthy and intact mucous membrane is able to deal successfully with the injurious germs which find an entrance, under certain circumstances, as after injuries, labour or abortion, septic or pathogenic organisms are able to establish themselves in the vagina. As a result, diseased conditions may be set up, either in the pelvic organs and tissues or in the more remote parts of the body. Locally, the germs luxuriate in the natural lumen of the genital canal, causing local inflammations and general septic intoxication; or they may invade the tissues themselves and spread along the tissue spaces, the lymphatics, or the blood-vessels, in which case local inflammations also result, along with true septicæmia or pyæmia. The micro-organisms that are of most frequent importance in these connections are, in addition to many varieties of putrefactive germs, the pyogenic organisms, including the bacterium coli commune, and the gonococcus.

This fact that disease germs are able to enter through the vagina and to set up serious disease affecting the whole body or remote parts of it, as in septicæmia, pyæmia, and allied conditions, may prove to be of even greater importance than has hitherto appeared. One of the main advances recently made in general pathology has consisted in the discovery of the large part that infection by micro-organisms plays in the production of disease. Thus peritonitis is now understood always to be produced by the action of germs, and the so-called idiopathic form of the disease has almost disappeared even from the text-books. Endocarditis is due to infection with germs of divers kinds, among which the gonococcus is of the greatest interest in the present connection. In the joints again, as Howard Marsh (*Brit. Med. Journ.* vol. ii. 1902, p. 1831) remarks, inflammation, while never idiopathic, is frequently infective. Acute rheumatism is also most probably due to invasion of the body by germs, and it is possible that the great class of chronic rheumatic and rheumatoid affections may also prove to be due to bacterial infection, as is suggested by Dr. Chalmers Watson (vol. viii. p. 577). Various affections of the internal organs have thus been proved, or are likely to be proved, to be caused by germs, and many of them, such as arthritis and pericarditis, may be due to the action of any one of a large number of different germs. Arthritis, for instance, may be set up not only by the ordinary septic and pyogenic organisms, by the gonococcus or the tubercle bacillus, but is also not rarely due to the pneumococcus and to the germs of typhoid fever, influenza, scarlet fever, dysentery, or erysipelas. Where the dependence of a disease on a particular germ has been proved, it is not always obvious by what road the germ gained an entrance to the body; such entrance must take place through one of the natural mucous-lined orifices of the body or through a breach of the surface. That the vagina is frequently the portal by which admission is gained is abundantly clear in certain diseases, such as septicæmia, pyæmia, gonorrhœa becoming generalised, and in some cases of tubercular infection. It may well be proved in the future that the vagina serves also as a frequent port of entry for germs that give rise to certain other affections, such as rheumatism and pernicious anæmia, whose etiology is at present obscure. Thus leucorrhœa in many of its forms may acquire a greater significance, and may call for more serious consideration than is frequently given to it now.

Inflammatory Affections

Vaginitis. — Inflammation may affect any part of the vaginal wall from the hymen to the cervical attachment, and may be more or less limited to the mucous membrane, the muscular wall, or the connective tissue sheath. Nothing is known with respect to inflammation of the muscular coat apart from that of the other layers of the wall. Inflammation of the connective tissue surrounding the vagina is known as peri-, or better para-vaginitis, and will be referred to later on. Inflammation of the mucous membrane lining the vagina is by far the most frequent form, and to it the use

of the terms vaginitis or colpitis is confined. The covering of the vaginal cervix has a similar structure to the mucous membrane of the vagina, and is affected by inflammation in similar ways; hence inflammatory affections of this part of the cervix are frequently referred to as vaginitis of the portio, and should be considered along with those of the vagina.

The lining of the vagina has rather the characters of skin than of mucous membrane; it is covered by a thick stratified squamous epithelium, and has no glands, and is thus little prone to become inflamed. On the other hand, the lining is very vascular, is exposed to many injurious influences, and the changes that take place in menstruation, pregnancy, and childbed predispose it to inflammation. This predisposition may be increased by hyperæmia, passive as in affections of the heart, lungs, and liver, or active as in sexual excesses, and by changes in nutrition in such affections as chlorosis, anæmia, and Bright's disease. The exciting causes of inflammation come from outside the body and may be mechanical, as in the case of lacerations or foreign bodies, thermal as in burns, chemical as in the use of caustics, or infective from the action of germs. Or they may come from inside, as in the case of fistulæ, where decomposing urine or fæces may irritate the mucous membrane, or in septic, carcinomatous, or other ulcers, from which the putrid or purulent discharges flow over the surface. Not unfrequently a vaginitis arises by extension from the neighbouring surfaces of the vulva, cervix, or body of the uterus. It is probable that in nearly, if not quite, all cases the *causa causans* is a micro-organism, but much work yet remains to be done before this can be looked upon as scientifically proved.

Vaginitis may be divided into catarrhal, exudative or pseudo-membranous, ulcerative, and specific.

1. Catarrhal vaginitis is by far the most common form; it may be acute or chronic, and is characterised by alterations in the secretion, and by redness and sometimes swelling of the mucous membrane. The secretion shows changes in quantity and in quality; in some cases there is an increased desquamation of the squamous epithelium cells in the form of thick, white flakes like milk curds, and in other cases the discharge of serum is increased, and gives rise, with these whitish masses, to a more or less milky, serous fluid. In acute infective processes suppuration occurs, and causes the discharge to take on a yellowish tinge; the more acute the process, the more yellow and purulent is the secretion.

The vaginal mucous membrane varies in appearance with the age of the patient and the acuteness of the process. In acute cases in young women the whole surface is diffusely reddened; but careful examination shows even here that the redness is more marked on the summits of the folds of the mucous membrane. The redness varies in shade in different cases, and there is sometimes a slight bluish tinge added to it. It may be equally diffused over the whole vagina or may be limited to a part, especially near the entrance or at the upper part of the posterior wall. In milder and more chronic cases red spots and streaks are the only signs, and these spots are specially marked in the vagina of old women where the general surface of the mucous membrane is smooth and yellow. The redness may be due to congestion, and disappear on pressure, or to ecchymosis, in which event the colour does not disappear on pressure (macular vaginitis). In some cases where the inflammation is more intense, and especially in pregnancy, the spots form distinct prominences (granular vaginitis). Rarely, a little fluid accumulates in the spots (vesicular vaginitis); as a rule, the vesicles have no special importance, but they have occasionally been found in conjunction with skin pemphigus. Occasionally, in more chronic conditions, there is hypertrophy of the papillary bodies, and the epithelium becomes secondarily hypertrophied, so as to form small white, firm, warty elevations, which are found scattered over the vagina or portio, feeling like grains of sand (warty vaginitis). This condition is usually, but not always, a gonorrhœal affection, and is especially met with during pregnancy. Sometimes, especially in elderly women, but occasionally also in younger ones, erosion of the epithelium covering the red spots takes place so as to lay bare the inflamed papillæ, which may then become adherent to similar spots on the opposite wall by fine bands or membranes, easily breaking down, with slight hæmorrhage, under the pressure of the examining finger (senile adhesive vaginitis). In addition to the increased redness of the mucous membrane, there is sometimes, especially in young women with acute catarrh, distinct œdematous swelling; in such cases the lining of the vagina is of soft, velvety consistence.

Microscopically, vaginitis is characterised in the more acute forms by injection of the blood-vessels, and by diffuse infiltration of the papillæ with small round cells, which are also found wandering through the epithelial layer in considerable numbers. The red spots above described are found to be due to increased vascularity and infiltration with round cells of a group of papillæ. The individual papillæ in such a group swell laterally and tend to become confluent, and also extend vertically, so as in some cases to become prominent above the surface. The epithelium covering such a spot may become thin, allowing the red colour of the papillæ to shine through, or may be thrown off, so as to lay bare the papillæ; or, in more chronic cases, may become greatly hypertrophied and thickened so as to form warty elevations.

Gonorrhœal Vaginitis.—Among the causes of vaginitis the gonococcus is the most frequent and the most important. This germ sets up an acute purulent vaginitis, which shows the usual naked-eye and microscopic characters of a severe catarrh, with the addition that gonococci in smaller and larger heaps and chains are found scattered through all the layers of the epithelium, either between the cells or typically inside the epithelium cells or pus corpuscles. Sometimes the germs are found to have penetrated deep into the sub-epithelial connective tissue. The course of gonorrhœal vaginitis is usually towards rapid recovery, the disease frequently, however, persisting in the cervix and urethra. In some cases the acute vaginitis gradually subsides into a chronic catarrhal inflammation, often with acute exacerbations, which are due to repeated infections, either from without or from the cavity of the cervix or body of the uterus.

The diagnosis of the gonorrhœal nature of a vaginitis depends on the recognition of the gonococcus; the vaginal secretion of adults is not suitable for this observation, and therefore the search has to be made in secretions taken from the urethra or cervix. The absence of gonococci does not prove that the inflammation is not gonorrhœal, but the examination should be repeated after a menstrual period, or during an exacerbation of the catarrh. In chronic cases, clinical signs are of great importance. Thus an acute, purulent urethritis is only rarely due to any other cause than gonorrhœa; chronic urethritis with milky secretion is most frequently the remains of an old acute gonorrhœal affection, though it is certainly sometimes observed without gonorrhœa. Infection of Skene's ducts, and red spots round the urethra, have a similar import. Purulent infection of Bartholin's glands, pus escaping from their ducts, tumours, abscesses, or cysts in the characteristic position of the glands, are all very suggestive, but not diagnostic of gonorrhœa. Red spots like fleabites about the orifices of Bartholin's ducts—the macula gonorrhoica of Sänger—are also suggestive; and vulvar warts are usually but not always due to gonorrhœa. The probability of a gonorrhœal origin is greater when there is disease of the appendages, especially purulent salpingitis and pyosalpinx, with recurrent acute attacks of pelvic peritonitis. The history of the onset has only a limited value; the immediate commencement of a purulent inflammation after sexual connection or after marriage, and the occurrence of pain and frequency in micturition at the onset, have some value; the recognition of gonorrhœa in the male is important, but not often available.

Mycotic vaginitis is found most frequently in pregnant women with gaping vulva, and is characterised by bright redness and swelling of the mucous membrane, which is covered with small white patches rarely exceeding a pin-head in size. Discharge is absent in most cases, but where present it is of an irritating, excoriating character. The patients complain of intense burning, smarting, and itching, and the mucous membrane is excessively tender to the touch. The affection is caused by the growth of the oidium albicans or some allied organism, which is derived in many cases from an infant suffering from thrush. The disease is more frequent in summer, and damp dwellings appear to favour its occurrence.

Emphysematous Vaginitis.—This occurs most frequently during pregnancy, and then tends to disappear within a week or two after delivery; less frequently it is found arising in the puerperium. Small soft elevations, rarely larger than a pea, are found scattered singly or in groups over the surface of the vaginal mucous membrane; on the summit of the elevations the epithelium is often so thin that the projection is seen to be hollow, with the contents shining through; on pricking such an elevation it collapses, but no fluid, or only a very minute quantity, exudes; a little gas can sometimes be heard to escape with a hissing sound, or if the puncture is made under water a gas bubble is seen. The rest of the mucous membrane, as a rule, shows nothing peculiar; the vaginal secretion is usually somewhat increased, and is not seldom frothy. Zweifel found trimethylamine in the gas.

Under the microscope the changes are very characteristic; the connective tissue close under the epithelium is pervaded by round, elongated, or irregular cavities lying more or less close together. These cavities are not lined by epithelium or endothelium, but represent simple clefts in the connective tissue. In the immediate neighbourhood of nearly all the cysts there is a fairly considerable number of large giant-cells with many nuclei, exactly like the giant-cells found in the tuberculous collections. In addition, the connective tissue in the neighbourhood of the cavities shows dilated capillaries and profuse infiltration with small round cells. The cause of the condition is a micro-organism, but observers are not yet agreed as to its characters; it appears to be a bacillus. The affection tends to spontaneous disappearance after delivery, and calls for no special treatment.

Exfoliative Vaginitis.—Exfoliative vaginitis is a rare affection which is characterised by the discharge of membranous casts of the vagina, frequently accompanied by dysmenorrhœal symptoms. The vaginal membranes are composed of masses of squamous epithelium cells, and are readily distinguished from the uterine by the absence of the openings of the glands. The use of strong astringent injections is sometimes followed by the discharge of larger or smaller shreds of membranes, which may even form casts of the whole length of the vagina.

Vulvo-vaginitis of Children.—Vaginitis is a

frequent and obstinate complication of the vulvitis that is observed in infants and young children. This vulvitus may be due to various causes. Dirt is one of these, and worms form a second; these are, however, rarely efficient in setting up an inflammation of the vulva. The exciting cause in the great majority of cases is a micro-organism. Of the germs giving rise to vulvitis, by far the most frequent and important is the gonococcus, which is conveyed by means of unclean linen, bedclothes, or towels; thus Robinson (*Obst. Trans.*, 1899, vol. xli. p. 14) investigated 54 cases of vulvitis in children, and found that in 41, or nearly 76 per cent of them, the gonococcus was present in the discharge. In further proof of the infective nature of many of these cases are the facts that frequently the affection occurs in epidemic form, and that in many instances some other person in the same household is affected by a gonorrhœal, or at least a suspicious, discharge.

Many cases of vulvitis yield readily to simple treatment. A large proportion, however, obstinately resist medication, and it is found on closer examination that these are usually complicated by the presence of a vaginitis. The vaginitis is shown, in some cases, by the escape of pus at the vaginal orifice. Examination of the vagina, which may conveniently be done even in the youngest child by means of Kelly's bladder speculum, shows that some portion of the vaginal mucous membrane is red and raised above the surface and secreting. These patches are most commonly found in the upper part of the vagina, especially on the posterior wall. Treatment must be directed to the vaginal condition as well as to that of the vulva; the most convenient methods of treating the vaginitis are by passing thin bougies composed of cocoa-butter or glyco-gelatin, and containing iodoform, boracic acid, or protargol, and by injecting by means of a syringe with a long fine nozzle some solution, such as one of nitrate of silver, 5 to 10 grains to the ounce. At the same time, the usual treatment appropriate to uncomplicated vulvitis is required, along with careful attention to the general health of the child.

2. Pseudo-membranous Vaginitis. — Pseudo-membranous vaginitis is rare, and in the vagina, as elsewhere, no sharp division is possible between superficial and deep processes. The pseudo-membranes appear as small grey patches, which form especially on the summits of the folds of the mucous membrane, and by increasing in size and joining other patches may form extensive membranes. The mucous membrane surrounding the grey patches is, as a rule, markedly red and swollen, and may show scattered hæmorrhages. When the membranes are removed, small erosions or superficial ulcers are left, the healing of which, especially when they are situated in the upper part of the vagina, may lead to narrowing or atresia. This pseudo-membranous vaginitis is rarely met with as a real diphtheria due to Löffler's bacillus; sometimes it is present as a complication of the acute exanthemata, of cholera, typhoid fever, or dysentery. In these cases the process may extend more deeply and give rise to true ulcers, or sometimes even to dissecting para-vaginitis. The putrid discharges from a gangrenous ulcer, especially when complicated with urinary fistulæ, may also lead to pseudo-membranous inflammation of the lower part of the vagina. After labour a similar condition may arise, especially in the so-called diphtheritic endometritis, or the surface of injuries caused by labour may become covered with a grey layer — puerperal wound diphtheria.

3. Ulcerative Vaginitis.—Small superficial erosions are not infrequent in catarrhal vaginitis or as a result of slight injuries or of the application of caustics. In these cases there is no destruction of the connective tissue under the epithelium, and no scar is formed, although adhesions to other similar erosions may take place. Sometimes, however, as a result of inflammation there is actual destruction of more or less of the connective tissue with consequent formation of scar tissue. For instance, what for want of a better name may be termed *pressure sores* are met with either in the normally situated or in the prolapsed vagina. In the former case the most frequent cause is an ill-fitting pessary, which by its pressure gives rise to local anæmia and superficial gangrene; in this variety of pressure sore, long-continued irritation gives rise to extensive, small round-celled infiltration around the ulcer, and the pessary may eventually become buried in granulation tissue, which, after the removal of the offending object, may contract so as to cause stenosis or atresia of the vagina. In some cases the pressure may be so severe or long continued as to cause perforation into the bladder or rectum.

In old-standing cases of prolapse deep ulcers sometimes form which may attain the size of the palm of the hand, or may be multiple; they are usually met with on the anterior wall of the vagina and near the attachment to the cervix; the form of the ulcer may be round, triangular, or more often irregular; the edges are sharply defined and slope towards the base of the ulcer, which is depressed and covered by a dirty greyish layer, sometimes with scattered imperfect granulations; the neighbourhood of the ulcer is not reddened or infiltrated. The cause in these cases is partial strangulation along with repeated irritation, mechanical by friction against the thighs and clothes, and chemical by the flow of decomposing urine over the surface.

Parturition is another fertile source of ulcerative vaginitis; in every labour there arise in the vagina breaches of surface which may be

converted into ulcers by the action of septic or pathogenic germs. Such ulcers show a firmly adherent greenish grey membranous deposit, which is composed chiefly of dead tissue, and often leads to the term diphtheritic being erroneously applied to the affection. In the base of the ulcer and in its neighbourhood there are œdematous swelling and profuse infiltration of the vaginal walls, and the whole vagina is often swollen and brawny as the skin is in erysipelatous conditions; hence this condition has been described as erysipelatous vaginitis. In the more severe forms the inflammation spreads in the para-vaginal tissues and upwards to the rest of the pelvic connective tissue, the whole of which may become involved as the result of a severe infection in a quite small injury. The most frequent cause of this severe type of puerperal vaginitis is the streptococcus pyogenes.

A similar affection may occur as the result of operation wounds where antiseptic measures have been imperfectly carried out, or of other injuries inflicted upon the vagina. Ulceration may also arise secondarily from suppuration in the neighbourhood, and perforating the walls of the vagina; the forms of suppuration most frequently found pointing in this direction are para-metritic abscesses and suppurating hæmatoceles.

4. Specific Inflammations and Ulcerations.—Certain specific causes in addition to those already mentioned are also found giving rise to ulceration or to inflammation of the vagina, and it will be convenient to mention some of them here. Sarcoma and carcinoma usually, and simple tumours occasionally, are found inflamed and ulcerated. Tuberculous disease is usually met with in the form of ulceration. A *simple round ulcer* has been described by Zahn; this ulcer was circular in shape, had a red base and sharply defined sloping edges, and was supposed to be due to arterial sclerosis leading to deficient nutrition of a circumscribed portion of the vaginal wall. It is at least possible that this ulcer may have been tuberculous in origin.

Syphilis in any stage may be found affecting the vagina; the primary chancre is rare; in the secondary stage red patches or papules or mucous tubercles are observed; in the tertiary period deep gummatous changes are occasionally met with. Soft sores have been observed in the vagina, and as a rare occurrence phagedænic sores resembling those found on the penis have been encountered.

This is probably the most convenient place to mention Noma of the vagina, although it is really a gangrenous process. Noma usually spreads inwards from the vulva, but has occasionally been found arising primarily in the vagina, even at its upper part, where it very soon spreads to the cervix and often to the bladder. In many cases noma of the face has been present at the same time.

Tuberculosis of the Vagina.—Tuberculosis of the vagina as a primary affection is extremely rare, only one well-authenticated case having been placed on record. Secondary tubercle is very uncommon; it is generally secondary to disease of the uterus or vulva, and is thus usually situated at the upper end of the vagina or just above the entrance. It is also sometimes met with in pulmonary tuberculosis, or in conjunction with disease of the urinary organs or of the rectum. The miliary form of vaginal tuberculosis, where greyish-white tubercles are found disseminated, or grouped, or sometimes joining to form small nodules, is extremely rare. Usually the disease when present gives rise to an ulcer with edges sharply cut, more or less irregular or jagged, not raised or thickened; the base of the ulcer is uneven and discoloured with caseous spots and patches; there is a red areola in which, as well as in the base of the ulcer, small miliary tubercles may be recognised; and the ulcer is sometimes very tender. Occasionally in advanced cases of the affection recto-vaginal or vesico-vaginal fistulæ are found. Microscopic examination of the edges and neighbourhood of the ulcer shows the usual collections of round cells, together with giant-cells, and the characteristic bacilli.

The treatment of vaginitis calls for the employment of general as well as local measures. Any constitutional tendency or taint, such as struma, gout, rheumatism, or syphilis, should receive attention; the general health must be improved by every possible means, and care is to be taken to encourage the regular and adequate action of all the excretory organs—the bowels, kidneys, skin, and lungs. The local treatment varies with the stage and activity of the disease. In acute cases rest in bed is required. The exciting cause must, where possible, be removed, and care taken to prevent other causes arising or persisting; thus foreign bodies must be taken out, harmful or dangerous germs removed or destroyed by antiseptic applications, and discharges or membranes, which offer a suitable medium for the growth of germs must be got rid of. Finally, means must be employed to further the formation of healthy epithelium, and where there is any deeper loss of tissue to promote granulation.

The methods that have been devised to meet these various indications may be divided shortly into treatment by wet methods, treatment by tampons, and the dry methods of treatment. Among the wet methods, *douches* are chiefly useful in aiding to get rid of discharges and other products of inflammation; they should be employed in quantities of at least 3 or 4 pints, warm at a temperature which is most grateful to the patient, and should be administered at low pressure, the douche-can being raised not more than 18 inches above the level of the pelvis; the douche ought further to be given

slowly, and the patient should lie down during its continuance and for at least ten minutes afterwards. Danger arises from the employment of too great pressure, especially at the time of menstruation. Water alone should never be employed, as it appears to promote the growth of putrefactive germs in the vagina; and generally all non-astringent substances are objectionable, as they tend to macerate the surface epithelium. Sometimes, however, especially in the early stages of acute vaginitis, emollient and sedative douches are of service; for this purpose gruel or linseed-tea, or water with the addition of lead lotion, or of borax and soda, may be used. In sub-acute or chronic cases antiseptics or astringents are more useful, and for these purposes there is a wide choice, so that only a few of the more serviceable medicaments can be mentioned here. As antiseptics, chlorine water, permanganate of potash, boracic acid, corrosive sublimate, creolin, and lysol are most in use; and among astringents which are at the same time antiseptic, the sulphates of copper, zinc, and iron, nitrate of silver, the acetates of lead, zinc, or aluminium, oxycyanate of mercury, tannin, and alum.

Fluids may also be applied by means of what is known as the "local bath of the vagina." A cylindrical speculum is introduced into the vagina and filled with solution of sulphate of copper (2 to 5 per cent), nitrate of silver (5 per cent), pyroligneous acid, or solution of protargol; as the speculum is moved about and withdrawn the solution comes closely into contact with every part of the surface of the mucous membrane. Stronger fluids may be pencilled on to the surface or to some part of the surface with a brush; in this way pure alcohol, tincture of iodine, or strong alum solution may be employed; in syphilitic affections a thick paint made of calomel, salt, and water, and painted over the surface, is specific.

The tampon treatment is best carried out by the medical attendant, who through a Sims' speculum places pledgets of wool first in a circle round the cervix and then so as to fill the whole vagina. In this way direct pressure is obtained on the walls, in addition to the action of any drug that may be chosen. As a vehicle for the applications, glycerine is best; in it may be dissolved alum, boric acid, ichthyol, iodine, or potassium iodide, tannin, and iodoform. Instead of the wool tampon, a gauze strip saturated with some of the above-mentioned solutions is sometimes introduced into the vagina. Applications of this kind require to be renewed every two to four days.

Among the dry methods of treatment may be mentioned the use of medicated pessaries made with glycerine and gelatine, or with gum arabic; the use of the "powder bottle," a strip of gauze 8 inches by 4 inches in which is wrapped up a drachm of tannin and boracic acid or tannin and iodoform, of alum sugar 1 in 20, airol, or other powder; the insufflation of powders; or the introduction, on wool tampons or by means of a syringe, of ointments made of vaseline or lanolin to which astringents may be added, such as nitrate of silver, boracic acid, ichthyol, or white precipitate ointment. The ointment method is specially indicated where the entrance of the vagina is narrow, and in young children. The methods of dry treatment are especially useful in out-patient practice. In pregnancy no strong astringents should be employed in the treatment of vaginitis, but where necessary warm weak solutions of borax and soda, of boracic acid, or of permanganate of potash may be employed with safety.

5. PARA-VAGINITIS.—Inflammation of the connective tissue surrounding the vagina is most frequently caused by wounds, especially small ones, occurring in labour and becoming infected. Operations carried out with insufficient antiseptic precautions, neglected pessaries, and instrumental attempts at criminal abortion may also give rise to the affection. Occasionally in certain diseases, such as typhoid fever and pneumonia, a purulent inflammation undermining large parts or the whole of the vaginal wall has occurred (phlegmonous dissecting para-vaginitis). The intimate causation of this affection is still obscure, and it is possible that it may not unfrequently run its course in young people with typhoid fever, variola, scarlet fever, and possibly measles, without attention being called to the symptoms.

Diffuse or circumscribed collections of pus may make their way high up along the connective tissue surrounding the vagina and even into the bases of the broad ligaments. Portions or the whole of the vaginal wall may be cast off as sloughs. Infection may be conveyed by the lymphatic vessels to the pelvic glands and even to the peritoneum. When the patient recovers, cicatrisation may lead to varying degrees of stenosis or to complete occlusion of the vagina, with its sequelæ.

The symptoms may be overshadowed by a grave general disease. They consist in fever and local pain, with purulent and bloody discharges, and the separation of sloughs.

Treatment. — The separation of gangrenous portions of the vaginal wall should be promoted by warm antiseptic applications, and after the separation of the sloughs efforts should be made to prevent the contraction of the resulting scar tissue.

VAGINISMUS is the term applied by Marion Sims to certain spasmodic conditions of the pelvic floor which he was the first adequately to describe. In patients suffering from the affection any attempt at sexual intercourse calls forth violent contraction of the muscles surrounding the vagina, and if the efforts are persisted in the woman frequently cries out

involuntarily in fright and pain, and may assume an appearance of abject terror. The spasm affects the sphincters of the vagina and anus, the small perineal muscles, and the levatores ani; and it frequently extends to other muscles, more especially those of the buttocks and the abductors of the thighs. When such a patient presents herself for advice the gentlest attempt at examination usually calls forth similar phenomena and fails in its object. Complete local investigation is therefore possible only when the patient is anæsthetised, and in a well-marked case, unless the anæsthesia is deep, a touch on the genitals immediately calls forth the spasm. Careful examination may fail to disclose any abnormal physical condition; more frequently lesions are discovered, though these are often slight in appearance. The most common appearance found consists in red spots and patches at the urethral meatus, and in the vulva just outside the hymen or its remains. Sometimes one or more of the carunculæ myrtiformes shows the same red patches, which are due to thinning of the epithelium accompanied by increased vascularity and round-celled infiltration of the superficial layers of the corium. In other cases small fissures or excoriations may be present at the entrance of the vagina. Occasionally a more remote cause may be found for the spasm, such as vaginitis, a prolapsed ovary, or a fissure of the anus.

Opinions differ widely as to the essential cause of the spasm. According to one group of authors, some painful local condition is always the determining factor, and where no lesion can be discovered at the time of examination it is supposed that, after originating the vaginismus, the local condition has healed and left no trace. According to another set of observers, the affection is a neurosis, and is set up by crude or imperfect attempts at coitus. It is probable that each of these theories expresses a part of the truth, and that in some cases the condition is a pure neurosis, but much more frequently the spasm is set up by some small injury or painful inflammatory condition. In many cases it has appeared to the present writer that the exciting cause has been a result of gonorrhœal inflammation of the urethra and vulva, probably derived from a chronic urethritis or gleet in the husband.

The *treatment* calls for much patience and tact; any local lesion that may be discovered must be treated, and cautious inquiries should be made into the husband's condition, so that any lesion in him may be treated at the same time as the vaginismus. The most successful treatment consists in forcible digital dilatation of the vulva under an anæsthetic, followed by the daily passage of a vaginal dilator. For this latter purpose Hegar's dilators do very well. At first cocaine should be applied to the vulva before passing the bougie; a small size is used to begin with, and after being passed is fixed in position with a T-bandage and retained for one or two hours. Each day a larger size should be passed, until a dilator 1 inch can be used; then the bougies should be passed daily without the use of cocaine, again beginning with a smaller number and gradually increasing the size. It is of great importance not to cause pain during the passage of these bougies; otherwise the patient's confidence is destroyed. No undue haste should therefore be employed in increasing the size of the bougies. The treatment usually extends over at least three weeks, and may be considered complete when a bougie 1 inch in diameter can be passed without cocaine.

New Formations

Vaginal Cysts. — Cysts in the wall of the vagina are not in all cases derived from the elements of the vaginal wall itself, but on account of their common situation it is convenient to consider all such cysts together. They may arise from Gartner's duct, from the glands that are occasionally found in the vagina, or by adhesion of the contiguous summits of folds of the mucous membrane. Possibly also certain of them may be derived from dilatation of the lymphatic vessels, or as the end results of injuries which have given rise to hæmatoma. From some or all of these sources vaginal cysts are fairly common; they rarely give rise to symptoms, and thus are usually discovered accidentally. As a rule varying in size from a pea to a walnut, they are occasionally larger and may rarely attain to the size of a child's head, in which cases they may seriously interfere with labour and the other functions of the pelvic organs. Cysts are most frequently found in the anterior and lateral walls of the vagina, but are not rare on the posterior wall; they are usually superficial, and over the apex the mucous membrane is often thinned so that the cyst contents are seen shining through; rarely a cyst may become pedunculated. The smaller cysts extend into the para-vaginal connective tissue; the larger ones may extend upwards to the side of the uterus and spread into the parametrium. Frequently more than one cyst is present, and several are sometimes found arranged in a series parallel to the longitudinal axis of the vagina. Diagnosis is easy on combined examination by the vagina and abdominal wall, aided, if necessary, by the finger in the rectum and a sound in the bladder. The consistence is more or less firm elastic. The best treatment is by excision, but this is frequently not possible, the wall of the cyst forming merely a condensed layer of the surrounding connective tissue. Where excision is possible the resulting wound may be brought together by superimposed layers of suture, or may be packed with gauze and allowed to granulate. A simple incision into the cyst is useless. Where excision cannot be done, a

portion of the wall must be excised and the remains of the cyst treated by curetting, by the actual cautery, or by some strong caustic such as Liquor ferri perchlor. Where the lining of the cyst is not destroyed, a small depression secreting mucus may persist after operation.

Myoma and Fibroma.—Myoma and fibroma are rarely found in the vagina; they originate from the fibro-muscular wall. They form round tumours, seldom larger than an apple, and are embedded in or attached by a pedicle to the surface of the vaginal wall. The surface of the tumour may be smooth or lobulated, and is covered by intact mucous membrane. The consistence is usually hard like that of a uterine fibroid, but may be soft and fluctuating where œdema or cysts are present. The tumour is, as a rule, sharply marked off from the surrounding tissues, but sometimes spreads diffusely into them. The anterior wall of the vagina is much more commonly affected than the other walls; occasionally more than one tumour may be present. The treatment consists in making an incision over the tumour and enucleating it; the bed of the tumour is then either brought together by series of buried sutures, or partially closed and drained; where the tumour has become pedunculated it is removed after division of its pedicle.

Sarcoma of the Vagina.—Sarcoma of the vagina presents marked differences according as it is met with in childhood or in adult life. In infancy it occurs as a polypoid tumour affecting, in the great majority of cases, the anterior wall. In its early stage it occurs as a more or less pedunculated growth, which, as a rule, first attracts attention by protruding into the vulva. As the tumour grows it forms a mass having numerous projections from the surface, which lend to it an appearance somewhat resembling that of a bunch of grapes or berries. As the disease progresses, the cervix and bladder tend early to become involved, and later the vulva and the inguinal glands; the uterine body and the broad ligaments frequently are implicated; the rectum never is invaded. In the later stages of the disease ulceration may take place, and lead to infection and the death of the patient; pyometra and purulent peritonitis have been several times observed. In other cases the tumour may cause death by pressing on the urethra, and setting up dilatation followed by infection of the bladder, ureters, and kidneys. Remote metastases do not appear to be common; in one case, however, the left ovary was found enlarged to the size of an orange.

Microscopically, the tumour is a sarcoma composed of round and spindle-cells with some giant-cells; a varying quantity of connective tissue and some smooth muscular fibres are generally present; striated muscular fibres have been observed in many cases.

The first symptom frequently is the appearance of the tumour at the vulva; sometimes, however, this is preceded by pain in the pelvic region, which may be attended by bleeding, troubles of micturition, and, not unfrequently, difficulty in defæcation.

The duration of the disease varies; in the longest case hitherto observed the child died at six years of age. As a rule, the diagnosis is made too late for radical treatment, but in two cases excision has been performed with good results; in one of these two cases the lower half of the posterior wall of the vagina was removed, and in the other the whole vagina and uterus were extirpated through a parasacral incision.

In the adult, sarcoma has been observed at all ages up to eighty-two. The tumour may be chiefly composed of round or of spindle-cells with, not unfrequently, giant-cells, and frequently mucoid tissue. The disease usually appears as a circumscribed tumour, which may be situated in any part of the wall of the vagina. Occasionally the mass is polypoid; rarely it forms an annular constriction or even a diffuse growth which may narrow a large part of the vaginal lumen. The tumour may be entirely inside the vagina, or may present in the vulva. The symptoms, in addition to the presence of the tumour, are bleeding, discharge, and symptoms of pressure on the pelvic organs. The treatment consists in early and wide excision.

Carcinoma of the Vagina.—Carcinoma of the vagina may be secondary to disease of the cervix or body of the uterus, or to disease of the bladder or of the rectum. The vaginal affection may be due to direct extension of the growth, to contact infection, or to metastases through the lymphatics. Much less commonly, carcinoma of the vagina is met with as a primary affection, and then it begins, as a rule, in the upper third of the posterior wall, close below the insertion of the cervix. The common variety of carcinoma met with is a squamous epithelioma. The disease usually forms a circumscribed tumour, only slightly raised above the surrounding surface, and when first recognised is almost always already ulcerating and breaking down on the surface, so as to form an irregular ulcer with hard infiltrated base and sharply defined margins; the floor of the ulcer frequently shows fine papillary outgrowths of small roundish elevations. In the neighbourhood of the ulcer the mucous membrane is usually macerated by the discharges from the ulcer flowing over it, and shows signs of catarrhal inflammation with superficial erosions which easily bleed. Occasionally, in the immediate neighbourhood of the ulcer there is thickening of the epithelium forming white, opaque, shining patches, which apparently represent a precancerous stage of the affection. Sometimes the proportion of new-growth to ulceration is greater, and the tumour may then form a mushroom-shaped projection

more or less distending the vaginal lumen. In some cases, less frequently met with, the epithelioma infiltrates the whole or the greater part of the length of the mucous membrane, so that the vagina is transformed into a rigid, unyielding, and narrow tube. In all forms the glands are stated to be early implicated.

The growth extends along the surface, the cervix, especially the posterior lip, being early involved; sometimes the vulva is invaded, and then the inguinal glands may be affected. Extension also takes place deeply through the wall of the vagina into the para-vaginal tissue and upwards into the parametrium, as well as backwards round the rectum.

Epithelioma of the vagina is usually a disease of advanced life, but has occasionally been met with in women under thirty. In several cases the long-continued wearing of a pessary has preceded the appearance of the disease, and in a few instances it has arisen on a prolapsed vagina. The symptoms are the same as those of cancer of the vaginal portion of the cervix. The first symptom is always bleeding, especially on touch, and thus especially in sexual connection; wasting is often observed as an early symptom. Later in the history of the case come fœtid discharge and pain, and finally the cancerous cachexia, with frequently the formation of fistulæ, especially recto-vaginal. The diagnosis depends on the recognition of induration with peculiar superficial unevenness of the surface, and friability. Difficulty may arise in distinguishing the affection from sloughing myoma of the vagina, and from ulcers attended by some degree of proliferation, such as may be set up, for instance, by a badly fitting pessary. The prognosis is very grave, even after operation.

The treatment consists in the radical removal of the disease, when it is recognised sufficiently early. The removal has been carried out through the vagina, through the perineum, and by the sacral method. Olshausen's perineal method (*Cent. für Gynäk.*, 1895, p. 1) is probably the best. In this the perineum is divided transversely, and blunt separation of the vagina from the rectum is continued as high as the bottom of Douglas's pouch; the peritoneum is opened behind the vagina and the uterus drawn down through the opening. The broad ligaments are then secured, beginning at the upper border and working down towards the side of the cervix. Finally, the anterior wall of the vagina is separated from the bladder.

In circumscribed tumours a partial excision of the vagina may suffice, but in all advanced cases the whole of the tube should, if possible, be removed. In women who still menstruate it is usually necessary to remove the uterus at the same time; in older women this is not so essential. Where the disease is already too far advanced for radical operation, treatment can only be palliative, and is carried out on the usual lines.

Injuries of the Vagina.—Accidental *wounds* of the vagina rarely are met with as the result of falls on to such objects as the leg of a stool or the point of a hay-fork. Occasionally lacerations are caused by coitus, but in such cases the injured vagina is the shrunken, inelastic canal of an old woman, or is abnormally formed or altered by cicatrices. In criminal attempts at abortion, wounds are not unfrequently made, and in these cases the wound is usually in the posterior fornix behind the cervix. The injuries that are caused by labour are the most frequent and important class, and are considered separately (vol. v. p. 268).

By whatever cause produced, wounds may give rise to serious hæmorrhage, which is, where possible, best treated by appropriate sutures, or, failing this, by packing with iodoform gauze. Many of the injuries are of importance only from the damage that is at the same time caused to neighbouring organs, the bladder, ureters, or rectum. In all wounds of the vagina there is risk of septic infection, to prevent serious consequences from which it is necessary to provide for adequate drainage.

Among the uncommon affections of the vagina may be mentioned *hæmatoma*, by which is meant hæmorrhage into the connective tissue surrounding the tube. This is extremely rare apart from pregnancy, and is due to an injury which causes no superficial wound or only a small one, while at the same time a vein or small artery is ruptured. A tumour forms in the course of a day or two, and is made up of blood, usually venous, partly fluid and partly clotted. The tumour is irregular in shape, has well-defined borders, and is not very tender; its consistence varies in different parts, is for the most part tense elastic, or may be fluctuating; the mucous membrane covering the tumour is livid and discoloured, and there may be a small breach of surface. The treatment of hæmatoma is in the first place expectant; if the tumour continues to increase in size an incision may be necessary, the clots being turned out and the cavity stuffed with gauze. At a later stage suppuration may take place, and a free incision is then demanded.

Foreign Bodies.—Foreign bodies may be introduced into the vagina for the purpose of mechanical treatment; thus pessaries are frequently left in the vagina, and may be neglected or even entirely forgotten by the patient. Not rarely objects are introduced into the vagina for the purpose of sexual gratification, either by the patient herself or by another person; bodies of the most varied and often surprising nature may thus be found, such as pieces of sponge, glass stoppers, spoons, drinking-glasses, hair-pins, and tobacco-pipes. A pessary properly constructed and applied, and made of a suitable

material, and carefully supervised, may be worn by a patient for years without any evil effects arising. In some cases, but then usually only after the body has remained in position for some time, serious and even fatal consequences may ensue. By pressure a local ulcer may arise, which in turn becomes infected by germs and transformed into a spreading ulcer; the ulceration extends deeply into the surrounding tissue, and may even penetrate neighbouring organs, the bladder, the rectum, or a higher part of the intestinal tract, or even the peritoneal cavity. Bridges of connective tissue may form and imprison the foreign body, and polypoid growths of the cervix or vaginal walls may arise. Putrid and purulent discharges are common. After the removal of the body urinary or fæcal fistulæ may persist, and contraction of scar tissue may take place and give rise to stenosis or to complete atresia, and their consequences. In several cases carcinoma of the vagina has appeared to be caused by the long-continued irritation of a pessary.

Treatment is required to prevent evil consequences arising from the use of a pessary; this includes, besides the correct sort of material and form for the instrument, constant attention to cleanliness, and regular supervision by the physician, who should remove and examine the pessary once in every two or three months; any pain or irritation should lead to a prompt examination. The treatment of neglected cases consists in the removal of the foreign body, a proceeding often giving rise to much difficulty and anxiety; in counteracting the evil effects that have arisen from the presence of the body; and in the attempt to prevent injurious contraction of the resulting scar tissue.

Acquired Stenosis and Atresia of the Vagina.—In their effects, symptoms, and treatment, these conditions present many points in common with the congenital affections of the same name (vol. iii. p. 448).

It is probable that many of the so-called congenital cases are really acquired either during intra-uterine life or in childhood. In the adult the chief source of these conditions is severe labour and its consequences, especially septic infection supervening on breaches of surface and leading to extensive ulceration or even gangrene of the vagina. Sometimes constriction is set up by other causes, as for instance by foreign bodies, the application of strong caustics to the cervix or vagina either for legitimate medical treatment or by criminal abortionists, or it may be due to septic infection of operative wounds on the vagina.

As a result of some of these causes scars of varying thickness and extent are found in the vagina. These may take the form of longitudinal bands, frequently extending upwards from the neighbourhood of the pubic ramus; or they may be present as semilunar folds or as complete rings round the vagina giving rise to annular stricture. The lumen may be almost completely occluded by a thin membrane with a small opening through which a fine probe passes with difficulty, or it may be reduced to a more or less narrow canal by a broader scar surrounding it for some distance. Complete atresia may similarly be due to a thin membrane or to a scar several centimetres in thickness, or finally the whole tube may be completely destroyed.

Urinary Fistulæ.—Urinary fistulæ may be caused by the misuse of instruments, such as blunt or sharp hooks, in effecting delivery, or more frequently by pressure necrosis in severe cases of obstructed labour. Sometimes the vesico-vaginal wall is perforated by foreign bodies such as ill-fitting pessaries or by stone in the bladder. Of recent years a prolific source of urinary fistulæ has been the great increase in the number of operations on the genital organs, and especially of total extirpations of the uterus.

The position of the fistula varies. The most common form is the vesico-vaginal fistula where the bladder is affected; this may extend so far downwards as to implicate the vesical opening of the urethra, or so far upwards as to involve the cervix. The urethra alone may be affected (urethro-vaginal fistula); the fistula may involve the cervix, either along with or independent of the vagina (cervico-vesical fistula and cervico-vagino-vesical fistula). Uretero-vaginal fistulæ are also far from rare, especially as the result of vaginal hysterectomy.

Urinary fistulæ are usually single, but occasionally more than one is present. The vesico-vaginal form is the most frequent; its borders may be sharp and well defined, or may be thickened and sometimes widely surrounded by scar tissue. In size the fistula may vary from an orifice so small as to admit a fine probe with difficulty to the size of a threepenny piece or a florin, or even larger. Sometimes in large fistulæ laterally situated the ureter is found opening in the wall.

Cervico-vesical fistulæ affecting the upper part of the cervix are always small. Uretero-vaginal fistulæ appear either as small clefts with sharp edges or as openings on small elevations; those arising from labour are found near the side of the cervix in a depression running outwards from the external os or a little behind this level. In these fistulæ the vesical end of the ureter frequently tends to become obliterated. In the puerperium urinary fistulæ usually become much smaller, and the process of contraction continues until the borders are covered with epithelium. Affections of the upper urinary tracts—dilatation of the ureters, pyelitis, stone in the kidney, abscesses, and so on—are not infrequently met with, especially in fistulæ of some standing, and are of great importance as influencing the prognosis, especially

where operation is contemplated. Amenorrhœa is frequently observed, and menstruation first returns after a successful plastic operation.

In vesico-vaginal fistulæ the whole of the urine dribbles away as it is formed, though it is not uncommon for a certain amount to be retained so long as the patient lies on the back. In uretero-vaginal fistulæ half the urine dribbles away from the vagina, and the other half is passed naturally by the urethra. It is sometimes difficult to decide whether a patient with incontinence of urine is the subject of a urinary fistula or not, and in these cases the injection of a coloured fluid, such as milk and water in equal quantities, into the bladder, while at the same time the vagina is carefully observed through a Sims' speculum, easily enables a diagnosis to be made.

The treatment of vesico-vaginal fistulæ consists in a plastic operation, to which there are practically no contra-indications. In exceptional cases where the closure of the fistula itself is impossible, the closure of the vagina below the seat of the fistula, or closure of the vulva, is performed, but in nearly every case the plastic operation is carried out on the fistula itself. The best time for the operation in a case arising after parturition is six to eight weeks after labour, at a time when the lochia has ceased, and the tissues are still vascular, but sufficiently firm to hold sutures. Attempts to close the fistula by means of cauterisation, as, for instance, by the actual cautery, nitrate of silver, chromic acid, caustic potash, and the like, have frequently been made; these attempts are useless and harmful except in the case of small fistulæ with edges still granulating, such as are seen soon after labour, or after a plastic operation where there is a little leaking in the line of union, or along a suture; in such cases the application of nitrate of silver is sometimes useful.

In all ordinary cases plastic operation on the fistula itself, by the preparation of raw surfaces and the application of sutures, is the method to be employed. In many cases a preparatory course of treatment is necessary to get rid of complicating irritation and inflammation, especially where these are due to the flow of alkaline putrid urine, which at the same time precipitates lime salts on the raw and inflamed surfaces; here bland, demulcent drinks, the frequent use of irrigations to which some mild antiseptic is added, and the administration of a mixture containing oil of turpentine, salol, or urotropin, are useful. Where there is much cicatricial contraction of the surrounding tissues Bozeman recommended that a preparatory dilatation should be undertaken by means of glass bottles, rubber plugs, or cylinders, and incisions, but the scars may be much more advantageously dealt with by the knife and speculum in the first stage of the plastic operation.

The operation may be described as consisting in three stages. The first stage consists in rendering accessible the fistula, the second in preparing the raw surfaces, and the third in applying the sutures.

First Stage.—The patient is placed in the lithotomy position; occasionally the semi-prone or the knee-elbow position may be found preferable. The usual preparatory cleansing is made, and the fistula is then rendered accessible by some modification of the Sims' speculum and by lateral vaginal retractors; the uterus may be pulled down by a volsella, or the edges of the fistula may be made prominent by a catheter passed through the urethra, or by a sponge held in forceps and passed through the fistula into the bladder. The urethra, which is always contracted in fistulæ of any standing, should be dilated. Bands of scar tissue may be stretched by the pressure of speculum and retractors, or an incision may be made through their thickness. In stenosis of the vagina it is often most difficult to expose the fistula properly, especially where it is adherent by scar tissue to the back of the pubes; in such cases the tissues surrounding the fistula may be loosened from the bones by an incision made external to the labium majus and carried down to the ramus of the pubes, or it may even be necessary to make an incision above the symphysis pubis, and in that way detach the borders of the fistula.

Second Stage.—A raw surface may be obtained either by denudation or by the flap-splitting method The important points to aim at are that the raw surfaces shall be sufficiently broad, that they shall come together without tension on the sutures, and that they shall be free from scar tissue and bleed freely. A denudation may be narrow as in Simon's method, where an incision is made all round the borders of the fistula, embracing the whole thickness of the wall, vesical as well as vaginal; or it may be wide as in the so-called American method, where the vesical wall is not touched, but a broad strip, half an inch or more in width, is removed from the vaginal wall all round the orifice of the fistula. In any case it is best to first mark the outer border of the flap proposed to be removed by the scalpel; afterwards the flap may be cut off by means of either scissors or sharp-pointed scalpel, the borders being held taut by fine sharp hooks or by hook forceps. If the ureter is present in the wall of the fistula it must be preserved.

The method of preparing raw surfaces by flap-splitting presents many advantages. In it the wall of the bladder and that of the vagina are separated from each other for the required distance all round the fistula, and the two walls are then sutured separately. There is no loss of tissue, and if the operation fails the conditions for the next attempt are no worse; a very broad surface can be obtained for suturing,

and, further, the sutures can be applied with much less danger of tension.

Third Stage.—The sutures may be applied by means of handled needles with suitable curves, or by small curved and half-curved needles with a needle-holder. The material employed may be silver wire, silkworm gut, or fine silk. Deep sutures at intervals of about a quarter of an inch should be passed under the whole width of the raw surface and brought out near its borders; all the sutures should be placed before any are tied. The direction of the line of union is often determined by the shape of the fistula or of the freshened surface; where there is a free choice a transverse line is preferable, because then there is less risk of tension on the sutures; in large defects the union will often not be in a single line, but will have a Y or T shape. After all the sutures are introduced, the wound is sponged dry, and the sutures are tied. Finally the bladder is washed out to remove any blood that may have entered and to make sure that the closure is perfect.

Where the flap-splitting method has been employed, the vesical wall is brought together by one or more layers of interrupted catgut sutures, the vaginal wound being then closed by silk or wire or silkworm gut sutures.

The after-treatment is simple. The wound is powdered with iodoform at the close of the operation, and is then left completely at rest until the sutures are removed; only if there is some special indication are vaginal douches needed. The catheter is passed every two or three hours until the patient is able to pass urine naturally, which she is allowed to do as soon as she is able. Ten days is the usual term for complete rest in bed; an easily digestible diet should be ordered, and daily evacuation of the bowels secured. Silk sutures may be removed on the eighth or ninth day; wire or silkworm gut may be left for a week or two longer. The failure of the operation is marked as a rule by irregular rise of temperature, or by some incontinence making its appearance again on the third or fourth day. Sometimes, however, the failure is not discovered until the patient is allowed to get up or the sutures are taken out. The cause of the incontinence may now be a stitch-hole, which will heal up with a few days' extra rest in bed, or after a few applications of nitrate of silver. If the operation has to be repeated, this should be done two to three weeks after the first one.

Recto-vaginal Fistulæ.—Sometimes after labour a complete rupture of the perineum may be followed by a superficial healing which leaves a communication between the rectum and the vagina, and the same result occasionally follows badly executed plastic operations for complete rupture. Fistulæ may also arise from partial gangrene during the puerperium, from long-continued pressure from a pessary, especially Zwanck's, or occasionally from a wound. Finally, recto-vaginal fistulæ are also found in advanced cases of malignant disease of the cervix, vagina, or rectum, and in the group of affections which are included under the name ésthiomene.

Fistulæ between the vagina and the large intestine above the rectum, or between it and the small intestine, are also occasionally met with. They are always situated high up in the posterior vaginal wall, and may arise either as a result of a vaginal rupture in labour, which allows coils of intestine to protrude, or as a result of a fall on a sharp penetrating object, such as a hay-fork. Occasionally a fistula of this kind may arise indirectly from an abscess in Douglas's pouch, usually the result of a salpingitis, opening into the vagina and the rectum; the abscess may be tuberculous. In these cases of intestino-vaginal fistula, either the whole contents of the bowel may pass through the fistula—an artificial vaginal anus; or only a part may pass, in which case the term intestino-vaginal fistula is appropriate.

The symptoms vary with the size of the fistula. In very small openings there may be a valve-like closure and no symptoms; in other cases flatus and fluid motions pass through the fistula, and in still larger ones most of the evacuations may pass through the fistula and thus through the vulvar orifice. The symptoms of a communication between the vagina and the small intestine consist in the passage of the characteristic contents of the small intestine at a longer or shorter interval after meals. Where all the contents of the small intestine pass through the vagina the general condition of the patient rapidly deteriorates, and on this circumstance, as well as on the cause of the fistula, the prognosis depends. Where only the smaller portion of the contents pass through the vagina, the fistula shows a great tendency to heal spontaneously, although the process of healing may take a very long time. It is important to bear this in mind, because it follows that operation should not be undertaken too hurriedly in these cases. Where there is an artificial anus the best treatment probably consists in laparotomy and separation of the adherent and fistulous intestine, followed by sutures or by anastomosis as may be indicated. Where the fistula depends on tubercular disease it is best left alone.

The prognosis of recto-vaginal fistula is the same as that of the condition which gives rise to it. The treatment in simple cases consists in plastic operation. This is usually carried out through the vagina, and comprises two steps: (1) the preparation of a raw surface, and (2) the application of sutures. The raw surface may be obtained either by denudation of a strip of vaginal mucous membrane bordering the fistula; or by a process of flap-splitting, the

vaginal wall being separated from that of the rectum for a sufficient distance all round the margin of the fistula. In the second step of the operation sutures are introduced; in the case of denuded surfaces one row of interrupted sutures is applied, the material employed being silkworm gut, wire, or silk. Where the flap-splitting operation is chosen the rectal orifice is usually closed by a layer of buried catgut sutures, and the vaginal wound is then brought together by interrupted silk or silkworm gut sutures. The method of flap-splitting has the great advantages that by it no tissue is lost, and that there is less chance of tension on the sutures. When the fistula is small and situated at the lowest part of the vagina, just above the perineal body, it is best to split the perineum and to operate in the way that is usual for this condition. The after-treatment in all cases is just the same as that following the operation of perineorrhaphy.

Vaginal.—Belonging to the vagina, or, less often, to any sheath-like structure, *e.g.* the vaginal arteries and vaginal plexus, and the vaginal coat of the eye (capsule of Tenon).

Vaginectomy.—Excision of the vagina or colpectomy, recommended in inveterate cases of prolapsus uteri in elderly women.

Vaginismus.—A painful contraction of the sphincteric muscles of the vagina, either at the vulva (inferior vaginismus) or higher up near the vault (superior vaginismus). *See* VAGINA, DISORDERS OF (*Vaginismus*).

Vaginitis.—Inflammation of the vagina. *See* VAGINA, DISORDERS OF (*Inflammatory Affections*).

Vagino-.—In compound words *vagino-* means relating to the vagina of the female or to any sheath-like structure; e.g. *vaginodynia* (vaginal neuralgic pain), *vaginofixation* (fixation of the uterus by means of an incision through the fornix vaginæ and suturing of the uterus to the anterior vaginal wall), *vaginotomy* (incision of the vagina, or vaginal section), and *vagino-vesical* (relating to the vagina and the bladder).

Vagitus.—The cry of a new-born child, and, in a special sense, the cry of a child while still in the mother's birth-canal (*vagitus uterinus*).

Vago-.—Relating to the vagus nerve (*q.v.*), as in *vagotomy* (section of the vagus) and *vago-accessorius* (the nerve-carrying fibres of the vagus and of the spinal accessory nerves).

Vagrant's Disease. *See* VAGABOND'S DISEASE.

Vagus Nerve. *See also* ALCOHOLISM (*Visceral Variations*); ANEURYSM (*Neck, Operative Treatment, Risks*); AUDITORY NERVE AND LABYRINTH (*Nerve Deafness*); BRAIN, PHYSIOLOGY OF (*Cranial Nerves, Origin of*); EPILEPSY (*Symptoms, Epigastric Aura*); HEART, PHYSIOLOGY OF (*Cardiac Nervous System*); LARYNX, NEUROSES OF (*Innervation of the Larynx*); PHARYNX, EXAMINATION OF (*Neuroses of Sensation*); PHYSIOLOGY (*Cranial Nerves, Circulation, Respiration*); PULSE (*Influence of the Vagus*); STOMACH AND DUODENUM, DISEASES OF (*Anatomy, Nerves*).—The vagus, pneumogastric, or tenth cranial nerve is a mixed nerve with a very wide peripheral distribution.

Anatomy.—The vagus nerve is intimately associated with two nuclei in the medulla oblongata.

The *dorsal nucleus*, to which a large portion of the vagus trunk proceeds, lies beneath the floor of the fourth ventricle, immediately external to the hypoglossal nucleus and internal to the nuclei of the posterior columns. The prominence on the floor of the ventricle which is known as the ala cinerea marks the position of this nucleus. A large portion of the glosso-pharyngeal nerve terminates in the same group of cells, which is for this reason commonly called the vago-glosso-pharyngeal nucleus.

The dorsal nucleus of the vagus is generally looked upon as having a sensory function, although some writers hold that motor fibres also arise from it.

Some of the vagus fibres take origin from a smaller mass of grey matter which lies ventral to the nucleus above mentioned and detached from it. This is the accessory vagal nucleus, or *nucleus ambiguus*. The seventh nerve and some fibres of the glosso-pharyngeal arise from an upward prolongation of the same group of cells, while from its downward continuation into the spinal cord the eleventh nerve takes origin.

The nucleus ambiguus is regarded by the majority of recent writers as the motor nucleus of the vagus. Marinesco is of opinion that the fibres which supply striped muscle arise from this nucleus, while those innervating non-striped muscle originate in the dorsal group of cells.

A small part of the entering vagus root has been traced into the *fasciculus solitarius*, a compact bundle of fibres which lies external and slightly anterior to the dorsal nucleus. This bundle also contains glosso-pharyngeal fibres. It passes caudally, and gradually splits up into end ramifications, which terminate in the adjacent grey matter of the medulla. The fasciculus solitarius is sometimes known as the ascending vago-glosso-pharyngeal root.

The fibres from the nucleus ambiguus pass for a short distance backwards; they then run forward at an acute angle, those from the anterior part of the nucleus joining the fibres passing from the dorsal nucleus to form the common vagus trunk, while those which arise posteriorly after leaving the brain unite with the nerve of Willis to form the spinal accessory.

The ninth, tenth, and eleventh cranial nerves pass together through the jugular foramen.

Function.—The vagus has a very wide distribution, supplying muscle, skin, and viscera. It is distributed to the pharynx, larynx, heart and lungs, œsophagus, stomach, and in part to the intestine. A small branch innervates the skin of the external auditory meatus.

Lesions.—The nerve may be involved by lesions within the medulla, at the base of the brain, or in its peripheral course outside the cranium.

The nuclei, or the intramedullary fibres proceeding from them, may be damaged or destroyed by hæmorrhage, acute inflammatory conditions, softenings due to arterial occlusion, tumours, or chronic degenerative processes. Various affections of the meninges, especially syphilitic meningitis and meningeal hæmorrhage, may damage the nerve trunk in its intracranial course. In this situation the nerve may be pressed on by a tumour, or more rarely by an aneurysm of the vertebral artery. In its peripheral course the vagus may be implicated in a tumour, and it is sometimes divided by the surgeon, or included in a ligature when the carotid is tied. A neuritis has been met with after a number of the infective fevers, more especially diphtheria, while alcohol, and, in rare cases, arsenic and lead, may act as the determining factor of the morbid process.

The recurrent laryngeal nerve is very frequently involved in cases of aneurysm and intrathoracic new growths.

Symptoms.—The vagus has a very extensive distribution, and the symptoms produced by a lesion affecting it are necessarily widespread and various. They depend largely upon the site of the lesion. Complete paralysis of the whole trunk is not uncommon where the nerve is involved at the base of the brain. The ninth, twelfth, and upper part of the eleventh nerves are usually involved at the same time in the case of lesions in this situation. Unilateral paralysis of the palate, larynx, and tongue, with difficulty in swallowing, is a typical combination of symptoms produced by such intracranial lesions. Injury to the nerve high up in the neck is often attended with the same symptoms. In such a lesion the tongue escapes, and difficulty in swallowing is not usually pronounced.

Increase of the pulse-rate is commonly present; the respirations are sometimes increased; more rarely both the rate of the pulse and respirations are diminished. These cardiac and respiratory symptoms are usually not pronounced unless both nerves have been injured. Pneumonia has occasionally followed division of the vagus. Vomiting, gastric pain, loss of hunger and thirst, which have been observed, are attributed to involvement of the gastric branches. As has been already stated, paralysis of the recurrent laryngeal nerve is frequently met with. This nerve curves around the arch of the aorta on the left side and the subclavian artery on the right. It supplies the mucous membrane of the larynx below the vocal cords, and all the muscles of the larynx except the crico-thyroid, which is supplied by the superior laryngeal.

In consequence of pressure on the nerve, unilateral abductor paralysis is frequently met with. Where this is the case the voice is somewhat hoarse, and there is no movement of the corresponding cord on inspiration. There is seldom much dyspnœa. The abductors often become affected at a later date, when the loss of voice may be pronounced.

In cases of bilateral abductor palsy the voice is little changed, but inspiration is difficult, and associated with a loud stridor.

Bilateral adductor paralysis is common in hysteria. There is an inability to approximate the cords, and, in consequence, a condition of aphonia. When the patient attempts to speak the cords remain in the position of abduction. There is no stridor or dyspnœa.

In cases of total bilateral palsy both cords are moderately abducted and motionless, there is complete aphonia, the patient cannot cough, and there is stridor only on deep inspiration.

The *treatment* in cases of paralysis of the vagus nerve must be directed to the cause of the paralysis.

Valée's Law.—The principle underlying the biological test for blood, or Uhlenhuth's test (*q.v.*). "If an animal, A, be inoculated repeatedly with an albuminoid material from an animal of a different species, B, the blood serum of A acquires the specific property of precipitating *in vitro* albuminoid fluids derived from animals belonging to the species B" (*Da Costa*).

Valencia. *See* THERAPEUTICS, HEALTH RESORTS (*Mediterranean*).

Valenta Test.—A method of discovering whether butter has had animal or vegetable fat added to it in place of the milk fat; the test consists in heating a mixture of the butter fat and glacial acetic acid (equal parts) to 100° C., and then allowing it to cool. If it be adulterated, opacity begins to appear at a temperature of 96·5° C.; if pure, not until the temperature has fallen to 61·5° C.

Valentine's Beef-Juice. *See* DIET (*Beef Juices*).

Valerian. *See also* ZINC.—The rhizome of *Valeriana officinalis* (*Valerianæ Rhizoma*) contains a volatile oil, which on decomposition sets free valerianic acid ($C_5H_{10}O_2$); the official preparation is the Tinctura Valerianæ Ammoniata (dose, ½ to 1 fl. dr.); valerian acts, by reason of the volatile oil in it, as a carminative and stimulant, and its use in hysterical and nervous maladies (alone or combined with

asafœtida) is empiric. The valerianates of quinine, zinc, and iron are used as tonics and nerve sedatives; the ammonium salt is a stimulant.

Valerian, Indian.—This is the rhizome and rootlets of *Valeriana Wallichii*, and it constitutes Valerianæ Indicæ Rhizoma, which is official in the Indian and Colonial Addendum to the B.P. of 1898; it has the actions and uses of ordinary valerian (*q.v.*), and there is an official preparation, Tincture Valerianæ Indicæ Ammoniata (dose, ½ to 1 fl. dr.).

Valescur. *See* THERAPEUTICS, HEALTH RESORTS (*Riviera*).

Valetudinarianism.—The invalid condition, or state of infirmity.

Valeur Globulaire.—The proportion of hæmoglobin in the blood to the number of erythrocytes, or the corpuscular richness of the blood in hæmoglobin; the blood quotient. *See* BLOOD (*Colour Index*).

Valgus.—The condition of club-foot in which there is eversion. *See* DEFORMITIES (*Toes, Foot, Leg*).

Vallecula.—An anatomical term meaning depression or furrow, *e.g.* the *vallecula cordis* or depressed area at the apex of the heart, between the anterior and posterior longitudinal furrows.

Valleix's Points.—Points tender to pressure, found along the course of affected nerves in neuralgia. *See* NERVES, NEURALGIA (*Symptoms*).

Vallum.—The outer margin or wall of the circular depression which surrounds each circumvallate papilla on the tongue; also the duplicature of skin at the lateral margins of a nail (*vallum unguis*).

Vals. *See* BALNEOLOGY (*France, Alkaline*); MINERAL WATERS (*Alkaline, Simple*).

Valsalva's Experiment and Method.—The forcing of air from the pharynx into the tympanic cavities by closing the mouth and nose and making a strong expiration; auto-inflation of the middle ear. *See* EAR, EXAMINATION OF (*By the Eustachian Tube*).

Valsalva's Sinus.—The pouch or pocket lying behind each cusp of the semilunar valves of the heart. *See* PHYSIOLOGY, CIRCULATION (*Heart, Structure*).

Valsuani's Disease.—Pernicious progressive anæmia as a complication of the pregnant state.

Valve.—A fold; *e.g.* the valves of the heart, of veins, of the rectum, or of the ileum at its junction with the cæcum. *See* PHYSIOLOGY, CIRCULATION (*Heart, Structure*, etc.).

Valvula.—A small valve or fold, *e.g.* the *valvulæ conniventes* or small transverse folds of the small intestine.

Valvular Disease.—Disease affecting the valves of the heart or arising in these structures. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Pathology, Valvular Affections*).

"Vana."—A pharmaceutical product containing "calcium glycerophosphate and the alkaloids of cinchona bark in a pure sound wine" (Burroughs Wellcome & Co.); it is recommended in convalescence, neurasthenia, and general debility, in doses of half a wineglassful, before or after food, three or four times a day.

Vapores.—Preparations which, in the form of inhalations, carry volatile drugs into contact with mucous membranes; there used to be five officinal *vapores* (of hydrocyanic acid, chlorine, conium, creosote, and iodine).

Vaporisation of the Uterus.—The cauterisation of the mucous membrane of the uterus by superheated steam (*atmokausis*) or by a metal catheter heated by the steam (*zestokausis*).

Vaporole or Vapourole.—A small glass capsule containing a volatile drug such as amyl nitrite; the capsule is broken and the drug inhaled.

"Vapours."—Hysteria or hypochondriasis, from the old belief that hysteria was due to the passage of vapours (*vapores uteri*) from the uterus to the brain.

Varese. *See* THERAPEUTICS, HEALTH RESORTS (*Italian Lakes*).

Varicella.

SYNONYMS 416
DEFINITION 417
ETIOLOGY 417
SYMPTOMATOLOGY 417
DIAGNOSIS 418
PROGNOSIS 419
PATHOLOGY 419
TREATMENT 419
RELATIONS TO PUBLIC HEALTH—PREVENTION 419

See also CONJUNCTIVA, DISEASES OF (*Exanthematous Conjunctivitis*); DISINFECTION (*Incubation Periods and Quarantine*).

SYNONYMS.—*Latin*, Varicella; *English*, Chicken-pox; *French*, Varicelle, variolette, fause vérole, vérole bâtarde, petite vérole volante, pustules de poulet, nasles; *German*, Windpocken, Wasserpocken, Varicellen, Schafblattern, Spitzblattern, Falsche Pocken; *Italian*, Varicella, Morviglione.

Some of these names indicate that the disease was formerly regarded as a minor form or variety of *variola* or smallpox.

DEFINITION.—A very infectious disease, occurring chiefly in children, attended by little constitutional disturbance as a rule, and displaying on the first day an eruption of small red papules, which are soon converted into watery vesicles—the vesicles ultimately drying up, and giving place to small crusts.

ETIOLOGY.—The disease is doubtless spread by a *contagium* derived from a previous case of the disease. Personal contact, or *fomites*, will convey the contagium.

Up till the present time, so far as the writer is aware, no specific organism has been identified in the disease. Attempts at inoculating chicken-pox have been attended with doubtful or unsatisfactory results. The possibility of doing so may be regarded as an open question.

SYMPTOMATOLOGY.—The period of incubation is variously stated, but the writer puts it at thirteen days (for convenience, say a fortnight).

Thomas gives thirteen to seventeen days, Trousseau fifteen to twenty-seven days.

The following may be taken as a description of an ordinary attack of chicken-pox. At the end of a fortnight, dating from the time of exposure to the contagium, the patient feels "out of sorts"—perhaps there is loss of appetite, some giddiness, headache, or slight pain in the back. As a rule, there is little or no elevation of temperature, but in cases with a profuse rash there may be very considerable pyrexia.

In many, perhaps most, cases of chicken-pox there are no prodromata.

The rash appears *on the first day* in the form of small red papules.

The spots appear on the chest, back, and limbs. There may be a few on the face, and they frequently occur on the scalp, where they are more easily felt by the patient than seen by the physician. The spots are converted into vesicles in the course of a day or so, and then present the appearance of little blisters or sacs, with clear watery contents, and each is surrounded by a red areola.

It is worthy of note that the eruption of varicella occurs on the mucous surfaces as well as on the skin. It is found in the mouth, on the throat, etc. The contents of the vesicles become milky or opalescent, and in a few days more are converted into little scabs or crusts. When the drying stage commences there may be flattening or dimpling of the top of the vesicles. On removal, these crusts leave behind them marks or stains which subsequently disappear, but in some cases permanent pits or cicatrices are left. This latter result is, however, exceptional. It is most likely to result from premature removal of the scabs by scratching, and the consequent formation of small ulcers which, on healing, leave scars. It must be admitted, however, that in some cases pits are left where there has been no mechanical irritation. In such cases the bed of the vesicle, being deep, has involved the true skin. It has been the custom to state that the vesicle of varicella is unilocular, while that of variola is multilocular, and this has been regarded as one of the means of distinguishing between the eruption of chicken-pox and that of smallpox. This view is not tenable in the light of our present knowledge.

The statements of Unna (*Histopathology of the Diseases of the Skin*, Walker's translation) on this point are so clear and precise that one cannot do better than quote his exact words: "I take the description of the eruption from a characteristic 'chicken-pox' which I excised from an eight-year-old boy on the second day of its existence. I choose this because it gives the most perfect contrast to the centrally depressed variola vesicle, for in it the most central point of the surface is the most elevated. It has a tent form. The lateral walls rise obliquely from a broad base towards the centre of the covering, which is formed by a few stretched, horny scales. From these, cellular partitions radiate downwards as in smallpox. The chicken-pock is consequently divided like the smallpock, but the point where the septa join lies here, not in the centre of the base of the pock, but on the covering." And again, at p. 639 (under *Variola*), he says: "The main character which distinguishes the vesicular formation in true smallpox (variola, varioloid) from that in chicken-pox consists, on the one hand, in the slow growth, on the other, in the prompt addition of suppuration to the fibrinoid degeneration, which obscures the effects of the latter."

If the reader wishes a detailed and particular account of the pathological changes which bring about the formation of the varicellar eruption, he is referred to the volume just quoted, in which will also be found plates illustrating the points in question. The eruption of varicella may continue to come out in *successive crops* for a considerable time. The fresh crops may be accompanied by elevation of temperature.

There are no *complications* to be noted in varicella, and there are, perhaps, no real *sequelæ* to be dealt with. It is true, however, that in dirty and unhealthy children the sores which originate in the picking and scratching of the vesicles may give rise to troublesome consequences.

Sir Dyce Duckworth, in his Lumleian *Lectures on the Sequelæ of Disease*, says: "Varicella has no true sequels in respect of changes which may occur in the rash. Prurigo may result, so-called varicella prurigo (Hutchinson). Ecthymatous sores resembling pemphigus are sometimes met with. The vesicles may become

gangrenous in delicate or tuberculous children, and the result is probably due to the supervention of extraneous microbic infection, and not to the influence directly of varicella poison. Like the eruptive fevers, varicella may be followed by pertussis, a lowered condition of health paving the way for fresh infection, with increased susceptibility to it."

With regard to the gangrenous affection, it may be said that it consists in the formation of sloughs which, on separation, leave deep ulcers. The ulcers may coalesce. When this morbid action is extensive there is pyrexia, while "lung complications, tubercular, pyæmic, or inflammatory, are very frequent, and determine or hurry on the fatal issue" (H. Radcliffe Crocker).

For illustrations of pruriginous impetigo (following varicella), vide *Atlas of Skin Diseases*, New Syd. Soc., plate xxxii.

DIAGNOSIS.—The diagnosis of chicken-pox is, as a rule, easy—*especially when the eruption is in the stage of clear watery blebs.*

The differential diagnosis between *smallpox* and *chicken-pox* is a matter of very great importance.

In the great majority of cases the distinction between the two diseases is made without any difficulty.

It must be honestly confessed, however, that (if the case is seen in the crusted stage, and with only a few spots, and without any clear history) there may be great doubt between chicken-pox and a mild or abortive attack of modified smallpox.

Some slight cases of varioloid have little constitutional disturbance, and only a few spots—spots which may be easily overlooked, or, if seen in the crusted stage, may be mistaken for chicken-pox. In such exceptional and doubtful cases much assistance will be obtained if the patient can be *associated* with previous cases of chicken-pox on the one hand, or smallpox on the other.

The following scheme presents in contrast the salient features of variola *versus* varicella, but, like many other elaborations of the study, it may sometimes prove unsatisfactory by the bedside:—

SMALLPOX	CHICKEN-POX
Prodromata severe	*Prodromata slight or absent*
Heavy sickness, severe pain in back, vomiting.	*Perhaps* slight headache, pain in back, shivering.
Spots come out on 3rd day. Spots come out in progressive order from above downwards—face, hands and arms, trunk, legs, feet.	Spots come out on 1st day. Spots come out in successive crops, the new spots being widely distributed.
Temperature: three days' pyrexia at onset, and pyrexia again when suppuration occurs, about the ninth day (called the secondary, or suppurative fever).	Temperature not high as a rule. But pyrexia accompanies each fresh crop of spots if the eruption is profuse.
Liable to have very grave complications and sequelæ.	Neither complications nor sequelæ to be reckoned with, as a rule.
The centre of the vesicle is depressed.	The centre of the vesicle is the highest point.
Eruption apt to leave pits, often deep and permanent.	Pitting occasional, and slight as a rule.

Dr Wanklyn deals with the diagnosis between variola and varicella in a very able way: he does not attach much weight to "history"; the terms "shotty" and "umbilication" he would dismiss as unreliable indications. He attaches great importance to *distribution* of the rash, which in smallpox abounds on face, wrists, hands, and feet frequently; while in chicken-pox the trunk is the favourite field. In some cases of varicella there may be abundant eruption on the face, but not on the forearms and hands or lower extremities. Dr. Wanklyn's article is worthy of careful attention.

Chicken-pox and smallpox are separate and distinct diseases.

Chicken-pox gives no protection against smallpox, and smallpox gives none against chicken-pox.

Vaccination gives no protection against varicella.

It is sometimes stated (as a point of differential diagnosis between variola and varicella) that while smallpox spots are found on mouth or palate or throat, chicken-pox spots are never found in those situations. The statement is not correct. The varicella eruption may also be found in these localities. A difficulty in diagnosis may arise between chicken-pox and some skin diseases, such as eczema pustulosum and impetigo. As regards impetigo, it is stated that the eruption may be distinguished by its pustular character, the absence of red areola, and the fact that the rash has not the wide distribution of chicken-pox.

Unna (p. 552) describes a *varicelliform syphilide*, which he says "is very rare."

Dr. R. S. Thomson has described a disease occurring in Lascars which in some points resembled smallpox and in others chicken-pox. He concludes that it is neither, but a separate disease. He refers to similar cases recorded in Jamaica more than thirty years since by Dr. Isett Anderson. The subject is one of much interest.

It is to be borne in mind that varicella may manifest itself at the same time as measles or scarlet fever.

Dr. Webber records a case of "concurrent

eruption of measles and chicken-pox" in *Lancet*, 5th Nov. 1898.

Trousseau states, on the authority of Dr. Delpech, that a child had smallpox and chicken-pox together.

PROGNOSIS is good. The disease is not a dangerous one. It is popularly regarded as a very trifling ailment.

It is to be borne in mind, however, that varicella is sometimes attended by pretty severe symptoms.

The writer has seen children with marked pyrexia, pronounced constitutional disturbance, and in misery.

And in delicate and unhealthy children we must not lose sight of the possibility of further trouble in the shape of prurigo—ulceration and gangrenous sloughing.

The PATHOLOGY of this disease is very meagre. As death from uncomplicated chicken-pox is practically unknown, opportunities for investigation do not occur.

TREATMENT.—The treatment of an ordinary case may be comprised in warmth, slop diet, attention to the bowels, and, perhaps, the administration of a diaphoretic.

RELATIONS TO PUBLIC HEALTH. PREVENTION.—As varicella is not a fatal disease, it might be considered unworthy of the attention of sanitary authorities. This, however, is far from being a correct view. All cases of chicken-pox should be made known to the medical officer of health, because *a mild case* of smallpox may be mistaken for chicken-pox, and so escape the control which is necessary for the public safety. This danger is not an imaginary, but a real one. "It is stated that the Chicago case, which was the starting-point in Montreal of the epidemic of 1885, was regarded as varicella, and not isolated. If so, the mistake was one which led to one of the most fatal of modern outbreaks of the disease" (Osler). Cases of varioloid, if seen in the crusted stage, and with only a few spots, are liable to be passed over as varicella, especially if the patients deny having felt ill, and cannot be *associated* with smallpox cases.

Varicella may prevail in epidemic form, and, although not dangerous, may give rise to much trouble and disorganisation in schools, hospitals, nursing homes, etc.

The writer can recall annoying experiences from outbreaks of the disease occurring in the wards of a fever hospital. When cases cropped up in measles, scarlet fever, or whopping-cough wards, no end of trouble and worry was the result. The children who took the disease in hospital could not be sent home till all the crusts were off, and those who did not take it had to be detained for a fortnight at least (incubation period) before being dismissed.

And how did such outbreaks originate? A child admitted with measles or scarlet fever might have a few little scabs among the hair which unfortunately were not immediately recognised as chicken-pox crusts, the result being the infection of the ward, with fresh cases cropping up in a tantalising fashion one or two at a time with a fortnight's interval. This might go on for months, till all the susceptible children had taken the chicken-pox. The disease seemed to increase in severity in the successive cases, as if the virus gained in intensity.

The writer has also seen the work of a surgical nursing home totally disorganised, and heavy expenditure incurred, all through the occurrence of chicken-pox in a patient who was admitted to the institution in the incubation stage.

PREVENTION.—The measures taken to prevent the spread of smallpox are applicable to varicella.

The patient should be isolated in a bare room with a special nurse.

When all the crusts have separated he should have several warm baths, and get a complete suit of fresh clothing before regaining his liberty. Crusts among the hair must not be overlooked.

The sick-room should be washed down and repapered, while all clothing and bedding should be disinfected, either by steam or boiling. While the patient is under treatment it is a good plan to put all soiled linen at once under a disinfectant solution (*e.g.* carbolic acid and water).

Varicocele.—Dilatation of the veins of the spermatic cord causing a swelling in the scrotum; also, swelling of the veins of the broad ligament in the female. *See* PELVIS, DISEASES OF CELLULAR TISSUE (*Tumours of Broad Ligament*); HERNIA (*Inguinal Diagnosis*); SCROTUM AND TESTICLE, DISEASES OF (*Varicocele*).

Varicose.—In a state of varix or varicosity, having a dilated and tortuous appearance, *e.g.* varicose veins; or, having an origin in a varicose vein, *e.g.* a varicose ulcer. *See* CONSTIPATION (*Symptoms*); FILARIASIS (*Varicose Inguinal Glands*); GOUT (*Circulatory System*); HÆMATEMESIS (*Causes*); LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism, Causes*); PREGNANCY, PHYSIOLOGY (*External Genitals, Varicosity*); PREGNANCY, CAUSES OF DEATH; ULCERS AND ULCERATION (*Due to Imperfect Circulation*); VEINS, DISEASES OF (*Varicose Veins*).

Variola. *See* SMALLPOX and *Cross References.*

Varioloid.—Resembling smallpox, or smallpox modified by vaccination or inoculation; a mild form of smallpox. *See* SMALLPOX (*Clinical Variations, Varioloid*).

"Varium."—The name given by Burroughs Wellcome & Co. to their preparation of ovarian substance; it is recommended, in 5-grain doses, in amenorrhœa and dysmenorrhœa and in troubles at the menopause (natural or induced).

Varix.—A varicose swelling or dilated and tortuous vein. *See* Aneurysm (*Aneurismal Varix*); Conjunctiva, Diseases of (*Malformations and Congenital Anomalies, Lymphatic Varix*); Scrotum and Testicle, Diseases of (*Lymph Scrotum or Varix Lymphaticus*).

Varnish-Making.—An offensive trade, as defined by the Public Health Act, 1875, and the Public Health (Scotland) Act of 1897.

Varolii, Pons. *See* Pons and *Cross References.*

Varus.—The deformity in which there is inversion of the foot. *See* Deformities (*Hallux Varus, Talipes Varus, Genu Varum*).

Vas.—A vessel or tube (plural, *vasa*); *e.g.* the *vas deferens* (excretory duct of the testicle), *vasa afferentia* (lymphatics going to a lymphatic gland), *vasa vasorum* (vessels supplying the walls of the larger blood-vessels). *See* Scrotum and Testicle, Diseases of; Arteries, Diseases of; Veins, Diseases of.

Vascularisation.—The act of becoming rich in blood-vessels or of developing vessels.

Vasectomy.—Resection of the vas deferens, as in diseases of the prostate gland. Prostate Gland (*Hypertrophy, Operative Treatment*).

Vaseline. *See* Paraffin (*Paraffinum Molle*).

Vasicin.—An alkaloid obtained from *Adhatoda vasica*, the leaves of which are official in the Indian and Colonial Addendum to the B.P. for 1898; it has been used in bronchitis (like senega) and in dyspepsia, and as an insecticide.

Vasiform.—Resembling a vessel, tubular; *e.g.* vasiform tissue, or tissue largely composed of vessels.

Vaso-.—In compound words *vaso-* has the meaning of vascular or belonging to a vessel; e.g. *vaso-constrictor* (causing contraction of a vessel), *vaso-dilator* (causing relaxation of a vessel), *vaso-formative* or *vaso-factive* (forming vessels), *vaso-inhibitory* (inhibiting vaso-motor action), *vaso-motor* (causing constriction or dilatation of vessels), *vasoparesis* (paresis affecting vaso-motor nerves). *See* Angina Pectoris (*Varieties, Vaso-motor Angina*); Bed-Sores (*Clinical Varieties, Acute Bed-Sore, Causes*); Erythema (*Pathology and Etiology*); Headache (*Migraine*); Nerves, Neuralgia (*Etiology*); Physiology, Circulation (*Blood-Pressure, Regulation of*); Syringomyelia (*Symptoms*); Thyroid Gland, Medical (*Exophthalmic Goitre, Symptoms*).

Vasogen.—Oxygenated vaseline.

Vasotribe.—An instrument for controlling hæmorrhage by crushing the vessels; an angiotribe.

Vastus.—Large, widespread, *e.g.* the vastus muscle which forms part of the quadriceps extensor cruris.

Vater's Ampulla.—The cavity into which the bile and pancreatic ducts open before their contents pass through the bile papilla into the intestine; a small vertical fold of mucous membrane in the ampulla is called *Vater's Fold.*

Vater's Corpuscles.—Pacinian bodies. *See* Skin, Anatomy and Physiology (*Nerves, Terminal Corpuscles*).

Veal. *See* Invalid Feeding (*Veal Panada*).

Vecordia.—Insanity or idiocy.

Vectis.—An obstetrical instrument resembling the single blade of a pair of forceps; a lever. *See* Labour, Obstetric Operations (*Vectis*).

Vector.—A suggested obstetric instrument to cause dilatation of the vagina in delayed labour and so prepare for the passage of the child's head.

Vegetables. *See* Diet (*Vegetable Foods*); Invalid Feeding (*Vegetables*); Invalid Feeding (*Diet in Old Age and in Diabetes*); Scurvy in Adults (*Treatment*).

Vegetarian Diet.—A diet consisting almost wholly of vegetable food. *See* Gout (*Dietetic Treatment*).

Vegetations.—Growths or excrescences found in hollow organs or in body canals; *e.g.* the fibrinous projections found on the valves of the heart in disease, adenoid growths of the pharynx, and urethral papillomata. *See* Heart, Myocardium and Endocardium (*Malignant Endocarditis, Pathology*).

Vehicle.—That part of a prescription which serves as the medium of administration of the active drug or drugs; an excipient, dry or moist. *See* Prescribing (*Prescriptions, Component Parts*).

Veins, Diseases of.

Anatomy and Physiology—
1. *Structure of Veins* . . . 421
2. *Venous Systems* 421
3. *Venous Flow* 421

Thrombosis and Phlebitis—
1. *Etiology of Thrombosis* . . 421
2. *Pathology* 422
3. *Phlebitis. Etiology and Pathology* 423
4. *Clinical Types* 423
5. *Treatment* 424
6. *Pulmonary Embolism* . . . 424
7. *Septic Phlebitis* 424

VARICOSE VEINS—
1. *Pathology* 425
2. *Etiology* 425
3. *Symptoms and Complications* . 425
4. *Treatment* 426
ENTRANCE OF AIR INTO VEINS . . 427

See also AXILLA (*Injuries, Axillary Vein*); BONE, DISEASES OF (*Suppurative Osteomyelitis, Symptoms, Veins*); BRAIN, PHYSIOLOGY OF (*Venous Circulation*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Thrombosis*); BRAIN, SURGERY OF (*Thrombosis of Venous Sinuses*); HÆMATEMESIS (*Causes, Varicose Veins*); HEART, PHYSIOLOGY OF (*Venous Sinuses*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Examination, Venous Pulsation*); LABOUR, POST-PARTUM HÆMORRHAGE (*Etiology*); LUNG, TUBERCULOSIS OF (*Complications, Venous Thrombosis*); LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism, Causes*); PREGNANCY, AFFECTIONS AND COMPLICATIONS (*Thrombosis*); PUERPERIUM, PHYSIOLOGY (*Circulatory System*); SYPHILIS (*Tertiary, Vascular System, Veins*).

1. ANATOMY AND PHYSIOLOGY

THE wall of a vein, like that of an artery, is composed of muscular and elastic tissue, lined internally by a pavement membrane, and strengthened externally by a layer of connective tissue.

The inner coat (intima) consists of a layer of flattened cells which is continuous with the lining walls of capillaries, and is supported by a small amount of elastic tissue which seldom forms a complete membrane. The middle coat, which contains much less muscular tissue than that of an artery, consists of bundles of white connective tissue, some elastic tissue, and bundles of circular muscular fibres. These circular muscular fibres are almost entirely absent in the superior vena cava and in the upper part of the inferior vena cava, and in some veins there are some longitudinal muscular fibres. The outer coat is composed of fibrous tissue stronger than the corresponding coat in arteries.

Valves.— Many veins possess valves, which are semilunar folds of the internal coat, containing a little fibrous tissue, and each valve consists usually of two cusps opposite one another.

There are no valves in most of the veins of the viscera, nor in those within the cranium or vertebral canal and bones, nor in the umbilical vein.

There is no muscular tissue in the walls of the veins of the pia mater, brain or cord, bones, nor in the venous sinuses of the dura mater or placenta. The walls contain vasa vasorum, nerves, and lymphatics.

Venous Systems.—Clinically it is useful to recognise certain great divisions of the venous system, along any one of which the effects of back pressure from an impaired heart may be evident, or which may be affected by pressure.

Such are—

(1) Superior vena cava system.
(2) Inferior „ „ „
(3) Pulmonary venous system.
(4) Portal „ „

The two former possess valves on their branches, the first from the heart outwards being found an inch from the lower end of the internal jugular vein, in the subclavian vein beyond the entrance of the external jugular, and in the external iliac veins, there being no valves in the internal iliac veins.

The two latter, the hepatic veins opening into the inferior vena cava, and the pulmonary veins opening into the left auricle, have no valves, and therefore it is along these systems that the effects of back pressure from the heart are first evident, from the right and left auricles respectively.

The coronary system possesses valves which may be incompetent.

Venous Flow. — Alterations in the venous flow are so frequent and so evident that it will be well to point out the causes to which this flow is due. They are the *vis a tergo* of the circulation, the aspirating effect of the heart, and muscular action compressing those veins which have valves.

Venous pulse may be observed when the arterioles are so dilated that the arterial pulse passes through the capillaries to the veins, and in the superior and inferior vena cava near their entrance to the heart, due to variations in the pressure in the right auricle, as in tricuspid incompetence, and also to respiratory movements.

THROMBOSIS AND PHLEBITIS

Thrombosis (θρόμβος, a clot); Synon.: Fr. *Thrombose*; Ger. *Thrombose*.

Phlebitis; Synon.: Fr. *Phlébite*; Ger. *Venenentzündung*.

Definition.—Thrombosis is the formation of a clot or coagulum of fibrin in the heart or vessels during life. Phlebitis is inflammation of the coats of a vein.

These two conditions are closely related to one another, and clinically are usually found together. When a thrombus forms in a vein the irritation produced by its presence sets up a phlebitis, and phlebitis is accompanied by thrombosis of the vessel when the inflammation spreads to the inner coat of the vein, since the inflammation causes coagulation of the contents of the vessel.

Etiology of Thrombosis.—The coagulation of blood, consisting in the formation of fibrin, is brought about by the action upon the fibrinogen of the serum of a fibrin ferment produced by the destruction of leucocytes and from blood

platelets. Clinically it is essentially the result of stagnation of the blood-stream, together with some other cause which can bring about the formation of the fibrin ferment.

Such causes may be found in the vessel wall or in the blood itself.

Among the first any injury or contusion, or laceration, or the exposure of the vessel in a wound, or any inflammatory or degenerative change in the wall of the vessel, may cause thrombosis; but the factors in this class are of less importance from the point of view of treatment than those of the second class which include the cause usually determinable in an ordinary and uncomplicated case of thrombosis.

The hæmic conditions may be described generally as any condition which affects the physiological integrity of the leucocytes, as thereby there is a tendency for the liberation of the fibrin ferment. We may include in this group all cases from those in which the patient is suffering from an actual septicæmia or pyæmia, to even that less virulent form which can only be described as a "gouty" condition, and including those in which there is a general condition of exhaustion, and in this last variety there is an added tendency from the slowing of the blood-stream. The tendency in any of these cases is increased by any local or general interference with the circulation, such as a weak heart, loss of the elasticity of the arteries, by the compression of a tourniquet or a tight bandage, or by pressure from a tumour.

The combined action of these two causes is exemplified in the thrombosis which occurs in the later stages of exhausting diseases, such as phthisis, typhoid fever, or prolonged suppurative conditions. This effect of slowness of the blood-stream is in part due to an alteration in the state of the endothelial cells, which are ill-nourished when the blood that bathes them is not renewed with the usual rapidity.

Toxic materials or parasites in the blood-stream frequently determine thrombosis. In malaria the parasites actually plug the small vessels of the brain and spinal cord, and in pyæmia and septicæmia the organisms have a direct coagulating effect on the blood.

Pathology.—Following upon such conditions a clot may form in a vein, and usually extends for some distance along the vessel, or at least to the point where the vein joins another one.

At this point the thrombus is liable to be broken, and part to be carried off in the blood-stream, forming an embolus. The thrombus adheres to the vessel wall, and if the circulation be active the red corpuscles and the colouring matter are washed out, and the clot is of a pale colour or white thrombus, while if the colouring matter remains a red thrombus is the result.

The next stage in the process is that an inflammatory change is produced in the vessel wall, and a series of changes ensues, the same as those found in an artery (*q.v.*), with the result that the thrombus becomes organised, and thereby converted into a fibrous cord which may permanently block the vessel, or may in part be absorbed, with the result that the lumen is restored to a certain extent.

Other changes which may subsequently occur are—

1. Calcification, consisting in a deposit of lime salts in the thrombus, and resulting in the formation of a phlebolith, a small round and hard nodule frequently met with in varicose veins.

Phleboliths are formed from the salts present in the blood, and consist, like bone, mainly of phosphate of lime, with the sulphates of lime and potash. They are commonest about the base of the bladder in varicosities of the prostatic plexus and in nævi in which thrombosis has occurred.

2. Simple disintegration may apparently occur, a change in which the thrombus is gradually broken up, and the fragments carried off in the general circulation without any bad effects.

3. Embolism and septic or infective softening of the thrombus are serious complications, the occurrence of which entirely alters the character of an apparently simple case, and which must always be borne in mind.

Septic or infective softening is due to the action of micro-organisms, which may have formed the original nucleus of the thrombus or may reach the clot when it has been formed from the vessel wall, as in cases of septic phlebitis.

As the result of their action, the thrombus is broken down into a soft pulpy mass, around which further thrombosis may occur, and so prevent the access of the infective material to the general circulation. If the softening proceeds with greater rapidity than this protective clotting, the broken-down particles enter the circulating blood, and are carried to the right side of the heart and to the lungs, producing secondary thrombi there and in other parts, which again break down and form abscesses.

Effects of Thrombosis.—These depend upon the situation and size of the affected vessel. In small veins the difficulty in the return circulation is soon overcome by the blood finding its way by collateral channels; but when there is important interference, the result is œdema of the distal part of the limb, which may be severe and persistent, and even lead to moist gangrene of the limb. In the lower limb the œdema usually commences about the ankle, and spreads upwards to the point of obstruction. As the œdema increases, the veins become obscured in it, and may be felt as gutters or grooves in a solid mass of tissue. There is at first a sensation of stiffness or tightness in the part, and

later weakness of the muscles and heaviness and pain.

The symptoms are practically indistinguishable from those of the phlebitis which accompanies it.

Recovery may be brought about by the enlargement of collateral channels or by the canalisation of the thrombus and restoration of the original channels, but it is a slow process when a large vessel is involved.

Embolism.—The dislodgment of a thrombus may occur either from mechanical causes, such as sudden movements or undue fingering of the part, or from softening and disintegration of the clot from septic changes in it. In the former case the subsequent symptoms are due to the mechanical interference with the circulation of the part in which the embolus lodges, while in the latter the infective nature of the embolus leads to the formation of widely disseminated foci of suppuration and to a condition of pyæmia. Following the course of the blood-stream, the embolus is carried first to the right side of the heart, and thence to the pulmonary artery, where it may be arrested and produce a pulmonary embolism, and such an occurrence is a frequent cause of sudden death. If the clot be smaller, and able to pass through the capillaries, it may pass to any other organ, and as such small emboli are often of a septic character, secondary abscesses are produced in the liver, kidneys, or joints, or in fact in any tissue of the body.

Phlebitis.—Etiology.—Phlebitis may arise in those conditions in which the vein is exposed to injury or irritation directly, as in wounds, burns, etc.; but the phlebitis in such cases is, as a rule, slight, and does not attract special attention. Much more important are those forms in which the phlebitis arises apparently without any external local cause, and is accompanied by thrombosis.

Much discussion has taken place on the question as to whether the thrombosis is the primary condition or is secondary to the phlebitis. It is known by experiment that mere stasis of blood in a vessel, if the vessel is healthy, does not produce coagulation, and it has therefore been supposed that the phlebitis is the primary condition. There is no doubt, however, that the walls of the veins are occasionally diseased, and exhibit appearances similar to those found in atheromatous arteries, with degenerative changes (endophlebitis obliterans), and in such conditions it is possible that there may have been no acute affection of the vessel wall previously. Atheroma may be more frequently present as a cause of phlebitis than is suspected, since thrombosis will more readily occur in a vein that is diseased than in a corresponding artery, as the flow of blood is slower. The phlebitis is, however, usually the primary condition, and is due to a variety of causes, mostly of a constitutional nature, such as gout, anæmia, convalescence from many illnesses, especially influenza and typhoid fever. A varicose condition of the veins always is a predisposing factor in the etiology.

The inflammation may attack one or other of the coats of a vein, but the walls are so thin that it is impossible to recognise the different forms clinically.

The inflammation may be of a simple plastic kind, or septic in character, the infection in such cases usually affecting the vein from without, and a chronic form of phlebitis occurs, frequently in syphilitic subjects.

Clinical Features. — Phlebitis is specially liable to occur in varicose veins, especially in the dilated veins of the rectum or lower limb. The inflammatory changes are accompanied by the classic signs of pain, redness, heat, and swelling, and the impaired function of the vessel consequent on the thrombosis produces characteristic swelling and œdema in the lower parts of a limb.

The pain may be dull and aching or sharp and shooting in character, and the swollen, red appearance along the line of the vessels is typical when a superficial vein is affected. There is tenderness, and on feeling the part a firm cord can be felt. The œdema is very marked in some forms, especially the gouty, but the free anastomosis between superficial veins may render it only slight. The firm cords present swellings at the position of the valves, but the parts must be examined with great caution.

Clinical Groups.—1. Phlegmasia alba dolens, occurring notably in childbed, but also occurring in convalescence from typhoid fever, is shown by a rise of temperature to about 102° for some days, by severe pain, and by a characteristic œdema of the limb.

2. Anæmic or chlorotic form, forms a clinical entity, though its etiology is obscure, and is accompanied by the usual phenomena of the disease. Both lower limbs are frequently affected, and there is a rise of temperature lasting about a week, with considerable pain and œdema of the limbs.

3. Gouty phlebitis is a common form, occurring in gouty subjects frequently apart from an acute attack. It frequently affects both limbs, and shows a tendency to shift from one vein to another. There is aching pain and tenderness which lasts for days, and there is considerable œdema of the parts below, the swelling being very tense in character. Such forms are very liable to embolism.

4. During convalescence from certain diseases phlebitis may appear, and the diseases after which it is most liable to occur are typhoid fever, influenza, and pneumonia. After typhoid, phlebitis usually occurs in a mild form, while the post-influenzal form is more acute and severe.

5. In tuberculosis a tubercular form of phlebitis is occasionally seen, caused, apparently, by the deposit of the organism on the vessel wall from the blood stream; and an acute phlebitis may be brought about in the same way in the course of septicæmia.

6. A chronic form of phlebitis not infrequently occurs in individuals affected with syphilis, and is characterised by thickening of the vessels, and by a certain amount of pain and slight tenderness.

Treatment.—In all forms of the disease the patient must be confined to bed, and the limb should be elevated, and it is advisable to immobilise it by the application of a light splint and a bandage, or by lateral sandbags. Heat relieves the pain, and the part should therefore be kept warm. Some local application is advisable, and an unguent, consisting of equal parts of glycerine and extract of belladonna, may be smeared on without friction, and the limb then covered with cotton-wool and a bandage.

The general treatment consists in that applicable to any form of acute inflammation, such as a saline purge and a light diet for cases where the circulation is good. When the thrombosis and phlebitis are consequent upon a general impairment of vitality, a nourishing diet, with tonics and cod-liver oil, is indicated. The rule is to treat constitutionally the general condition, and the frequency of a gouty element being present should not be lost sight of, and such cases treated with colchicum, alkalies, and iodide of potash.

Should the phlebitis show a tendency to spread upwards along the course of a superficial vein and to affect larger vessels, the danger of embolism is increased, and it may be advisable to excise a portion of the vein and thus to interrupt the conducting medium.

Pulmonary Embolism. — Whenever there is recent thrombosis, the possibility of part of the clot being detached, carried to the heart, and thence to the lungs, must never be forgotten. Blocking of the smaller branches of the pulmonary artery is often the cause of pulmonary apoplexy, lobular pneumonia, circumscribed abscesses, patches of gangrene, and the like, but it is in the obstruction of the larger divisions that the chief source of danger is found. Complications of this kind occur with a tragic suddenness; for the patient may have been in fair health, suffering only from a slightly painful swelling of some varicose veins in the leg, and suddenly present symptoms of the most severe interference with the circulation and respiration, only gradually recovered from, or terminating in sudden death. When a branch of the pulmonary artery is blocked, the circulation is at once cut off from the area of lung supplied by it, since the branches of the pulmonary artery form only capillary anastomoses with one another, and the respiratory function of the part is at once lost.

It has been disputed whether death is due to syncope or to asphyxia, but it is certain that the sudden obstruction of the pulmonary artery causes shock or collapse, and that the patient sometimes dies of this shock within a few seconds.

The symptoms of pulmonic obstruction are by no means typical, and are often indistinguishable from those of angina, or rupture of an aneurysm or of the heart itself into the pericardial cavity. It must also be borne in mind that many of the recorded cases of sudden death from pulmonary embolism are cases in which the only foundations for the diagnosis were—suddenness of death, possibly from syncope, and the discovery after death of an ordinary fibrinous clot in the right ventricle, prolonged thence into the pulmonary artery and its branches, a clot originating in the spot in which it was found, and the consequence of dying, not the cause of death.

The symptoms of the occurrence of a pulmonary embolism are sudden pain at the heart, with distress and faintness, rapid and irregular action of the heart, and gasping breathing, with distress and lividity. There are not usually distinct physical signs, and the treatment consists in the administration of ammonia in frequent doses, as this substance has the double use of stimulating the circulation, and is said also to have the property of dissolving thrombi.

Suppurative or septic phlebitis, brought about by the invasion of organisms, is often due to infection from some focus outside the vessel, and therefore may occur in any septic wound, or with any suppurative condition, especially when there is tension. It is well typified in the condition sometimes found in the lateral sinus of the cranium and the internal jugular vein in connection with middle ear disease (*q.v.*).

When the thrombus undergoes infective softening the vein wall and the tissues round it become the seat of an intense inflammation, the signs of which are obvious in the case of the superficial veins and in the deep veins, such as the internal jugular; fulness and tenderness along its course may be discovered.

The condition also occurs in connection with septic inflammations in cellular tissue, especially when the tissue is dense, as in the subcutaneous tissue of the face, and consequently there is tension. Such conditions as "facial carbuncle" are liable to produce a septic thrombosis of the branches of the facial vein, and the gravity of the condition is increased by the liability of the infection of the intracranial veins and sinuses by spread of the thrombosis by direct continuity.

The local symptoms are those of an acute phlebitis, with great redness and swelling, and with severe general symptoms of a high fever. The prognosis is very grave, and everything depends upon limiting the spread of the disease. The condition must be treated on surgical lines,

by laying open the inflamed vein, removing the infected thrombus, and thoroughly cleansing the cavity and stuffing lightly with iodoform gauze. In advancing cases the vein should be exposed on the cardiac side, tied and divided, and a current of fluid directed along the channel from the opening to the seat of thrombosis, as in lateral sinus thrombosis in middle ear disease (*q.v.*).

Varicose Veins

Varicose veins; Synon.: *Varix, Phlebectasis;* Fr. *Varice;* Ger. *Krampfader.*

The condition indicated by this term is one of permanent over-distension of the walls of veins, with lengthening and tortuosity of the vessels, accompanied by changes in the walls of the veins. Such a condition occurring in the veins of the scrotum is known as a varicocele (*vide* "Scrotum"), and it is most common in that site at the anus, and in the veins of the lower limb, but it may occur in any set of veins from obstruction to the return circulation.

The condition is only recognisable when the superficial veins are affected, but deep veins may be varicose also, especially in the venæ comites of the veins of the leg.

The etiology of the condition is not quite clear in all cases, and many causes have been suggested.

Before considering the probable causes that bring about a varicose condition in that commonest region, the lower limb, it is necessary to understand the nature of the pathological changes that occur in the vessels.

Either of the saphena veins may be affected, or both, and the distension may only be noticeable over a small part of the vessel. Frequently one sees a bunch of enlarged veins and the formation of a distinct tumour; in other cases no stem of a single vein is affected, and a long sinuous prominence is seen.

The veins are thickened as well as dilated, and they do not collapse when divided; the thickening is due to an increase of the fibrous tissue in the wall, and the vessel is elongated, as there is a hyperplasia in the long axis as well as in the transverse.

The valves, at first thickened, become incompetent, and may degenerate into fibrous cords.

Large sacculated dilatations may form at the bends of veins, or where a superficial one joins a deep vein. The essential change is apparently an over-distension of the vessel wall, and the wall once over-distended undergoes the fibrous changes which are common to tissues which are over-exerted.

One is often at a loss to discover a reason for the primary over-distension.

Dilatation of the prostatic plexus is common in elderly persons, and of the rectal and anal veins in persons who have congested liver or who suffer from constipation.

A general impediment to the venous return, such as chronic heart or lung disease, causes a general over-distension of the veins, and those of the lower extremity, being the longest and having the weight of the column of blood to bear, will suffer first.

Local obstructions, such as an overloaded colon or an enlarged uterus, will tell on the veins of the lower limb; and muscular strain, such as severe efforts in bicycling or running, may determine varicosity of the veins in healthy young adults.

Occurring in the veins of the upper limb, it is usually due to pressure on the main vessels from a tumour in the axilla or in the superior mediastinum, or to an arterio-venous aneurysm.

The veins of the anterior abdominal wall become highly varicose in cases of obstruction to the inferior vena cava, and a similar condition is found in the veins of the alimentary canal, especially of the stomach, in hepatic cirrhosis.

Occurring in the lower limbs it is frequently due to obstructive causes, such as a loaded rectum, a pregnant or enlarged uterus, the wearing of tight garters, and excessive muscular effort; but all these factors may be present and yet no varicose veins result; the importance to be attached to them is somewhat doubtful.

Certain individuals appear to be more liable, and a tendency of this kind may be inherited; and deficient support of the veins is also a cause, especially in the lower limb, where the weight of a long column of blood has to be supported by the vessel wall, if for any reason the valves be incompetent. The gradual distension of the veins leads to this incompetence, and thus a *circulus vitiosus* of vascular dilatation and valvular incompetence may be set up, and lead to progressive spread of the varix.

The signs and symptoms are quite distinct when the parts are examined, as the enlarged veins stand out in the form of rounded, tense, tortuous cords, often with local areas of dilatation amounting to the formation of a tumour as large as a walnut. The internal saphenous vein and its tributaries are often affected, and a mass of tortuous vessels is found on the inner side of the knee.

Changes are seen in the cutaneous venous radicles, which are injected, and there may be œdema of the lower part of the limb. The skin appears glazed, and as the nutrition of the limb suffers some complications are common.

Cutaneous conditions of the nature of eczema, and a diminished power of resistance to injury or irritation, often lead to ulceration, and there may be rupture of the veins, leading to hæmorrhage, which is always severe and may even be fatal, as the bleeding is aided by the whole weight of the column of blood in the veins between the point of rupture and the main abdominal vessels. The patient complains of tired, aching feeling, or even of acute pain, of

frequent cramps, and is disabled from prolonged walking exercise. The pains may be mistaken for rheumatic conditions unless an inspection of the limb be made. The examination of the part is to be completed by getting the patient to lie down with the limb elevated till the distension has subsided; and then, putting the thumb upon the termination of the internal saphenous vein and pressing firmly upon it, the patient is asked to stand up. The pressure on the vein removes the weight of the column of blood in the upper vessels, and as it usually renders the venous distension to be only slight, we can judge of the effect likely to be produced by ligature or removal of this part of the vein.

Treatment.—1. *Palliative.* 2. *Radical.*—Palliative treatment consists in the use of some appliance which gives artificial support to the veins, and it is often found to give great relief to the symptoms, and to lead to improvement. The support may be a well-fitting elastic or silk stocking or a bandage, applied to the limb from below upwards.

A domette or cheese-cloth bandage, or one of elastic webbing, applied by the figure-of-eight, or "Watson's" method, is the most suitable, and it should not be applied very firmly, as subsequent swelling of the limb may cause injurious constriction. The patient should sleep with the lower end of the bed raised, and apply the stocking or bandage before getting out of bed, and not remove it until after he has gone back to bed.

Massage removes the tired, aching feeling, and improves the nutrition of the limb, and in addition a moderate amount of walking exercise is advisable.

Intercurrent attacks of phlebitis are fairly common, and may lead to a cure from the accompanying thrombosis, and these attacks are to be treated on the lines previously indicated.

The radical treatment is to be advised when there is much pain, when the palliative treatment cannot be carried out, when there is a tendency to the occurrence of complications, if the patient is otherwise suitable for operation, and in candidates for the physical examination for His Majesty's services.

The cases most suitable for operative treatment, and in which a good prospect of cure may be entertained, are those in which the varicosity is confined to one vein, or where the condition is definitely localised.

In the former class the internal saphenous vein, for instance, may be the main vessel affected, and it may show an over-distension in one or two parts of its course.

In the latter there may be a definite and localised dilatation of both saphenous veins, but those cases where there is a diffuse enlargement of both veins and of their tributaries do not afford such suitable conditions for operation. The more the varices lie longitudinally, *i.e.* in the line of the trunk, the better and more lasting is the result.

The operation for varicocele is successful because the whole affected mass can be isolated, and the same condition in the lower limb, similarly treated, gives equally good results.

Many different forms of operative interference have been recommended.

The earlier ones depended for their success upon producing a localised thrombosis of the vessels, and this was attained by the injection of irritating or astringent fluids, but as this was by no means free from risk, it has been abandoned. Other methods consist in removing skin, so as to tighten up the cutaneous covering of the limb and provide an artificial support like a stocking.

It is of the greatest importance to be sure of asepsis in undertaking any operation on the veins.

The usual and best form of operation is that which consists in taking out the affected vein by a long incision and dissection, or by removing an inch or two at intervals, by separate incisions, and by treating the terminal part of the internal saphenous vein in the same way when its tributaries are affected.

The affected veins should be marked out in the skin before operation with carbol fuchsin, as it may be difficult to find the vessels when the patient is recumbent.

Longitudinal incisions are made over the varices, and about three inches of the vessel removed.

A modification of the operation consists in exposing the vein by a short incision, freeing it for a short distance, and then with forceps seizing the vein and twisting it, so drawing out a considerable length and at the same time arresting the bleeding.

Careful ligature of the tributaries is important to prevent subsequent hæmorrhage.

The incisions are carefully sewn and a dressing applied, and the limb should be bandaged over the whole length and put in a splint. The patient may be kept in bed for three weeks, and should be instructed to avoid muscular overstrain, and, if necessary, to wear an elastic or silk stocking for a little longer.

It is often necessary to excise a varicose vein above an ulcer in order to get healing of the ulcer. This may be done although the other indications for operative interference are absent.

Other diseases of veins, including the presence of the embryos of tænia and the bilharzia hæmatobia, and the occurrence of some functional conditions, of which a spasmodic contraction of the veins of the lower limb is the best recognised, require only to be mentioned. The latter condition resembles a thickening of the wall of an organic nature, but is dis-

tinguished by being removed by the application of heat.

ENTRANCE OF AIR INTO VEINS

The explanation of this occurrence is found in one of the factors already mentioned as bringing about the venous flow, namely, the aspirating effect of the heart and of respiratory movements. The thoracic cavity being a closed chamber, increase of the size results in the aspiration into it of air, or of blood into the great veins leading to the heart, and therefore at every respiratory movement the thorax acts as a pumping mechanism, drawing in and expelling blood as well as air. This effect is propagated along the great veins of the neck.

To resist this tendency, we find that the veins of the neck are, as a rule, surrounded by strong fibrous sheaths attached to adjacent structures, so that the vein does not collapse, as it would do were the wall not supported, and an arrangement of this kind is found even so far from the heart as in the axillary vein.

In pathological conditions, further, a vein may be embedded in dense fibrous tissue, may be adherent to adjacent structures, or the wall may be infiltrated and rendered firm.

Lastly, undue traction upon tissues during the course of a surgical operation may prevent a vein collapsing.

In any of these conditions, should the wall of the vein be opened, air is aspirated into the vein. The accident is especially liable to happen if a small piece has been cut out of a vein, *e.g.* in removing an adherent gland, or if the wall is only partially divided.

Practically, the accident is most liable to happen during the course of operations about the root of the neck or the axilla, when the tissues are matted from chronic inflammation or from tumour growth, and when a slight wound is made in the side of a large vein, or if a tributary vein be divided close to its termination in the main vessel.

The accident has occurred, however, even in the facial, subscapular, femoral, and uterine veins.

The signs and symptoms are a gentle hissing or sucking sound, intermittent in character, and the presence of some frothy blood in the wound, together with an immediate and serious interference with the patient's circulation and respiration, evidenced by sudden pallor, deep and sighing respiration, and rapid, flickering pulse, with violent and turbulent cardiac movements. These effects are due to two causes: first, the cardiac muscle is deprived of its usual stimulus, viz. an incompressible fluid, in place of which it has only a mixture of air and fluid in which to act; and, secondly, to impairment of the natural action of the cardiac valves, which are only suited to act in a fluid medium, and are useless in air or froth. The pulmonary circulation is arrested, and the patient may die rapidly and suddenly, or may gradually recover, if the quantity of air that has entered is only small.

Treatment.—The treatment must be immediate, and, first of all, the entrance of further air be prevented, either by pressure with the finger or swab upon the aperture, or by flooding the wound with some non-irritating and non-poisonous fluid, such as warm saline solution. It is doubtful if anything can be done to get rid of the air in the heart, but violent expiratory movements sometimes eject it, and the expiratory movements may be assisted once very rapidly. To prevent cerebral syncope the head should be lowered, and the main arteries of the limbs compressed, or even the abdominal aorta, so as to divert the arterial blood to the brain as much as possible. At the same time stimulants should be administered, such as ether subcutaneously, brandy by the rectum, hot cloths to the præcordia.

It has been suggested to aspirate the right auricle by a hollow needle and an aspirating syringe, but this is a *dernier ressort* of a desperate character.

Velamentous.—Membranous, like a membrane (Lat. *velamen*, a covering), *e.g.* velamentous insertion of the umbilical cord (attachment of the cord to the membranes outside the placenta). *See* PREGNANCY, DISEASES OF PLACENTA AND CORD (*Anomalies of Placenta*).

Veldt Fever.—A form of febricula or ephemeral fever.

Veldt Sore. *See* SKIN DISEASES OF THE TROPICS (*Veldt or Natal Sores*).

Vella's Fistula.—A loop of bowel separated from the rest of the intestine and stitched by its ends to the abdominal wall; it is used experimentally to collect the intestinal secretion for examination.

Velpeau's Hernia.—Femoral hernia in front of the vessels.

Velum.—A veil or sheet of tissue resembling a veil; *e.g.* the *velum interpositum* of the third ventricle of the brain, the *velum palati* (soft palate), and the *velum pupillæ* (pupillary membrane). *See* MENINGES OF THE CEREBRUM (*Anatomy and Physiology, Pia Mater*); PALATE (*Diseases*).

Vena.—A vein; *e.g.* the *vena cava, vena portæ*, etc.

Venereal Disease.

INTRODUCTION 428
HISTORY 428

SOFT SORES OR CHANCROIDS . . . 428
Pathology 428
Course 428
Symptoms 428
Position 428
Differential Diagnosis . . . 428
Complications 429
Buboes 429
Phimosis and Paraphimosis . . 430
Gangrene and Sloughing Phagedæna . 430
Serpiginous Ulceration . . . 430
ALLIED DISEASES 431
Venereal Warts 431
Frambœsia or Yaws 431
Radesyge or Spedalsked . . . 431
Sibbens 431
Scherlievo 431
Falcadina 431
Morula or Button Scurvy . . . 432
Ulcerating Granuloma of the Pudenda 432
VENEREAL DISEASES IN ANIMALS—
"*La Dourine*" *in Horses* . . 432
Infective Granulomata in Dogs . . 432

See also GONORRHŒAL INFECTION; SYPHILIS; URETHRA, DISEASES OF; YAWS; etc.

THE term "Venereal Disease" is applied to certain affections due to the inoculation of different varieties of micro-organisms, which often occurs in those who are addicted to promiscuous sexual intercourse. But venereal disease is in every way a bad expression. It has neither a pathological nor a clinical significance, for it groups together a number of wholly different conditions which are not even causally connected, since each of the component affections can only be produced by inoculation with the appropriate culture. The diseases included in the general heading venereal disease, are—gonorrhœa, syphilis, soft sores, venereal warts, and certain affections such as yaws and radesyge, which a better knowledge of pathology has now relegated to their appropriate place. Gonorrhœa (vol. iii. p. 479), the urethritis caused by gonorrhœa (vol. x. p. 253), and syphilis (vol. ix. p. 457), have already been described sufficiently, and the following article deals chiefly with soft sores and venereal warts.

HISTORY.—Much useless but ingenious speculation has been devoted to the history of venereal disease, but the hyperbole of Oriental languages, the vague descriptions of classical satirists, and the reticence of the early physicians and surgeons, do not allow of any accurate statements. It appears, however, that syphilis is a new disease so far as the Old World is concerned, that gonorrhœa was less troublesome in classical times than it is now, and that soft sores and venereal warts have long been recognised as a consequence of impure intercourse between the sexes.

SOFT SORES or CHANCROIDS are ulcers produced by the inoculation of a particular form of micro-organism known as Ducrey's bacillus. The organism is short and thick, with rounded ends, looking much like a figure of eight. It is found in the cells as well as in the intercellular spaces of the affected parts, and its presence has been demonstrated in some of the buboes which are so often associated with soft sores. The strepto-bacillus can be cultivated on a medium consisting of two parts agar and one part of blood from the human subject, the dog, or the rabbit, and it is stained by alcoholic solutions of fuchsin, methyl-violet, and gentian violet. A successful inoculation with this micro-organism alone will produce all the characters of chancroid, but the course of the chancroidal ulcer contracted in the usual manner is modified by the presence of other pyogenic and pathogenic organisms, of which the staphylococci and streptococci are the most important.

Course.—Soft sores and the initial manifestation of syphilis or the hard sore are often mistaken for each other, owing to the fact that a person who contracts syphilis may also be infected with the poison of a soft sore. A chancroid, however, has only a short incubation period, whilst the hard sore does not show itself for many days after inoculation.

The first manifestation of a soft sore occurs within a few hours of inoculation, and is very rarely delayed more than a week. A vesicle appears at the seat of inoculation, and rapidly becomes pustular, forming a rounded ulcer which soon becomes inflamed, though its base never attains to the induration of the hard sore, which is the first manifestation of syphilis. The walls of the ulcer are undermined, its floor is irregular and covered with a yellow exudation. The ulcer secretes pus in abundance, and the pus is infective, for it is capable of inoculation upon the same individual as well as upon other people. Soft sores, therefore, are often multiple, whilst it is quite unusual for a syphilitic sore to be other than single (*see* vol. ix. p. 461).

Differential Diagnosis.—Soft sores are found on the genital organs, as they are usually contracted during sexual intercourse. They occur in men on the glans penis and on the prepuce near the frænum; in women at the fourchette and on the inner surface of the labia, as well as near the anus. Soft sores have to be distinguished from the initial manifestation of syphilis, from herpes, from balanitis, from gummatous ulceration, and from tuberculous ulceration. They can be distinguished from syphilis by their number, by their capability of inoculation, by the shorter incubation period, and by the smaller amount of induration. Yet these differences only hold good for typical cases, and may each and all fail in the very cases where it is of the greatest importance to distinguish between soft and hard sores, for the simple reason that a person who has contracted a chancroid may at the same time have become inocu-

lated with syphilis. It is unwise, therefore, to make the diagnosis too absolute until lapse of time has rendered it quite certain that the patient has not got syphilis.

Herpes progenitalis, or præputialis, when it occurs in men, is very likely to be mistaken for soft sores if the vesicles have become inoculated with pyogenic organisms. The condition of herpes occurs both in men and women: the eruption is vesicular at first, and is often unilateral or in accordance with the nerve distribution. Dr. R. W. Taylor lays particular stress upon the fact that chancrous erosions assume a round or oval outline, while herpes has a polycyclic form with festooned margins, due to the fusion of groups of vesicles, a polycylic outline being that which is presented by two pieces of three-leaf clover placed base to base. Bacteriological examination of the secretion from the inflamed surface will assist the diagnosis in difficult cases; but as a rule the sensations of heat, itching, and burning, the superficial character of the lesion, its less profuse secretion, the scarcely undermined edges, and the tendency to relapse, will distinguish the eruption of herpes from soft sores.

Balanitis is an inflammation of the mucous membrane of the prepuce or glans penis. It is often associated with soft sores, though it may occur quite independently. The diagnosis can often only be made after the prepuce has been incised, and the soft sores are laid open to view.

Gummatous ulceration is to be distinguished from soft sores partly by the slower course which it runs, partly by the history, and partly by the difference in the glandular enlargement, for in soft sores buboes are formed, whilst in gummatous ulceration there is less tendency for the glands to suppurate.

Tuberculous ulceration of the penis or vulva can only be distinguished from soft sores by a thorough bacteriological examination, which should include more than one inoculation of the suspected tissue. The patient often shows other signs of active tuberculosis, and the tuberculous inflammation runs a slower course than that due to chancroids.

Complications. — Buboes, phimosis, paraphimosis, gangrene, sloughing, and phagedæna are the more important complications attending the inoculation of chancroidal pus.

Buboes are enlargements of the superficial inguinal glands, which lie horizontally along Poupart's ligament; the more numerous glands grouped vertically over the upper part of the internal saphenous vein are not so often affected. The inflammation varies from a slight lymphadenitis, which shows no tendency to suppurate, to a virulent inflammation which spreads with alarming rapidity, and may cause ulceration of the femoral vessels. Buboes are seen more frequently in hospital than in private practice, so that carelessness and want of rest are predisposing factors. The nature of the infection, too, bears some relation to buboes, for the worst forms are not necessarily associated with the most angry sores. The bacteriology has not yet been worked out fully, but the specific strepto-bacillus has been found in the abscess wall of the glands, as well as in glands which have not yet suppurated. Some buboes, on the other hand, have been found to be free from Ducrey's bacillus though they were due to soft sores.

The symptoms of a bubo are those of an ordinary infective lymphadenitis. A tender swelling, which at first is movable, occupies the position of a lymphatic gland. The swelling soon becomes fixed, and the overlying skin shows signs of inflammation.

Treatment.—Rest in bed, with the application of fomentations of boric acid (20 grains to the ounce), will often allay the glandular inflammation if it be seen early, especially if this treatment be supplemented with a calomel purge, followed by a course of quinine and iron. In three cases, when the soft sores had been cured and the glands were on the point of suppuration, whilst the patient was unusually anxious to avoid the inconvenience of a prolonged healing, I have consented to dissect out the affected glands by a formal operation. The end justified the means in every case, for healing took place by first intention, and the patient has been repaid for his fortnight in bed. Such cases, however, must be chosen with care, and are useless if the chancroid is uncured. The more usual treatment consists in carrying an incision into the suppurating gland parallel with the femoral vessels, *i.e.* at right angles to Poupart's ligament. The pus is allowed to escape, and the remains of the gland should then be scraped away with a sharp spoon, the cavity being swabbed out with a solution of chloride of zinc (40 grains to the ounce) and afterwards irrigated with a saturated solution of boric acid at a temperature of 110° F. The cavity is then lightly plugged with iodoform gauze, and is allowed to heal from the bottom. It is often advisable to order a patient to take prolonged warm baths in cases where there is extensive destruction of the skin in connection with the buboes, and it may be necessary to employ various escharotics to arrest the ulcerative process when it is found to be spreading rapidly. It should be borne in mind, however, that the rapid extension of the ulceration is due to the multiplication of micro-organisms in a favourable medium, and that no escharotic acts far from its point of application. The general condition of the patient, therefore, must be improved, and the diseased tissue should be thoroughly scraped away before the nitric acid, formalin, carbolic acid, or nitrate of silver is applied.

The abortive treatment of buboes by the injection of iodoform emulsion or carbolic acid at a comparatively early period has not proved satisfactory in my hands as a general rule, and in the successful cases I have thought that the result was rather due to the rest in bed than to the antiseptic injected.

Phimosis and Paraphimosis.—Phimosis is one of the commonest complications of soft sores in men who are careless, or who have been treated too actively. It is due to an inflammatory swelling of the prepuce with an accumulation of pus beneath it. The inflammatory process runs a rapid course, and may end in such perforation of the prepuce as to cause exposure of the glans or even ulceration of the urethra.

Paraphimosis is a rarer complication of soft sores, and is due either to the swelling of a short foreskin or to the forcible retraction of an unduly long one. The swelling of the glans penis is greatly increased, and great œdema occurs owing to the inflammation of the soft sores.

Treatment.—A patient with commencing phimosis must, if possible, be kept in bed with his penis enveloped in a compress of lead lotion and slung against his abdomen. He should further be ordered a calomel purge, and he should be placed upon a course of quinine. If the inflammation continues in spite of this treatment the prepuce must be slit up, for in no other way can the inflammation be arrested so satisfactorily. The parts should be rendered as aseptic as possible, and the patient is placed under the influence of nitrous oxide gas or of somnoform, though many prefer the use of cocaine or eucaine. A director is introduced through the orifice of the prepuce, taking care that it does not pass into the urethra, and is carried onwards to the corona. A curved bistoury is passed along the groove, and is made to transfix the prepuce at the level of the corona, and the incision is carried through the whole thickness of the prepuce to its free edge. The cut is generally made along the dorsum, but Dr. Robert A. Taylor has lately expressed his belief that better results are obtained by employing two lateral incisions. The prepuce is then converted into an upper and a lower flap, and the glans is much more freely exposed than by the old method. The bleeding is not serious in either case, and is readily arrested by the application of a dry dressing of tannoform or iodoform gauze kept in place by a couple of turns of rubber plaster.

Paraphimosis is more amenable to treatment than phimosis. Rest in bed with the application of a compress of lead lotion will usually allay the inflammation sufficiently in a few hours to allow the prepuce to be replaced over the glans, and it is only in the rarest cases that it is necessary to divide the constriction. Reduction is effected in the usual manner by first squeezing as much blood as possible out of the glans either by digital or elastic pressure, and then pulling the foreskin over it.

Gangrene and sloughing phagedæna affect the penis, the groin, or the skin over the pubes by an infection which extends rapidly in persons who have a diminished power of resistance either from acquired or constitutional causes. The tuberculous and diabetic are said to be more likely to suffer from phagedæna if they contract soft sores than are healthy persons. It is certain that habitual drunkards are more liable to this form of inflammation; but phagedæna is much less commonly seen since the introduction of antiseptic treatment, and since the administration of mercury has been restricted to those who have syphilis.

Treatment.—The treatment consists in placing the patient under an anæsthetic, scraping away all the diseased tissue with a sharp spoon, brushing the wound with nitric acid, paying special attention to the edges, which must be thoroughly destroyed, and afterwards packing the whole surface with dry iodoform gauze which has been sterilised. The patient ought to be well fed, he should be given a liberal allowance of alcohol, and a grain or two of opium in the course of every twenty-four hours will often prove to be of the greatest benefit.

The most severe cases of sloughing phagedæna can sometimes be cured by almost continual immersion in a warm bath. Fortunately, such cases only occur in hospitals, for it is only in a public institution that this course of treatment can be carried out satisfactorily.

The patient can easily be kept in the bath continuously for days or weeks, and if a horseshoe air cushion be tied round his neck he can sleep securely without danger of being immersed. The water must be changed and the bath thoroughly cleaned once every twenty-four hours. The patient should be wrapped in blankets and laid on a bed whilst this is being done, and it is convenient that he should have an action of the bowels at this time. It is quite possible, however, to employ a bed-pan in the bath with very little soiling of the water. The bath may be so arranged as to allow of a constant flow of water through it, or, if it be medicated (vol. i. p. 349), some of the water may be drawn off and fresh water may be added every hour and a half. The temperature of the water is maintained at 99° to 100° F.

The base of a soft sore is sometimes covered with a greenish or yellow membrane, and the ulcer is then called *diphtheritic*. Such ulcers run a somewhat chronic course, though they can be easily stimulated into activity and heal readily.

Considerable tracts of skin occasionally become involved in chronic ulceration as a result of infection with chancroidal virus. The edges of the advancing ulceration are curvilinear—whence the name *serpiginous*—whilst the seat

of inoculation cicatrises. Serpiginous ulceration in this country is often associated with previous syphilitic infection, and always occurs in unhealthy people. It is treated constitutionally, as if the patient had tertiary syphilis; locally, by thoroughly scraping and disinfecting the surface and edges of the ulcer. An analogous condition, which is certainly not syphilitic, is known as ulcerating granuloma of the pudenda (*see* p. 432).

ALLIED DISEASES.—Certain affections have always been branded as venereal, though in many instances pathology has relegated them to very different classes, and has shown that they are not necessarily contracted during sexual intercourse.

Venereal warts are perhaps the best known of these allied affections. They are papillomata found about the genital organs. They grow at the coronary sulcus, at the inner surface and margin of the foreskin, near the frænum and at the meatus in men, whilst in women the labia, the urethra, and the margins of the anus are the commonest seats of growth. They may arise *de novo* from various causes of local irritation, and their growth is favoured by heat and moisture. They occur in two varieties, the hard and the soft warts. Hard warts are often small in size, bright red in colour, round or pointed in shape; soft warts vary in size from minute points to enormous cauliflower-like masses. Both hard and soft warts are usually multiple. They are convertible one into the other, and both, unless they are kept clean, may produce a very foul discharge. Such warts grow rapidly in pregnant women and dwindle again after delivery. There is reason to suppose that venereal warts are contagious in some cases, but it is necessary for the recipient of the infection to supply a proper soil, as it is not every skin or mucous membrane that will grow warts.

Treatment.—The following applications have often given me good results in the slighter forms of venereal warts:—(1) ℞ Pulv. cupri. sulphatis, pulv. foliorum sabinæ, amyli, āā, partes æquales. Fiat pulvis. Signa. Dust the powder over the warts on four successive evenings, and wash away the secretion with soap and warm water twice a day. (2) ℞ Tannin, camphoræ, pulv. foliorum sabinæ, āā ℥ss., pulv. zinci oxidi ℥vj. Fiat pulvis. Signa. To be dusted over the warts night and morning for fourteen days.

It is often necessary to remove the growths by operative measures, and it should be remembered that mere ligature and snipping the warts off is insufficient. The surface from which they grow should first be painted with a 10 per cent solution of hydrochlorate of cocaine, and it should then be scraped thoroughly with a sharp spoon, a dry dressing of iodoform or cyanide gauze being afterwards applied. The bases of the large cauliflower excrescences must be dissected out whilst the patient is under a general anæsthetic, the wound being sutured in the usual manner. Cleanliness, in every case, is a great feature in the treatment of warts, and all irritating discharges over the skin or mucous membrane upon which they grow should be stopped as quickly as possible. The persistence of warts in old people will naturally raise a suspicion of epithelioma.

Framboesia—known in its different localities as yaws, parangi, patéh, pian, buba, Tonga, verruga, coko, purru, and Amboyna button—is one of the best known affections allied to venereal disease. It is endemic in many tropical countries, all its names appearing to have their origin in some African dialect. (*See* "YAWS.")

Radesyge is known in Norway as spedalsked. It was long described as a form of syphilis, but it is now classed with leprosy. It occurs on the sea-coast of Norway, Sweden, Iceland, and Greenland, and is characterised by an ulceration of the nose, mouth, and fauces, which superficially resembles the ulceration of syphilis. Cleanliness, good food, and warm clothing form the prophylactic treatment. Sarsaparilla with small doses of mercury may be given with advantage, and the sores should be dressed with boric fomentations.

Sibbens was endemic for a long time in the south of Scotland, though it seems now to be extinct. It attacked infants, who transmitted it to their nurses. The eruption was a fungating, copper-coloured growth, associated with ulceration of the palate and an aphthous ulceration of the mucous membrane of the mouth. The eruption on the skin was widely distributed and usually scabbed. Alibert described the disease as a syphilitic mycosis, but it is probable that its true pathology will never be known.

Scherlievo, or the mal de Fiume, appears to have been syphilis occurring amongst an unprotected and poverty-stricken population. It derives its name from the small village on the shores of the Adriatic where it was noticed to be epidemic in June 1800. The first symptoms were pains in the bones, especially at night; the pains were followed in a fortnight or three weeks by dysphagia and hoarseness, with the appearance of rounded ulcers on the velum palati and on the inner sides of the cheeks and lips. Large coppered-coloured spots appeared upon various parts of the body, which underwent deep and extensive ulceration. The disease was contagious, and one attack protected. Mercury was curative when administered internally.

Falcadina presents many points of resemblance to scherlievo. It was first observed at Facaldo, a village in the Austrian Tyrol, about 1786. It became epidemic, and was propagated by sexual intercourse. The disease was characterised by ulcerating pustules with blennor-

rhagia, condylomata, and buboes, whilst the bones showed nodes. The mortality was great at first, but afterwards the epidemic died away.

Morula or *button scurvy* appears to have been allied to syphilis. It occurred in Ireland during the early part of the last century, but is now extinct, and as the pathology of venereal disease was at that time in a state of extreme confusion, it is unlikely that the exact nature of the disease will ever be known. It has, therefore, to be classed with Saint Euphemia's evil (1727), the disease of St. Paul's Bay (1760), of Brunn in Moravia, and of Chavanne Lure, as probably syphilitic in origin.

Ulcerating granuloma of the pudenda is also known as "The Groin Ulceration" of British Guiana. It begins in the form of papules which increase in size, become abraded and then granulate. There is not much suppuration, but the abraded surfaces bleed readily and finally scar over. (*See also* "PUDENDA," vol. viii. p. 347.)

VENEREAL DISEASE IN ANIMALS.—Horses and dogs are both liable to contract disease during sexual intercourse. "La Dourine," badly called veterinary syphilis, is a disease communicable by coitus, which has long prevailed amongst the horses of the French army. It was first noticed in France at Tarbes in 1851, though it was observed and described by J. Ammon in the grand-duchy of Posen as early as 1796. The disease attacks the reproductive organs, where it presents certain local manifestations, followed by numerous eruptions and by constitutional symptoms, the most prominent being a paralysis of the animal's hind quarters. The duration of the affection is a few months to a year. Death occurs from exhaustion or hypostatic pneumonia, though recovery sometimes occurs.

Dogs suffer from certain infective granulomata often called papillomata, condylomata, and warts. The contagion is conveyed during the act of coitus, and the tumours are not dependent upon the irritation of any discharge. The tumours can be transplanted artificially. Secondary deposits occur in the lymphatic glands, as well as in the liver and spleen, but the growths can only be inoculated upon dogs. Much further investigation is required in connection with the pathology of these interesting growths, for it is said that they may be inherited, and it is possible that the tumour cells are themselves infective.

Venesection.

THE OPERATIONS 432
THE INDICATIONS FOR 432

See also BERIBERI (*Treatment*); BRAIN, SURGERY OF (*Compression, Treatment*); BRONCHI, BRONCHITIS (*Treatment*); CHLOROSIS (*Treatment*); ECLAMPSIA (*Treatment*); LIVER, CONGESTION OF (*Treatment*); MEDIASTINUM (*Growths, Treatment*); URÆMIA (*Treatment*).

THIS operation consists in the abstraction of blood by incision into a vein. The median cephalic vein at the bend of the elbow is the vessel usually selected. The operation is done as follows:—The skin of the part is thoroughly cleansed, and a bandage is then rolled tightly round the upper arm and the patient is directed to grasp something firmly in the corresponding hand. The skin over the vein is then gently stretched by the fingers of one hand of the operator, and an *oblique* incision is made across the vessel with a sharp sterilised knife. The blood is allowed to flow into a vessel until a sufficient quantity has been withdrawn. This operation was formerly very much used, and in a somewhat indiscriminate manner, for all sorts of diseases, acute or chronic, whether in sthenic or in asthenic individuals. It is now comparatively rarely used, but there are good reasons for believing that it is a valuable therapeutic measure, and that its judicious use at the present day would be beneficial.

There are two types of disease for which it is specially applicable: (*a*) in advanced cases of heart disease, when the heart is dilated, its muscle weak, the peripheral circulation distended, and the orthopnœa and general distress of the patient are extreme, the abstraction of from 4 to 10 ounces of blood is sometimes followed by the most striking relief, this occurring in cases where the usual cardiac remedies have been tried with little or no result. Even although the relief in such cases may not be permanent, the favourable results of the operation warrant its adoption in some cases. It should only be carried out after the more ordinary modes of depletion have been tried. *e.g.* judicious use of cathartics and diuretics, combined with the administration of general and local cardiac tonics. Before considering venesection in such cases care should be taken to see that the aggravated nature of the symptoms are not in great part due to the presence of hydrothorax, the relief of which by aspiration may be followed by very satisfactory results.

(*b*) In cases of acute pneumonic fever, especially in sthenic subjects, venesection may be beneficial in some cases. Its manner of action is probably a twofold one. It directly relieves the strain on the cardiac muscle, and is indirectly beneficial, through the removal of a quantity of the toxic material that is present in the blood in these conditions, and which is responsible for the symptoms of the disease.

Interesting sidelights on the value of venesection are sometimes afforded in other departments of medicine. Thus in obstetric practice it is occasionally observed that in acute infection occurring after pregnancy or in the puerperium, *e.g.* eclampsia or puerperal septicæmia, the

occurrence of a free uterine hæmorrhage is followed by a very marked improvement in the symptoms, and in some cases has apparently been the means of saving the patient's life. The manner of action in such cases is probably identical with that already described.

In cases where venesection is indicated the use of transfusion of saline fluids (*see* "TRANSFUSION"), or the subcutaneous or rectal injection of neutral salines, may be a very useful adjunct to treatment.

Venom. *See* SNAKE-BITES AND POISONOUS FISHES.

Venous.—Relating to veins or to the blood in veins; *e.g.* a *venous hum* (*see* CHLOROSIS, *Symptoms*, *Bruit de diable*), *venous nævus* (*see* CAPILLARIES, DISEASES OF), *venous pulsation* (*see* PULSE, *Venous and Liver Pulsations*), etc.

Venter.—The abdomen or belly; also, the fleshy part of a muscle. *See* ABDOMEN.

Ventilation and Warming.

THE PROBLEM OF VENTILATION . . 433
AMOUNT OF AIR NECESSARY PER PERSON PER HOUR 436
EXTRACTION SYSTEMS 437
Open fire with Flues . . . 437
Ventilating Grates or Stoves . . 438
Ventilating Shafts 438
Cowls 438
Inlets and Outlets 438
PLENUM OR PROPULSION SYSTEMS . . 439
NOTES ON WARMING 439

1. THE PROBLEM.—The problems associated under the term Ventilation arise out of the relation of the lung-breathing organisms to their environment. For simplicity, these problems may be studied on the human organism, but all the higher mammals, in their degrees, require similar conditions. If a human being always lived in the "open air," there would be no problem of ventilation; for the organism would absorb from the air precisely the amount of oxygen necessary for perfect respiration, and discharge into the air a certain amount of carbonic acid, aqueous vapour, and the other metabolic impurities that issue normally by the lungs. If the air became disagreeable, or unwholesome, as from offensive gases, from marshes, or fires, or volcanoes, or factories, or decomposing organic materials of any sort, the ventilation problem could be solved by walking away; but where the open air of a region becomes too impure for respiration, ventilation in the ordinary sense is not a remedy. The real problem of ventilation arises from the fact that to live at all in a variable climate the human organism requires shelter; the shelter being necessarily limited, rapidly accumulates impurities, and as these would, in concentration, become fatal, they must either be reduced to an amount compatible with physiological living, or, if possible, be altogether eliminated. The central problem of ventilation, therefore, is how to maintain, within a partially closed space occupied by a human being, the same quality of atmosphere as may be obtained in an open healthy locality.

2. The chief factors in the solution of this problem are the following:—

First, the composition of average, unpolluted air.

Second, organismal pollutions—respiration.

Third, environmental pollutions—combustion of coal, coal-gas, paraffin or other oils, candles, or other substances used for lighting or heating; pollution from floors, drains, walls, domestic industries, as cooking, washing, etc.

Fourth, the physiological standard of air-purity.

Fifth, the dynamics of ventilation, and ventilation systems.

The relation of ventilation to warming will emerge in the exposition of these five topics, which form a related sequence. Some further notes on warming will be appended.

3. *First, the Composition of Air.*—The composition of average country air free from aqueous vapour is, according to Haldane, as follows:—

Oxygen	By volume,		20·94
Nitrogen	,,	,,	78·09
Argon, with traces of metargon, xenon, crypton, neon, helium, and hydrogen	,,	,,	0·94
Carbonic acid	,,	,,	0·03
Total			100·00

Normally, air contains aqueous vapour in amounts depending on temperature and pressure, traces of ammonia, traces of sodium salts and other mineral substances, organic matter, particulate or gaseous, micro-organisms, including moulds and bacteria, dust of various degrees of fineness.

For ventilation, the ingredients of primary importance are the oxygen, the nitrogen, the carbonic acid, the aqueous vapour, and the organic matter, living and dead. The others may be neglected.

Oxygen.—This gas is indispensable to metabolism. On the slight variations in the amount of oxygen depends mainly the wholesomeness or unwholesomeness of the air in a locality. Dr. Angus Smith found that it varies from 20·999 per cent (by volume) on an open heath in Scotland to 20·980 in an open space in London in summer; 20·650 in the Court of Queen's Bench, 2nd February 1866, and 18·270 in a badly-ventilated mine. If, however, abundance of air be available for ventilation, the exact percentage of oxygen is not very material; but when the percentage is high, ventilation is easier, and certainly localities where there is a very high percentage are healthier.

Nitrogen.—For respiration, nitrogen is inert; but it forms a diluting vehicle for the oxygen, which, if breathed in concentration, produces symptoms of excitement.

Carbon Dioxide, or Carbonic Acid.—For ventilation, this is always an impurity; but in the small percentages present in average air it is harmless. In normal air it varies from less than 3 to more than 6 parts per 10,000 volumes. Haldane states that the results by Pettenkofer's method are usually too high by about 0·5 parts per 10,000 volumes of air. In open country, Haldane has found that CO_2 varies from 3·5 volumes by night in summer per 10,000 volumes of air to 2·6 volumes by day in the lower strata in consequence of the influence of vegetation. In dry air the amount of CO_2 in the open country in Scotland is amost exactly 3·0 parts per 10,000 volumes. This result Haldane gives as the average of a series of exact determinations. In 29 analyses made between 1st April and 30th September, he found a day maximum of 3·11 parts per 10,000 volumes, a day minimum of 2·58—average, 2·88. In 23 analyses he found a night maximum of 3·55, a night minimum of 2·82—average, 3·08. Average of night and day analyses, 2·98. In 6 analyses made between December and January, he found a day maximum of 3·12, a day minimum of 2·93—average, 2·99. In 5 analyses he found a night maximum of 3·06, a night minimum of 2·94—average, 3·01. Average of night and day, 3·00. In London air the CO_2 varies from 4·5 in clear air to 14·1 in a fog. Analyses made by Dr. Russell during 1882-83 gave an average of 4·01. Probably a more correct average is 3·7. Fogs should be avoided at the time of estimation.

Organic Matter.—This includes micro-organisms, organic dust, organic effluvia of unascertained composition, organic gases. The amount depends on local conditions and on respiration. The hypothesis that the deleterious effect of air from an overcrowded room is due to a particular poisonous organic substance is not borne out by recent analysis (Haldane).

Aqueous Vapour.—For ventilation and warming, aqueous vapour is not unimportant. A very moist atmosphere favours dampness of walls, with consequent chilling; a very dry atmosphere, especially if warmed, causes discomfort. This is probably due to the absorption of moisture from skin and mucous surfaces. Dry air, also, by increasing the rate of evaporation, chills damp objects in a room. To prevent discomfort when air is artificially warmed, the "humidity should not be less than 60 per cent of saturation" (Shaw). Saturation depends, not on the absolute amount of aqueous vapour in the given quantity of air, but on the temperature of the air. Thus 1·1 grain of aqueous vapour saturates a cubic foot of air at 10° Fahr.; but at 50° Fahr. it takes 4·2 grains to saturate the same quantity of air; at 60° Fahr., 5·8 grains; at 80° Fahr., 10·7 grains; at 100° Fahr., 19·1 grains; at 130° Fahr., 42·5 grains. In all these cases the percentage of humidity is 100, which means saturation. It follows that if, for example, air at 60° Fahr. contains only 2·9 grains of aqueous vapour per cubic foot—that is half its saturation quantity at that temperature—it is less humid than air at 10° Fahr. when this contains in each cubic foot only 1·1 grain, which at that temperature is saturation. The amount of aqueous vapour in the air, either in rooms or in the open, can be estimated very precisely by various hygrometers, *e.g.* the wet and dry bulb thermometers.

The humidity of the air conditions, to a large extent, the variations and effects of temperature. A moist air secures greater uniformity of climate. In Britain, owing to the greater humidity, lower temperatures in warming of houses can be tolerated than in America, which is relatively dry. Billings states that in America, to secure comfort, rooms must be kept at a temperature of 68° Fahr. to 70° Fahr.; in England, comfort is secured at 60° Fahr. The amount of aqueous vapour in inhabited rooms varies more or less as the amount of respiratory impurity, and this fact has been made the basis of a method for estimating the impurity. But, as a rule, the amount of aqueous vapour varies so rapidly from hour to hour that, except in great excess of impurity, the relation of impurity to vapour is too inconstant to be a reliable index.

Second, Organismal Pollutions—Respiration.—In parts per cent, the difference between inspired and expired air is, according to Foster, as follows:—

	Oxygen.	Nitrogen.	Carbonic Acid.
Inspired air contains .	20·94	79·15	·03 to ·04
Expired air contains .	16·033	79·581	4·38

The quantities for inspired air differ slightly from the latest averages, but are sufficiently accurate.

Thus the expired air contains from 4 to 5 per cent less of oxygen and some 4 per cent more of carbonic acid. At the rate of 25 cubic inches (tidal air) each respiration and 17 respirations a minute, a man will, in an hour, pollute 102,000 cubic inches to the extent of 1 per cent (volume) of CO_2, which means an excretion of 0·6 cubic foot CO_2 per hour. This quantity approximates to the theoretically expected result of food-metabolism, and has been verified by experiment. But average air contains ·03 to ·04 CO_2 per 100, or 0·3 to 0·4 per 1000 volumes. Consequently, if a man breathes for an hour in a room of 1000 cubic feet (10 ft. × 10 ft. × 10 ft.), the amount of CO_2 at the end of the hour, if no air is admitted or withdrawn meanwhile, will be $0·6 + 0·4 = 1·0$ cubic foot. But air containing this amount of CO_2 due to respiration is unwholesome, and must be diluted until the total CO_2 does not

exceed 0·6 parts per 1000. This is done by the admission of 2000 cubic feet of normal air per hour. The man thus is provided with 1000 + 2000, or 3000 cubic feet of air per hour The amount of CO_2 in cubic feet will then be as under:—

$(0{\cdot}4 \times 3)$	+	$(0{\cdot}6)$	
‖		‖	= 1·8 parts in 3000 cubic feet, or 0·6 in 1000 cubic feet.
Normal CO_2 in air per 1000 vols.		CO_2 contributed by respiration.	

This standard—0·6 per 1000—is aimed at in dwelling-houses, etc., but in practice it is rarely attainable. Nor, except in prisons, hospitals, and the like, where a patient is entirely confined for long periods, is it necessary for health. Temporary exposure to such polluted air is not injurious; but repeated exposure for prolonged periods is extremely injurious to sick and healthy alike. Probably, CO_2 by itself is not, in the above quantities, very deleterious; but it is the only easily tested impurity, and is thus the best index. The disagreement among physiologists as to its poisonousness relatively to the exhaled organic matter and aqueous vapour does not affect its value as a practical test. The "close" smell of over-occupied or over-crowded rooms is probably due to organic effluvia from breath, mucous membranes, or skin; the concentration of CO_2 is an index of the concentration of other impurities.

Third, Environmental Pollutions—Coal-gas, Oil, etc.—(*a*) *Coal-gas.*—One cubic foot of gas when consumed is equivalent in polluting effect (in times of CO_2) to one adult person. Each gas-burner in common domestic use consumes from 4 to 5 cubic feet per hour, and is therefore equivalent to 4 or 5 adults. Three ordinary gas-lights in a room are equivalent to the presence of 12 to 15 additional people. If the products of combustion mix with the room-air,—the commonest case,—then, in order to keep the CO_2 down to respirable limits, 1800 cubic feet of fresh air per hour must be supplied for each cubic foot of gas burned. This is approximately the same amount as that required for an adult breathing in a room of 1000 cubic feet. Put otherwise, one person requires 3000 cubic feet of air per hour; 1 cubic foot of gas burnt requires 1800 cubic feet of air; 5 cubic feet require 9000 cubic feet. Coal-gas, unconsumed, is poisonous; minute escapes are very dangerous. The ingredients of unburnt coal-gas are, methane (CH_4) or marsh-gas, ethylene (C_2H_4) or olefiant-gas, acetylene (C_2H_2), hydrogen, carbon monoxide, nitrogen, vapours of volatile liquid hydrocarbons, and vapour of carbon bisulphide. Of these, all, except hydrogen, nitrogen, and the hydrocarbon vapours, are in various degrees poisonous. When burnt, coal-gas yields also CO_2 and sulphur dioxide. Consequently, coal-gas, burnt or unburnt, is always an impurity in the air of a room.

(*b*) *Gas-stoves.*—Gas-stoves are simply large gas-burners (usually Bunsen-burners). They may burn from 5 to 40 or more cubic feet per hour, producing corresponding pollution. To prevent this, flues are absolutely necessary. The "condensing" gas-stoves are said not to pollute the air; but carbon dioxide—one of the chief products of combustion—is not removed in the liquid due to condensation. Nor can combustion proceed without oxygen. Consequently, the condensing stove does not collect all the impurities and does abstract the oxygen.

(*c*) *Paraffin Oil.*—According to Notter and Firth, 1 lb. of paraffin oil requires 138 cubic feet of air for complete combustion, and in polluting effect is equivalent to 15 cubic feet of coal-gas. An ordinary oil-lamp requires as much air as a gas-jet burning 4 or 5 cubic feet of gas per hour.

(*d*) *Candles.*—One candle requires 150 cubic feet of air per hour if the air is to be kept sufficiently pure.

(*e*) *Stoves.*—Closed coal-stoves are liable, especially when over-heated, to emit carbon-monoxide—which is extremely fatal. Charcoal-stoves have the same effect.

(*f*) *Drain-air.*—Drain-air, or sewer-air, may sometimes find access to the occupied rooms. Such air may contain hydrogen sulphide, ammonium sulphide, carbonic acid, and fœtid organic vapours.

(*h*) *Dust.*—Dust is always a pollution, but cannot always be eliminated; the very fine dust never can be. The grosser dust, which may consist of illimitable varieties of filth from sinks, water-closets, skin, unclean utensils, unwashed clothes, over-used blankets, befouled floors, floor-deafening, dirty carpets, epithelial scales, voided mucus, bacteria, etc., can be effectually rendered, at least temporarily, harmless by wet-cleaning or spraying.

Fourth, Standard of Air-purity; and Quantity of Air necessary to maintain it.—Cubic Space.—The standard of air-purity aimed at varies with the nature of the premises to be ventilated. The physiological ideal is: a maximum of 6 volumes of CO_2 per 10,000 volumes of air, that is, 2 volumes above the 3·5 to 4 volumes existing in average air. But this standard cannot be maintained except where the air supplied constantly amounts to 3000 cubic feet per head per hour, or where the CO_2, with the other impurities, is directly removed before it diffuses into the air of the room. Now the ordinary methods of ventilation rarely secure 3000 cubic feet of air per head per hour, either in a private dwelling-house or in work premises. Without a specially adjusted system of ventilation, it is not possible to provide this amount of air, or to diffuse it uniformly through the occupied premises. Hence, in practice, a compromise has to be made between the temporary ill-effects of air polluted beyond the 6 parts of CO_2

per 10,000, and the discomfort or ill-effects due to cold draughts, or to excessive radiation of body-heat to cold objects in the room. The excessive radiation of body-heat to cold solid objects, or to objects persistently cooled by evaporation from moist surfaces, or to thin exposed walls, or to windows, is as important a cause of discomfort as exposure to draughts. If the solid objects in a room are warm, cold draughts are easily tolerated, and may be even pleasant. Radiation is, of course, independent of air-currents; heat rays pass, like light, in straight lines in all directions equally from the centre of radiation, and are absorbed or reflected according to circumstances. Hence the necessity of combining ventilation with warming, not necessarily with warming of the air, but with warming of the solid objects. If the body is clothed in good non-conducting clothes, and the room-objects are warm, cold air is not injurious. The "open-air" treatment of phthisis is a standing demonstration of this. It may be called treatment by large draughts.

It is, however, for ordinary life, in limited spaces, difficult to provide without discomfort the requisite quantity of air, which increases with work. Thus an adult male during work requires 9800 cubic feet of air per hour; during repose, 3600 cubic feet: an adult female, in repose, requires 3000 cubic feet; a child, in repose, 2000 cubic feet. In practice, less than this must be accepted. Morin lays down the following approximations:—

Amounts of Air necessary per Person per Hour

Hospitals (ordinary)	2000 to 2400 cub. ft. per hr.
,, (epidemic)	5000 ,, ,, ,,
Workshops (ordinary)	2000 ,, ,, ,,
,, (unhealthy)	3500 ,, ,, ,,
Prisons	1700 ,, ,, ,,
Theatres	1400 to 1700 ,, ,,
Meeting Halls	1000 ,, 2000 ,, ,,
Schools (per child)	400 ,, 500 ,, ,,
,, (per adult)	800 ,, 1000 ,, ,,

Haldane recommends for factories and workshops a maximum standard of 12 volumes CO_2 per 10,000 volumes of air where no gas is used, and 20 per 10,000 where gas in used.

To secure this standard, 1250 cubic feet of air per hour per person must be supplied. He bases this estimate on the fact that about 1 cubic foot CO_2 per hour is produced by a man at work. He has found that even in very badly ventilated workshops the amount of CO_2 rarely surpasses 50 volumes per 10,000, and that it takes six times this amount to interfere seriously with respiration. Professor Thorpe (*see* Jacob's *Ventilation and Warming*, p. 14) found in a sitting-room, near floor, 7·33 CO_2 per 10,000; half-way up, 9; near ceiling, 14·65. In the Grand Theatre, Leeds: pit, 15·01; upper circle, 14·29; balcony, 14·16. In the Philosophical Hall, after lecture, 13·38. Professor Clowes, in Nottingham (*ibid.* pp. 14, 15), found in the chemical laboratory of University College, 6·9; in the grounds of the College, 4·3; in the Masonic Hall during a dance, 31; in the Circus, 32·6; in a committee-room with 15 persons and 27 gas-jets, 41·8. Dr. Angus Smith, in three Manchester mills, found (1) with 400 people, 28·6; (2) with same number, 29·6; (3) with same number, 30. Recent results confirm these analyses.

Cubic Space.—If the impurities of each expiration could be instantly removed, and fresh air supplied directly for each inspiration, the amount of cubic space for inhabited rooms would be of little moment. But the expired impurities diffuse rapidly through the surrounding atmosphere, and the only mode of keeping them down to respirable limits is to dilute them freely with fresh air. To do this without draughts is difficult in a small space. Hence a minimum space compatible at once with fresh air and comfort must be fixed. For dwelling-houses, this cannot be fixed precisely; but the standard for common lodging-houses, namely, 400 cubic feet per bed, may be applied to individual houses. The following table indicates the amount of space required by statute, or order, or otherwise found desirable for various types of building:—

Minimum Floor Space and Cubic Space in various Buildings

	Floor space in sq. ft. per bed.	Cubic space in cub. ft. per bed
Barrack-room, occupied day and night	50–80	600
Common lodging-house	42	300–400
Factories and workshops—		
Ordinary hours	...	250
Overtime	...	400
Special trades, by order of Secretary of State	...	various
Hospitals—General	90–140	1200–1600
Maternity	100–140	1200–2000
Infectious disease	140–200	2000
Poorhouses and workhouses—		
Sick	72	850
Lying-in women	100	1200
Offensive cases	...	...
Infirm persons occupying same room day and night	...	700
Infirm persons able to leave dormitory during day	42	500
Healthy persons	42	300
Prison cells for continuous confinement	80–120	600
Schools—Day schools	8	80
Dormitories	50–60	400

Unless properly ventilated, a large cubic space becomes ultimately as much fouled as a small. All the above quantities presuppose the most adequate ventilation. Floor space is very important: (*a*) because it is essential to management; (*b*) because it minimises contacts in infection. Height should not exceed 14 feet, unless there is a ceiling ventilator; a greater height tends to cool the fouled and heated air, and thus cause it to re-mix with the air of the room. Where gas is freely burned, the heat of

the combustion practically makes a separate circulation above the level of the gas-jets; the result is the same as if the ceiling were lowered to that level (Shaw).

Fifth, Dynamics of Ventilation.—Practical ventilation is an engineering problem, the conditions of which are never twice the same. This is obvious from the following enumeration of premises needing ventilation:—Dwelling-houses, including cellar-dwellings, one-roomed, two-roomed houses, one storied, back-to-back, tenements of flats, semi-detached and detached villas, mansion-houses, common lodging-houses, poorhouses, workhouses, prisons — all being occupied for the greater part of the twenty-four hours; Schools, day-schools, night-schools. lit by gas or lamp—all being occupied for several hours continuously; Churches and meeting-halls—being occupied for two hours or more by persons in repose; Drill-halls, gymnasia, etc.—being occupied for longer and shorter periods by persons in great activity; Workshops and factories—being occupied for twelve hours or more continuously in varying conditions as to dust, dampness, trade effluvia, etc., by persons active and passive; Hospitals, occupied by the sick—including general hospitals, maternity hospitals, infectious disease hospitals, sanatoria for consumption, etc. No single system is equally applicable to all these, not to speak of ships, canal-boats, mines, etc. The fundamental principles, however, are the same for all.

The physical theory of ventilation may be stated in two propositions: (*a*) Given a "head of air," then a continuous flow of air through a room can be maintained, the quantity of air entering by all inlets being exactly equal to that issuing by all outlets; (*b*) a "head of air" is produced by difference of pressure between the air within and the air without a room. Air is not alive; it will not move of its own accord.

From these two principles, it follows that the essence of ventilation as a physical problem is how to produce such a head of air as will cause a required quantity of air to flow continuously at a required rate through a room with given inlets and outlets. In practice, the problem is how to provide each person with approximately the quantities of air specified in the above tables.

Sixth, Systems of Ventilation: Ventilating Mechanisms.—Systems of ventilation may be classified into (A) Vacuum Systems, and (B) Plenum Systems—these terms representing the two fundamental methods of producing a "head of air," with the consequent flow of air into and out of the room. The common division into "natural" systems and "mechanical" systems is peculiarly inept; since the so-called "natural" systems are highly artificial, and the "mechanical" systems involve precisely the same principle, namely, alteration in air-pressure, as the "natural" systems.

(A) Vacuum or Extraction Systems

(*a*) *Open Fire with Flue.*—The heat of the fire expands the volume of air in the chimney, reduces the density, so causes an upward flow, which in turn causes a flow in the room-air. The air removed must be made good by air from without. The chimney is the outlet; the inlets are all other openings, including cracks, crevices, doors, windows, etc. The ventilating efficiency depends on the difference between the temperature maintained in the chimney and the temperature of the air outside.

The following table from Morin indicates the extraordinary capacity of the open fire as a ventilator:—

Relative Size of Chimney Flue for different Rooms

Cubic Capacity of Room in Cubic Feet.	Volume of Air to be removed by the Chimney per Hour in Cubic Feet.	Area of Section of Rectangular Chimney Flue in Square Feet.	Diameter of Section of Cylindrical Chimney in Feet.
3,500	17,500	·99	·88
4,200	21,000	1·19	·98
5,300	26,500	1·48	1·08
6,350	31,750	1·78	1·21
7,750	38,750	2·17	1·31
9,200	46,000	2·57	1·44
10,600	53,000	2·97	1·54

The open fire is a compromise between a ventilator and a heater. To secure the best results for ventilation, without sacrificing too much heat, "Morin recommends that the temperature of the air in the chimney be maintained at about 45° Fahr. above the external air, and the velocity of the smoke issuing from the chimney should be about 10 feet per second, in order to secure stability in the draught, and that the chimney should be capped with a cone-shaped top—the area of the orifice of discharge to be one-half that of the chimney" (Shaw in *Stevenson and Murphy*, vol. i. p. 126). From the table it is seen that a ward of 10,600 cubic feet, which would accommodate from five to six infectious patients at 2000 cubic feet per patient, may, by a rectangular flue of less than 3 feet square, or a cylindrical flue of 1½ feet square, have an air supply of 53,000 cubic feet per hour drawn through. Thus if the inlets are of the proper size and properly placed, each patient may be supplied with from 9000 to 10,600 cubic feet of air per hour, which is three times more than the amount theoretically necessary. Allowance, however, must be made for irregularity of draught, closing of inlets, sinking of fire, etc. Where gas-jets are the heaters, the current may be kept constant, provided the temperature is maintained in the flue.

(*b*) *Ventilating Grates or Stoves.*—Where the fireplaces are "ventilating grates," such as Galton's or the Manchester Grate, the same quantities of air may be obtained with flues of less dimensions. For rectangular flues, the areas, taken in the same order as in the above table, would be:—0·54 sq. ft., 0·66, 0·81, 0·97, 1·2, 1·4, 1·6. For cylindrical flues:—1·5, 1·8, 2·3, 2·7, 3·3, 3·9, 4·6 sq. ft. The ventilating grates not only extract the air, but also admit air, and warm the air admitted. Consequently, a ventilating grate will maintain a circulation of air in a room that has no other outlet or inlet.

Ventilating gas-stoves, such as Bond's Euthermic or George's Calorigen, are designed to fulfil much the same functions as the ventilating grates, and for halls, lobbies, consulting-rooms, and the like, are extremely convenient. Why ventilating grates and stoves (coal, gas, and oil)—though suggested by Dr. Neil Arnott two generations ago—have not yet become as common as open grates can be explained only by the inertia of unscientific habits of house-building, and the popular craze for non-essentials in a house. Already, however, there are several good patterns in the market. (*See* Notter and Firth, 2nd ed., p. 239 *et seq.*)

(*c*) *Ventilating Shafts.*—These, unless fitted with an extraction fan or steam-jet or other motor, depend for their efficiency on three circumstances—the aspirating effect of air-currents across the top, the height of the shaft, and the difference between the temperature within the shaft and the temperature without. All three factors operate in the ordinary chimney with fire-place, where the difference in temperature is created by the fire. Gas-jets or hot-pipes may be made to serve the same purpose. The velocity of the air-current may be calculated from the height of the outlet opening above the inlet opening, when the temperature within and without the shaft is known. The engineering tables show the velocities corresponding to given heights of shaft at given temperatures. Thus, with a shaft 30 feet high, where excess of temperature in shaft is 50° Fahr. above external air, the velocity of air-current will be 14 feet per second.

(*d*) *Cowls.*—In so-called "natural" ventilation, which is only the general case of which a ventilating shaft is the special, cowls are sometimes placed on the outlet. The design usually is to "increase the draught"; but, as shown above, the draught depends on the head of air produced by temperature, or aspiration by wind, etc. No cowl has an active effect. The chief value of a cowl is to prevent foreign bodies, or birds, or rain, or snow, from entering the shaft. When wind is blowing, they prevent down-draughts, provided the head of air at the lower end of the shaft is not greater. To suppose that they always cause up-draughts, and always prevent down-draughts, is neither good observation nor sound theory. The varieties of cowl are numbered by the score. Some are rotatory, being fitted with an Archimedean screw; they are active in a high wind, when they are useless, and stationary and obstructive in a calm, when they are wanted. Some are so moved by a vane as to keep the opening always on the lee side, so assisting extraction of air. Some are static, as in Boyle's or Donald's or Buchan's. The openings are so flanged that the wind from any direction causes aspiration upwards. Hellyer found Buchan's the most effective. Billings prefers the simple conical cap, or rather truncated cone, with fixed plate above; it is one of the oldest forms, and one of the cheapest; it gives the greatest velocity, and offers least resistance. The top plate prevents in-dropping of rain or other foreign matter.

(*e*) *Inlets and Outlets.*—For inlets, the trumpet-shape, with large end inwards, secures better diffusion than a tube of uniform calibre. When a grating covers the inner end, the holes should be bevelled on the side *from* which the air is blowing. As to size, inlets should be "capable of being one and a half times as great as the outlet" (Shaw). This secures less friction and less draughtiness. As to structure, inlets should be as unobstructed, either by bends or gratings, as possible. A right-angled bend on an inlet duct reduces the velocity of the incoming air by about 50 per cent. Inlets should be adapted for regulation. Thus Sheringham's valve, which is shaped to direct the air upwards, can be closed or opened to any degree. A double-sash window can, by pushing up lower sash and blocking the lower opening, be made to admit air upwards between the sashes (Hinckes-Bird). But shape or direction is less important than position. Galton's grate is a good type of well-placed inlet and outlet. The inlet admits warmed air near the ceiling; the air diffuses easily in the upper strata, passes to the far side of the room, descends, runs along towards fire, and then up the flue. Inlet and outlet are on the same side of the room, and this arrangement secures the best diffusion, provided other openings do not interfere. If air be admitted at floor level, it should be warmed. Forms of inlets are:—Sheringham's valve; Steven's drawer, which is much the same in principle; Jenning's air-brick, placed near ceiling; Mackinnel's ventilator, which consists of an inner tube within an outer, the inner taking heated air from ceiling, the outer admitting cool air; Tobin's tube, which admits air from ground level, and carries it along duct vertically to any height within the room. Of the last, the primary defect is the taking of air from ground level. Dust is apt to lodge in the tube, which is usually unheated and too long. Tobin's tubes are best made of metal, which heats more readily to room temperature. Steam-

or water-heated radiators, placed opposite simple openings at floor level, secure good ventilation, *e.g.* in hospitals. Mixing boxes (Jacob's and others) have been designed for warming air on admission. Inlets may be placed behind cornices or pictures. They should, as a rule, be placed more than 6 feet high. In mechanical systems of ventilation, the ratio in size of inlets and outlets must be adjusted to the special system, —the quantity of air, the velocity, etc.

Outlets.—These have been already indicated. A fire-flue is the chief domestic outlet. As a general principle, outlets should be reduced where possible to one; friction will be less, there will be less risk of contrary action between outlets, and efficiency can be better regulated. In exhaustion systems a fan or steam-jet may be placed in the outlet. Sometimes, as in one form of Arnott's valve, an opening is made into the chimney at ceiling level, the opening being guarded by a mica flap-valve to prevent regurgitation of smoke. This is good in certain forms of room, as where windows or doors are the main inlets. Sometimes a flue for air is made vertically parallel with chimney, with an opening at ceiling level. They act well, provided inlets are sufficient. Mica flap-valves are noisy in windy weather.

(B) Plenum or Propulsion Systems

In a Plenum system, the air is forced into the rooms by mechanical power—rotating fans, "fanners," or blowers—or by steam-heated coils, stoves, furnaces, water-spray, steam-jets, or other heat-producing appliances. The Galton grate is at once a vacuum and a plenum appliance, forcing in warmed air and exhausting fouled air. In America the heating furnace for forcing in hot air is a very common house-fitting.

For large rooms, or buildings like schools, factories, hospitals, and the like, where cubic space per head is necessarily small, a mechanical system of Plenum ventilation is frequently the best. For large schools, it has emerged as the best. But its suitability depends on the skill of the designing engineer. A Plenum system (Key's) may be seen at work in many Scottish schools: in particular—Aberdeen Board Schools, Dundee Board Schools, the Leith High School, the Victoria Hospital (Glasgow), the Govan Parochial Hospital, and elsewhere. In Key's system, the parts are arranged in the following sequence: — Inlet, louvred or otherwise protected; screen for washing and filtering the air; forest of steam-heated pipes, arranged in sections or groups for greater or less heating; rotary fan, driven by steam, or oil, or gas, or water, or electricity; horizontal main air-duct; horizontal branch-ducts; vertical sub-branch ducts, with doors and extra heating-coils; openings near ceiling in rooms; outlet near floor; vertical duct, opening to outside. The air drawn or driven in by the fan passes through all those structures in the order given. The walls and windows are hermetically sealed. The air can be regulated in amount, in temperature, and in humidity. The system requires the attention of a competent mechanic. It is at once a system of ventilation and a system of heating. A special feature of the system is the placing of inlets and outlets on the same side of the room —the inlet near the ceiling, the outlet at or in the floor. In winter, the outlet at the floor level is found best; in summer, the outlet near the ceiling is found best (Billings).

The system is costly, both in installation and in use; but when properly arranged and properly managed, it is efficient. There are many other varieties of mechanical systems, *e.g.* the Sturtevant system, the Blackman system (Blackman's fan is commonly used in Key's system), and so on. As already noted, mechanical power can be applied for extraction as well as for propulsion.

Carnelley and Haldane, after an elaborate investigation, decided in favour of mechanical ventilation of schools. The comparative efficiency of "mechanical" and "natural" (or temperature ventilation—shafts, cowls, chimneys, etc.) is shown below, the mechanical system being taken as unity:—

Excess over Outside Air.	Dundee Schools (Carnelley, Haldane, and Anderson).		Aberdeen Schools (Brazier, Thomson).	
	Mechanically Ventilated.	Naturally Ventilated.	Mechanically Ventilated.	Naturally Ventilated.
Temperature .	1	0·66	1	0·9
Carbonic acid .	1	1·7	1	1·6
Organic matter .	1	7·0	—	—
Micro-organisms .	1	9·2	1	7·2

These results leave no doubt of the superiority of the mechanical systems. "By mechanical ventilation the whole of the air in a school may be easily changed in less than fifteen minutes, and when the system is well arranged, in less than ten minutes." The same observers prefer "blowing in" (propulsion) to "exhausting" of air, because in the propulsion system the source of the air admitted can be better controlled and the air better distributed.

Notes on Warming.—The following notes are supplementary to the details given under "Ventilation."

(1) *Open Fires.*—Advantages are—cheerfulness and good extraction of air. Defects are—great wastefulness (about 80 to 90 per cent) of fuel and production of floor draughts. In meeting these defects, very few of the many devices are entirely successful. A good grate should have little or no iron in its structure. The ribs should be thin and vertical, not thick

and horizontal. The back and sides should be of fire-clay. The sides should make an angle of about 120 degrees with the back; a line at right angles to the posterior edge of one side should just clear the anterior edge of the other; the heat from each side will thus be radiated into the room. The back should overhang, so that upper margin at chimney throat should be nearly as far forward as margin of grate bottom. If practicable, the grate should stand forward into the room. The air should circulate round it. A movable damper or regulator should control the chimney throat. The slits between the bars of the bottom grid should be narrow. The passage of air through them should be controlled by a tight-closing ash-pan or a "Teale's economiser." A still simpler method of control is a piece of sheet-iron slid in to cover the bars. This makes the fire burn more slowly, and can be used as a regulator for almost any grate.

The following patterns realise these conditions in various degrees:—The Nautilus grate, which stands out in the room, slow or quick combustion, chimney-throat regulator. The Galton grate, or stove, already described. The Grundy grate, fire-clay back, same principle as Galton's. The Teale grate, air-chamber below grate, no air admitted through bottom grid, very thin vertical bars in front, other conditions as above enumerated, two patterns, ordinary and front-hob, air in latter admitted at fire level; these patterns common in Yorkshire. The Rational grate, sunk ash-pit. Boyd's grate, same as Teale's with adjustable canopy at chimney-throat, regulating ash-pit, no iron parts; air circulates round whole grate. Heim's "Helios" smoke-consuming grates, fire enclosed, shines through mica, very economical, less cheerful than open fire, but convertible into open fire at will. Heim's "Hestia" stove, smokeless, economical, suitable for halls, etc. Neil Arnott's grate, practically smokeless, fuel burns from above downwards, coals put in first, lit from top, bottom movable upwards by capstan mechanism, chimney-throat valve, very economical, needs almost no attention for six to twelve hours continuously. Arnott's stove, double-cased, fed from top, anthracite coke, automatic air-valve, burns from twelve to twenty-four hours without attention, very economical, suitable for entrance halls, may be used as ventilator also. Arnott suggested opening into chimney at ceiling level for hot foul air removal. Falkirk Iron Company's "controlled combustion air-chamber heating apparatus," claims to heat room of 40,000 cubic feet sufficiently (with gas-coke, 2 lbs. per hour), at less than twopence per hour. Shorland's grate, ventilating, same principle as Galton's, common in hospitals. Shorland's calorigen, for heating rooms from under floor level.

(2) *Stoves.*— Advantage: greater economy; defect: less cheerfulness, danger of carbon monoxide formation from coke and coal stoves. For good patterns of coal or coke stove, see under (1). Oil (paraffin or petroleum) stoves are convenient for small rooms. A flue is always advisable. Gas-stoves should always have a flue. Many varieties. Best are radiant heat and ventilating forms. The "ball-fire" fitted with gas-burner, in ordinary grate, very wasteful. When stove is fitted, chimney access should be perfectly closed with sheet-iron, containing opening only for flue. All ball-fires waste heat by inter-radiation among the irregular balls (*see* Thos. Fletcher, *Coal-gas as Fuel*). Many patterns provided by gas companies; advisable to test by experiment before selection.

(3) *Heating by Steam, by Hot Water, and by Combination.* — Hot-water systems may work with high pressure (*e.g.* Perkin's patent) or with low pressure. In the former, the pipes are of wrought iron and small, about 1½ inch diameter; in the latter, about 3 or 4 inches diameter. Length of pipe necessary for given size of room, calculated by experiment. Thus Hood found that, with temperature of pipe at 200° Fahr., and temperature of external air at 40° Fahr., it is necessary, in order to heat 1000 cubic feet per minute up to 60° Fahr., to have at least 80 feet of 4-inch piping. Hood's tables give too low estimates. Radiators are an important factor. They vary in efficiency (*see* Carpenter, *Heating and Ventilating Buildings*). When steam is available, it may be used to heat a "calorifier" (spiral condensing tube passing through water). This in turn may be used to form low-pressure circulation. Waste steam frequently available. Heat liberated on condensation proportionally very great. All these systems may be applied to warm the admitted air. Important to warm the walls (*see* Galton's *Healthy Dwellings*, p. 184 *et seq.*)

Ventnor. *See* THERAPEUTICS, HEALTH RESORTS (*English*).

Ventose.—A cupping-glass; also, as an adjective, flatulent.

Ventrad.—Directed towards the ventral or abdominal surface of the body.

Ventral.—Related to the abdomen, belly, or venter, or, in its widest sense, to the parts farthest away from the vertebral column; *e.g.* a ventral hernia (*see* HERNIA, *Ventral*), ventral fixation of the uterus (*see* UTERUS, DISPLACEMENTS OF, *Prolapse, Operative Treatment*).

Ventricle.—The hollow part of an organ such as the brain, the heart, or the larynx; formerly it had the meaning of stomach or uterus (Lat. *ventriculus*, diminutive of *venter*, the stomach). *See* BRAIN, PHYSIOLOGY OF (*Lymphatic Circulation*); HYDROCEPHALUS; MENIN-

GITIS, TUBERCULOUS AND POSTERIOR BASIC (*Treatment, Surgical*); VERGA.

Ventrofixation or Ventrosuspension.—Ventral fixation or ventrohysteropexy. *See* UTERUS, DISPLACEMENTS OF (*Prolapse, Operative Treatment*).

Veratrine.—An alkaloid derived from the dried seeds of *Schœnocaulon officinale*. It is a greyish powder, odourless, but very irritating to the nostrils and causing prolonged sneezing. *Dose*—$\frac{1}{70}$-$\frac{1}{10}$ gr. in pill. *Preparation*—Unguentum Veratrinæ. It has an exceedingly toxic effect on the heart and nervous system, and is rarely given internally. Externally, in the form of the ointment, it is used for neuralgia, muscular rheumatism, and articular rheumatism. *See also* ALKALOIDS (*Veratrina*); MUSCLES, DISEASES OF THE (*Myotonia Congenita, Resemblance to poisoning by Veratria*); PHYSIOLOGY, TISSUES (*Muscle, Contraction*); *Spasm* (*Causes, Veratrine*); TOXICOLOGY (*Hellebore*).

Veratrum Viride.—American hellebore; it contains jeroin and veratroidin (alkaloids), and its action is due to their combined effect (slowing of the pulse, lessening of blood pressure, and vomiting); it is recommended in eclampsia gravidarum (*see* ECLAMPSIA), in sthenic pneumonia and peritonitis, etc.; it is given in the form of the tincture (dose, 1 to 3 m.) or of the fluid extract.

Verbigeration.—The frequent repetition of a word, or group of words, or simply of a sound, occurring as a symptom of insanity. *See* INSANITY, NATURE AND SYMPTOMS (*Speech*).

Verga's Ventricle.—When the psalterium is not entirely fused to the under surface of the corpus callosum, a small cleft-like space is left between them, known as Verga's ventricle.

Verheyen's Stars.—Stellate venous plexuses seen on the surface of the kidney, under the capsule.

Vermicelli. *See* DIET (*Vegetable Foods, Cereals*).

Vermicides. *See* ANTHELMINTICS; PARASITES (*Cestodes*); PHARMACOLOGY.

Vermiform.—Having the shape of a worm, *e.g.* the vermiform appendix of the intestine and the vermiform processes of the middle lobe of the cerebellum. *See* APPENDIX VERMIFORMIS.

Vermifuges. *See* ANTHELMINTICS; PARASITES (*Cestodes*); PHARMACOLOGY.

Vermination.—The state of being infested by worms.

Vermin Paste. *See* TRADES, DANGEROUS (*Phosphorus*).

Vermis.—A worm, and so, in Descriptive Anatomy, a worm-shaped structure, such as the middle lobe of the cerebellum. *See* PARASITES; PHYSIOLOGY, NERVOUS SYSTEM (*Cerebellum, Structure*).

Vernet-les-Bains. *See* BALNEOLOGY (*France, Sulphur Waters*).

Vernix Caseosa.—The sticky deposit found on the skin of the new-born infant, especially in the flexures of the limbs, back of the neck, etc., consisting of the secretion of the sebaceous and sweat glands and some shed epithelium and hairs; its literal meaning is "cheesy varnish." *See* NEW-BORN INFANT (*Physiological Peculiarities, Management, Bath*).

Veronal.—Diethyl malonyl urea, a hypnotic drug which has been widely used for the production of sleep in cases unaccompanied by pain; it has been given in doses 5 to 10 or even 20 grains, but it appears to have a cumulative action and may sometimes be followed by toxic effects (rashes, diuretic action, etc.); the conditions which have been regarded as indications for its use are insomnia, seasickness, hyperemesis gravidarum, and phthisis (for insomnia and night-sweats); it is also used to prevent chloroform sickness and the unpleasant effects of morphine.

Verruca.—A wart. *See* SKIN, TUBERCULOSIS OF (*Verruca necrogenica*); WARTS OR VERRUCÆ; etc.

Verrücktheit.—The German equivalent for *paranoia* (*q.v.*).

Verruga Peruana. *See also* SKIN DISEASES OF THE TROPICS (*Constitutional Infective Diseases*).—SYNONYMS.—Verruga Andicola, Peruvian wart, bouton des Andes, la maladie de Carrion, fievre delà Oroya, fièvre maligne de "las quebradas."

Definition.—A general infective disease, peculiarly limited in distribution, and usually marked by anæmia, severe constitutional disturbance, and the subsequent development of a characteristic skin eruption.

History.—As far back as the sixteenth century verruga apparently attacked the Spanish soldiers at Coaque (Ecuador) and in Puerto Viego and Cusco, but of late years it has never established itself outside certain deep-cleft inland valleys on the western slopes of the Peruvian Andes in the departments of Ancachs, Lima, and Libertad, between 8 and 13 degrees of latitude, and at elevations of 1400 to 9000 feet. Although Haviland Hall heard from Zaruma (Ecuador) of a mild outbreak of very similar disease, and Beaumanoir reported a solitary case in Reunion, in neither case did it spread. In 1870 the out-

break of fever among the Trans-Andean railway constructors, which Espinal suggested was a phase of verruga, aroused fresh interest; and in 1885 Carrion, a Lima student, threw additional light on the subject by inoculating himself with blood from a verrugous tumour, and producing a fatal disease strongly resembling this "Oroya" fever. His experiment gave strong evidence in favour of the identity of the two diseases (a view supported by clinical experience), and also went far to prove the inoculability of verruga.

Etiology. — That verruga is inoculable is certain, but it cannot be highly contagious, as cases are treated in general wards. The very young, if attacked, are apt to suffer from the graver forms, whereas the elderly enjoy a certain immunity from these (Odriozola), but sex has no influence. Unlike yaws, the disease is by no means confined to coloured races, but the actual inhabitants of the endemic areas acquire some immunity, while the new arrival is particularly liable to suffer. Malarial infection frequently obscures the clinical picture, but has no etiological significance. Like that disease, however, but more markedly, verruga is confined to certain districts, while its outbreaks bear distinct relation to soil disturbance, and coincide with the drying of the soil after floods. The endemic valleys have rocky sides and earthy bottoms, and the streams which traverse them overflow twice a year. From the stillness of the atmosphere and the high temperature, their vegetation is tropical even at considerable altitudes. The water itself has often been suggested as, and is popularly supposed to be, the source of infection, but is probably guiltless, although its floods no doubt exercise a subsidiary influence. The connection with soil-disturbance and frequent occurrence in soil-workers suggest a telluric factor in causation; and Firth, on account of the prevalence of helminthiasis, mentions the possibility of an association with some worm passing its free state in mud. From the peculiar limitation, some local condition, perhaps the physical features, evidently plays an important part in development; and the infrequency, mildness, and apparent inability to spread of cases occurring elsewhere point the same way. An organism seems by far the most probable cause, and the limitations of tsetse-fly disease, and the recently discovered connection of malaria with mosquitoes, suggest the possibility of a somewhat analogous conveyance of infection by insects peculiar to these districts.

Symptoms.—The incubation period is not yet accurately determined, and may last from a week to two months or perhaps more: Odriozola gives the average as about forty days. Vague pains, chills, and alimentary disturbances, with gradually increasing anæmia, enfeeblement, and slight nocturnal fever, constitute the common prodromal symptoms. Of the developed disease there are two manifestations: the constitutional disturbance and the eruption. Both usually occur, but at times the first is hardly noticeable, and, on the other hand, is occasionally so profound that death ensues without any appearance of eruption, constituting the grave form or Oroya fever. If the eruption disappears too rapidly, very similar symptoms may supervene, even without internal verrugæ. The onset is usually marked by chills and irregular, remittent, or intermittent fever, often accompanied by profuse sweats. There is profound prostration, anorexia, thirst, and frequently marked gastric irritability and hiccough. The tongue is pale and flabby. More characteristic, however, are the extreme and rapidly produced anæmia, the intense headache, muscular cramps, and bone and joint pains, and the frequent tenderness and enlargement of liver, spleen, and lymphatic glands. The red cells and hæmoglobin are diminished, and there is a leucocytosis. From anæmia there results pallor, or (from blood destruction) an icteric complexion, marked vertigo, or even syncope, cardiac murmurs, quickened pulse and respiration, œdema and tinnitus, insomnia and dreams. The pains, dull or acute, are worse at night, and especially involve the extremities. Very rarely there is slight joint-swelling. Cases are often complicated by hæmorrhages, particularly epistaxis or petechiæ, serous diarrhœa, or pulmonary congestion. With gradual fall of temperature the general condition may improve and recovery follow, but severe cases proceed with continual or occasional falling temperature, increased restlessness and low delirium, and end in coma and death, hastened perhaps by pulmonary or intestinal complications.

Eruptive Phase. — Usually this supervenes after an interval of one or two months marked by vague "reminders," such as pains or slight fevers. It may, however, coincide, or to some extent alternate, with the general symptoms, or even precede them. Its time of appearance, duration, and degree of exuberance bear distinct relation to the severity of attack, and when scanty or delayed render prognosis graver. Constitutional manifestations are less severe than in non-eruptive cases, and lymphatic enlargement, vertigo, and vomiting are rare, but œdema (resulting from the eruption) is more marked. The clinical features may, however, be altered by the development of verrugæ in internal organs. The two types of eruption may occur separately or together. The smaller (superficial) commence in the superficial layers of the skin as petechiæ, shining transparent spots, little horny elevations, or rarely as vesicles or pustules (odriozala), and enlarge into little reddish shining tumours from a pea to an orange in size. They are sessile or pedunculated, discrete or confluent, localised or general, and

usually appear in crops. The limbs, face, and neck are chiefly involved; the trunk, palms, and soles being less liable. Prickling sensations sometimes precede them, and itching is often extreme. Many are very vascular and readily bleed; others may ulcerate and exude a brownish fluid, which forms a crust under which they heal, or they gradually pale, desquamate, and disappear. Not only do they involve the skin, but also the conjunctiva, any part of the alimentary or respiratory mucous membranes, the connective tissue of the parenchymatous organs, the muscles, periosteum, or bones, and even the meninges and nervous centres.

The larger (subcutaneous) forms are never found internally, and commence in the subcutaneous connective tissue. Invisible but palpable nodules appear, which become adherent to the skin, causing reddening and tenderness and finally rupturing, and appear as reddish painful tumours with offensive discharge, and spherical, conical, mushroom-like, or irregular in shape. Their size varies from that of a nut to that of an apple, and they are sessile or pedunculated. Vascularity is a marked feature, and hæmorrhages, sometimes dangerous and always increasing the anæmia, are not infrequent. Rarely the initial swelling simulates fluctuation, but from the entry of pyogenic organisms actual suppurative foci, ulceration, and even gangrene may arise. The growths have an especial predilection for the face, knee, and back of hands, especially over the metacarpo-phalangeal joints of thumb and index finger; the trunk is, again, less liable.

Course and Sequelæ.—This stage of eruption generally lasts four to six months, but with intermissions may even last two years or more. Generalised cases are usually more rapid. In most cases the growths shrivel or ulcerate and gradually disappear, but considerable anæmia and debility are left, and neuralgias are often troublesome. Profuse hæmorrhages, entry of pathogenic organisms, or some other complication, may cause death, and the mortality at periods of exceptional prevalence is great. Although one attack is supposed to confer immunity, recurrences have been reported.

Morbid Anatomy.—In non-eruptive cases the dominant features are the anæmic condition (with the exception of localised congestions of lung or elsewhere) of all organs, and the fluid and slowly coagulating blood. The liver, from destruction of red cells, is frequently dark coloured, the lymphatic and mesenteric glands and Peyer's patches are enlarged, and there are signs of activity in the bone marrow.

The tumours arise from the connective tissue of the skin or elsewhere, and are essentially composed of a mass of embryonic cells with large nuclei, some polynuclear leucocytes, and a few fixed cells, supported by a delicate fibrous stroma. The epidermis over them is infiltrated with leucocytes, and in the ulcerated cases foci of suppuration are seen. The vessels are often large, numerous, and distended with blood; they may appear as if hollowed out of the tumour mass, and small hæmorrhagic foci may be present. According to Letulle, the lymphatics are dilated; and Nicolle, contrary to the general experience, claims to have found giant-cells and caseation in internal verrugæ.

Bacteriology.—Bacilli have been found by Isquierdo and Letulle in the depths of the non-ulcerated skin tumours, and by Nicolle in internal verrugæ. They somewhat resemble the tubercle bacillus, and, after prolonged staining, resist, as it does, the decolorising action of acids. They are sometimes intracellular (Nicolle). Various kinds of pyogenic cocci, probably from secondary infection, have also been found in the ulcerative lesions. Florez obtained chain cultures of cocci by inoculating agar-agar with verrugous blood; and Odriozola found in the grave febrile forms a very short bacillus, only colourable by Loeffler's stain, until after cultivation in bouillon, when it was larger and stained readily. Up to the present, however, all attempted inoculations of animals with these organisms have failed, and the bacteriology is therefore indefinite.

Diagnosis.—This presents little difficulty except in non-eruptive cases or in the pre-eruptive stage. In Peru, residence in the endemic districts must always be carefully inquired into. Malarial fevers, which are frequently simulated, are best distinguished by blood examination; but the presence of the plasmodium is not conclusive against verruga, as the diseases sometimes co-exist. The inefficacy of quinine, the more rapidly produced anæmia, the glandular enlargement and vertigo, with the localised pains and less marked splenic involvement, are therefore additional distinctions. Pernicious anæmia, ankylostomiasis, rheumatism, and acute hepatitis can be readily distinguished. The eruption resembles in many respects that of yaws; but the latter is a disease of low lands, attacks coloured races almost exclusively, is more contagious, has no local limitations, and is acquired quite differently. The general symptoms, too, are hardly noticeable, the growths are never found internally, and it is chiefly prevalent in early youth.

Lastly, unlike yaws, animals can be inoculated with verruga, and suffer from a similar disease; and there are cases recorded of apparent infection "in utero," which is at least doubtful in yaws.

Prognosis.—With severe constitutional symptoms and repressed eruption, especially with a comparatively low temperature, or where pulmonary complications supervene, prognosis is extremely grave. So also are cases with symptoms pointing to verrugæ in important organs, while the most favourable cases are those with

early development and gradual retrogression of eruption with coincident general improvement.

Prophylaxis.—This consists in the avoidance of endemic areas by new-comers, especially at periods of prevalence and when the soil is being disturbed.

Treatment.—Removal, if possible, from the endemic area and to a warm place at sea-level is the first indication, as cold represses the eruption, and low barometric pressure favours hæmorrhage. In severe cases diet should be nourishing and easily digested, and drugs by the mouth avoided, if possible, in order to prevent disturbance of digestive function. There is no specific treatment, but Odriozola recommends the administration in milder cases of arsenic and iron. He also thinks a decoction of maize a useful diaphoretic, and recommends oxygen inhalations and, for the obstinate headache, iced compresses. Cold bathing is contra-indicated, but the skin must be kept clean and aseptic, and any ulcerating or gangrenous growths ligatured or excised. Complications must be treated on ordinary principles, but styptics, compresses, and transfusion apparatus should be at hand in case of hæmorrhage.

Version. *See* LABOUR, OBSTETRIC OPERATIONS (*Version*); LABOUR, DIAGNOSIS AND MECHANISM (*Transverse Lies*); PREGNANCY, HÆMORRHAGE (*Accidental and Unavoidable, Treatment*); etc.

Vertebra.—One of the bones composing the spinal or vertebral column; it is cervical, dorsal, lumbar, sacral, or coccygeal according to the region in which it is situated. *See* SPINA BIFIDA; SPINE, SURGICAL AFFECTIONS OF; etc.

Vertebral.—Belonging to a vertebra or to the spinal column, *e g.* the vertebral artery. *See* ANEURYSM (*Vertebral Artery*); ARTERIES, LIGATURE OF (*Vertebral*).

Vertex.—The top or crown of the head, especially in relation to the part which presents or comes first in normal labour. *See* LABOUR, PHYSIOLOGY OF (*Factors, Passenger*).

Vertigo. *See also* AUDITORY NERVE AND LABYRINTH (*Aural Vertigo, Ménière's Disease*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Anæmia, Symptoms*); BRAIN, TUMOURS OF (*Symptoms*); BRAIN, CEREBELLUM, AFFECTIONS OF (*Experimental Physiology*); EAR, EXAMINATION OF (*Symptomatology, Vertigo*); EQUILIBRIUM; GOUT (*Irregular, Nervous System*); HYSTERIA (*Infantile*); LARYNX, NEUROSES OF (*Laryngeal Vertigo*); PHYSIOLOGY, CEREBELLUM (*Functions*); STOMACH AND DUODENUM (*General Symptomatology, Vertigo*).—Except when the body is at rest and when equilibrium is maintained, as in any mass of inert matter, by the ordinary laws of gravity, equilibration is normally effected by muscular activity. The muscles specially concerned in the maintenance of equilibrium are those of the head, neck, spine, and lower limbs, all of which are innervated by the cerebral cortex, acting largely under the influence of the cerebellum.

The cerebellum is a co-ordinating centre to which are transmitted various centripetal stimuli, affording information as to the relation of the body to surrounding objects. The chief afferent impressions are (1) those from the semicircular canals of the internal ear, whose endolymph exercises a varying pressure upon the end-organs there, under the influence of movements of the head and of gravitation; (2) those from the skin of the parts on which the body is resting; (3) those from the muscles and joints concerned in maintaining the posture, especially the muscles of the spine and lower limbs; (4) those from the muscles moving the head and eyes, which indicate the relation to the body of objects seen. The cerebellum, receiving impressions from all these various peripheral sources, exercises in turn an efferent action upon the cerebrum, and thence upon the various muscles which subserve equilibration. A direct influence of the cerebellum upon the spinal muscles has been described by some, the hypothetical cerebello-spinal paths being through the nuclei of Deiters, and thence by the vestibulo-spinal tracts to the cord. More probably, however, the cerebellum exerts its influence upon the muscles not directly, but by intermediation of the cerebral cortex. Each cerebellar hemisphere is intimately connected with the contra-lateral motor cortex by means of the corresponding superior cerebellar peduncle, leading from the cerebellar cortex and dentate nucleus to the contra-lateral red nucleus and optic thalamus, and from both the latter onwards to the frontal lobe. That this is the true mechanism is rendered still more probable by the recent researches of Pagano, who, on stimulating one lateral lobe of the cerebellum, obtained movements of the homo-lateral trunk muscles. Such movements were abolished if the contra-lateral motor areas of the cerebral cortex were extirpated.

By vertigo or giddiness is meant that disagreeable sensation which results if the sense of equilibrium is disturbed. The motor act of normal equilibration, once acquired, does not form part of our ordinary consciousness. When an individual has once learned to balance himself, whether standing, sitting, walking, riding, skating, etc., the movements necessary for maintaining that particular posture are, generally speaking, carried out automatically or subconsciously by menas of the mechanism already described. It is only when some flaw occurs in this cerebellar mechanism or in the higher centres that the individual becomes aware that something is wrong. The sensation of giddiness then at once arises. This sensation may be accompanied by movement or sense of

movement either in the individual himself or in external objects. Reeling or staggering, it should be remembered, is not synonymous with giddiness, but is a motor phenomenon, representing the individual's effort to preserve his equilibrium in the presence of abnormal sensations.

Vertigo may result from affection either of the higher cerebral or of the subordinate cerebellar centres, or it may be due to interference with the normal peripheral impressions to which reference has already been made. But whatever be its cause, whether central or peripheral, the individual does not, as a rule, take cognisance of the particular source to which his vertigo is due. All that he notices is the result upon his consciousness, namely, that he feels giddy. Whether the giddiness be due to labyrinthine, ocular, or other peripheral defects, to gross cerebral or cerebellar disease, or to some toxic affection of the centres, is a problem for the physician to solve.

When vertigo is severe, nausea and vomiting tend to become superadded, as in the familiar instance of sea-sickness. Such vomiting has been ascribed by some to connection between the cerebellum and the gastric fibres of the vagus, whilst others trace it to the connection between the auditory and vagal nuclei in the bulb. It may also be due to disturbance of still higher centres, the same centres which perceive the giddiness. It is, in any case, a true cerebral vomiting.

Considerable attention has been paid to the question of whether there is a sensation of movement in the individual himself—so-called "subjective" vertigo; or whether the apparent movement is in surrounding objects, the individual himself not seeming to move—"objective" vertigo. "The appearance of movement in stationary bodies," says Dr. Hughlings Jackson, "must, as a sensation, depend upon movements of the patient's eyes, occurring involuntarily, and leading to erroneous visual judgments. Where the mental impression is that of movement of the patient's own body, the explanation is not so obvious."

In some cases of vertigo, surrounding objects seem as if they were revolving round the patient, either horizontally to one side or the other, or vertically up or down. The direction generally corresponds to that in which the patient feels himself moving. But sometimes objects appear to move in the opposite direction to that of the subjective movement, as in the vertigo of waltzing, probably because of the direction of movement of images across the retina. In still other cases, surrounding objects seem to move in directions which do not in any way correspond with the subjective vertigo, as, for example, when objects seem to advance and recede, or when each object appears to rotate round its own axis. The explanation of such cases is obscure.

Vertigo in Healthy People.—Before passing on to the various diseased conditions associated with giddiness, a brief reference may be made to certain varieties which scarcely fall within the category of pathology. Thus, for example, the giddiness produced by the passage of a galvanic current transversely through the head is probably referable to stimulation of the cerebellum. The curious fact is observed that, on closure of the current and during its flow, the individual tends to fall towards the side of the anode, whilst on opening the current he falls towards the kathode. Stronger currents produce nystagmus in addition, the direction of nystagmus during the current being towards the anode. Another variety of giddiness is that which results from rapid rotation of the body around its own axis, as in waltzing, or associated with sudden changes in our relation to space, as when on a swing or switchback. Such giddiness is probably due to rapid reversal of pressure in the endolymph when the direction of movement suddenly changes. The giddiness which some people experience on stepping unexpectedly from firm ground on to a somewhat boggy piece of moorland turf is probably due to sudden deficiency in the sensation of resistance from the skin of the soles and the muscles of the lower limbs. The vertigo which affects certain individuals when on "giddy" heights—so-called tower- or cliff-giddiness—is probably attributable largely to the loss of normal afferent impressions from the muscles of the head and eyes. Ordinarily we are accustomed, by movements of the head and eyes, to compare our position in space with that of surrounding objects at our own or a higher level, and when such objects are absent, vertigo results. Closing the eyes removes the symptom.

Diseases associated with Vertigo.—Many pathological intracranial conditions are associated with vertigo. Thus it occurs in head-injuries, slight or severe, and in cases of sudden alterations of intracranial circulation, whether in the form of anæmia or hyperæmia. The symptom is very common in women during the menopause. It is still more evident in cases of intracranial hæmorrhage or softening, and may be a premonitory symptom of various vascular lesions of the brain. Old people with atheromatous cerebral vessels often complain of giddiness, and if this be associated with headache, and especially if there be no evidence of labyrinthine affection, its significance should never be minimised. Vertigo also occurs in chronic as well as in acute increase of intracranial pressure; thus it is present in many cases of cerebral tumour, and is especially severe when the growth is in the region of the cerebellum or of its middle peduncle. In other diseases of the central nervous system, more especially in disseminated sclerosis, attacks of vertigo are common. *Epileptic vertigo* is a

familiar variety, occurring either as the aura of a major attack or, it may be, constituting the chief evidence of the epileptic attack, in which the patient becomes suddenly giddy, falls to the ground, and may be unconscious. The writer has met with a case in which a young adult, after a single attack of "grand mal," was subject for several months to sudden attacks of momentary giddiness associated with overpowering fear, but without loss of consciousness. Vertigo is also met with in some cases of migraine, and in them, according to Dr. Liveing, it occurs after the visual phenomena and simultaneously with or following the headache. Sometimes vertigo replaces the attacks of migraine. Such cases are distinguished from the paroxysmal auditory vertigo about to be described, by the history of previous attacks of migraine and by the absence of tinnitus or deafness.

Certain poisons act upon the centres for equilibration. Amongst them the most familiar examples are alcohol and tobacco. Uræmic vertigo is to be included amongst the auto-intoxications, and probably so also are most cases of gastric vertigo, though some may be reflex, through stimulation of the vagus. *Gastric vertigo* is commoner in slight than in grave diseases of the stomach, and is associated with the ordinary symptoms of dyspepsia, such as pain and distension, perhaps vomiting. Sometimes the vertigo is suddenly relieved by gaseous eructation. But many so-called gastric cases on examination reveal some aural affection, so much so that Sir W. Gowers has questioned the existence of vertigo of purely gastric origin. Constipation is a frequent cause of giddiness, and it is a well-established fact that vertigo may sometimes be caused by intestinal parasites, disappearing at once when the latter are expelled.

Amongst other less common varieties may be mentioned nasal vertigo, which sometimes occurs in cases of recurring congestion of the turbinate bodies. Laryngeal vertigo, a somewhat doubtful variety, is said to occur in various laryngeal affections. Giddiness is often a symptom of neurasthenia and of hysteria, and in these affections it is frequently to be elicited by the slightest exciting cause; such, for example, is the probable explanation of the giddiness described as occurring on rectal examination or on compression of the testicle. A hereditary family form of giddiness has also been described.

Ocular Vertigo.—This variety, though somewhat rare, is quite definite. It is a form of giddiness occurring in cases of paralysis of any of the ocular muscles, and is associated with diplopia. The visual field being erroneously projected, the patient judges wrongly as to the relation of his body to objects seen. "Objects appear to be in certain positions where the patient's feet, as a matter of fact, fail to find them" (Hughlings Jackson). The giddiness in such cases is not due directly to the diplopia, for it persists when the sound eye is covered. (The condition can be imitated in health by closing one eye and displacing the other eye inwards by pressure with the finger, when, if the subject tries to walk along a straight line, the gait becomes very unsteady.) The explanation is to be found rather in the unusual effort on the part of the brain to turn the eyes in the direction of action of the paralysed muscle. Ocular vertigo, therefore, occurs only when the affected muscle is called into action. If the ocular palsy becomes permanent, the cerebral centres learn in time to neglect the error, and the giddiness then disappears.

Aural Vertigo.—Abnormalities of the auditory apparatus undoubtedly constitute by far the commonest cause of giddiness. Thus, for example, we may have conditions in the external auditory meatus producing irritation or obstruction with pressure on the tympanic membrane, as in accumulation of cerumen, foreign bodies in the meatus, or during ear-syringing, especially if a perforation of the drum be present. All these produce their effects by indirectly altering the pressure on the endolymph. Equally readily understood are the cases where the pathological process is in the middle ear, as in otitis media, obstruction of the Eustachian tube with diminished pressure, sneezing or blowing the nose, with consequent rise of pressure in the middle ear, also in paralysis of the stapedius or spasm of the tensor tympani muscle. Lastly, there is an important class of cases due to primary affection of the labyrinth itself without intermediation of the middle or outer ear. It is this particular variety to which Ménière called attention in 1861, and with which his name is commonly associated.

MÉNIÈRE'S DISEASE OR LABYRINTHINE VERTIGO has three main groups of symptoms. *Firstly*, That due to affection of the semicircular canals, viz. giddiness with its associated motor phenomena of reeling and sometimes abnormal ocular movements, the sensory stimulus in this case being transmitted along the vestibular root of the auditory nerve to the auditory nuclei (of which Deiters' nucleus is one) in the floor of the fourth ventricle, and thence to the cerebellum. *Secondly*, Auditory phenomena, tinnitus and deafness, due to affection of the ordinary path for hearing, along the cochlear or truly auditory part of the auditory nerve to the nucleus accessorius, thence along the striæ acusticæ, and ascending in the lemniscus upwards to the brain without intermediation of the cerebellum. *Thirdly*, Associated bulbar or "vital" phenomena, explainable by affection of adjacent medullary centres, consisting in cardiac, vaso-motor, and secretory signs in the form of pallor, prostration, and even syncope, cold clammy sweat, nausea, and vomiting.

The vertigo of Ménière's disease is paroxysmal, and comes on with such suddenness that the patient may fall to the ground as if struck down by an unseen hand. In other cases he reels, but has time to clutch at some neighbouring object to prevent himself from falling. He may either feel as if rotating himself or as if surrounding objects were whirling round him, or again as if the ground were sinking down under his feet. The giddiness lasts sometimes for hours; slighter attacks may pass off in a few minutes. It is increased by movement, and the slightest attempt to raise the head may induce vomiting. The direction to which the patient falls is usually forwards or towards one side, and this movement, whether real or apparent, is more commonly away from the side of the affected ear. In recurring attacks the direction of falling and of apparent movement of objects is usually the same. Not infrequently nystagmoid jerks of the eyes occur during the attack, and double vision has also been observed. Slight degrees of diplopia have been explained by Sir W. Gowers as possibly due to nystagmus of unequal degree in the two eyes, since no true ocular palsy exists. Momentary loss of consciousness sometimes, though rarely, occurs. The vertigo is frequently accompanied or followed by nausea and vomiting, lasting sometimes for hours. Headache is also commonly present. The patient is pale, and his skin covered with a cold, clammy sweat. Together with the symptoms already mentioned, and simultaneous with them, there are characteristic auditory phenomena, generally a sudden loud noise. This has been compared by patients to the whistling of an engine, the roar of the sea, or the discharge of a gun. It is usually unilateral. At the same time, if attention be paid to the point, more usually later, it is discovered that there is deafness, more or less complete, in the affected ear, with diminution or loss of bone-conduction.

As the attack passes off, the tinnitus becomes less intense, the pallor and sweating cease, whilst the giddiness, nausea, and vomiting tend to continue, though less severe, for hours or days. A certain degree of deafness usually remains, but is rarely absolute.

Such symptoms, occurring with apoplectiform suddenness, constitute the typical picture of Ménière's disease. But frequently the paroxysms are much slighter and unassociated with nausea or vomiting, so that the patient may simply have sudden transient giddiness. But the attacks, whether slight or severe, tend to recur; rarely does a patient escape with a single attack. The intervals between them vary from days to months or years; they may gradually decrease in frequency, or may progressively increase until, with successive attacks, the deafness becomes absolute, when the vertigo generally ceases. Between the attacks the patient sometimes feels a slight persistent giddiness, but not invariably. In a certain number of cases the attack occurs at night when the patient lies down, so that he has to sit up for a time until it passes off. Or it may even occur during sleep and wake the patient up.

The pathological investigations recorded in Ménière's disease are somewhat scanty. In Ménière's original series of cases the symptoms came on suddenly in patients previously free from ear trouble. More usually, however, the phenomena occur in patients the subjects of previous ear affection of some sort, often in the middle ear. But it should be borne in mind that auditory vertigo, *per se*, is referable to changes in the semicircular canals of the inner ear. The lesion there is probably most commonly of inflammatory nature. In a few cases it has been a direct extension from adjacent middle-ear inflammation; more often it is the result of general constitutional disease, especially gout and syphilis. In other cases the slowly progressive character of the symptoms point rather to degenerative changes in the labyrinth, either senile or associated with atheroma of the vessels of the inner ear, as has been demonstrated in one case. Actual hæmorrhage into the labyrinth, as described by Ménière, is undoubtedly rare, though it is the probable explanation of cases occurring in leukæmia or other bleeding diseases, also in some cases of chronic renal disease and in fractures in the base of the skull. Cases which have been recorded as supervening in tabes dorsalis are probably due to primary disease of the vestibular nerve.

The paroxysmal nature of the disease is more difficult to explain. Perhaps the labyrinthine disease causes a permanent instability of the equilibratory mechanism, so that it becomes acutely deranged by minimal superadded excitations, as, for example, by toxic, gastro-intestinal, or vaso-motor disturbances, or even by emotional excitement.

Four-fifths of the cases occur after the age of thirty, and the disease is twice as common in men as in women.

Diagnosis.—To those familiar with the phenomena of labyrinthine vertigo its diagnosis, as a rule, presents no difficulty. From ordinary epilepsy it is distinguished by the co-existence of vertigo, tinnitus, and deafness, and by the tendency for slight giddiness to persist between the attacks of severe vertigo. Loss of consciousness, which is the rule in epilepsy, is rare in labyrinthine vertigo. From cerebral hæmorrhage or softening the diagnosis is made by the presence of aural phenomena and the absence of signs of a focal brain lesion, either in the form of paralysis or affection of reflexes.

Paralysing vertigo or Gerlier's disease is a curious affection which occurs among cowherds in certain Swiss cantons. The symptoms con-

sist of sudden giddiness with ptosis, flaccid paresis of the muscles of the neck, legs, and sometimes of the hands, pain in the neck and occiput, and occasionally diplopia, transient amblyopia, difficulty in swallowing and in chewing. The affection has been attributed to toxic exhalations from the stalls, but its nature is obscure; possibly it is hysterical. The attacks occur several times a day, and between them the patient feels quite well. In any case the prognosis of the disease, which is known locally as "le tourniquet," is good, and the attacks after a time cease spontaneously.

The *treatment* of any individual case of vertigo depends upon the accurate diagnosis of its cause. Aural vertigo being by far the commonest form, it is the physician's duty in every case carefully to examine the ears. Vertigo dependent on cerebral hyperæmia is to be treated by appropriate depletory measures; whilst gastro-intestinal affections, if present, must be treated by careful regulation of diet, with suitable remedies to aid digestion. Intestinal parasites must be expelled. Even in cases of aural origin, attention to the digestive system must not be overlooked. Lack of food, according to Dr. Hughlings Jackson, is a factor in many cases of aural vertigo, and a patient may sometimes have an attack if he accidentally misses a meal. The beneficial effect of food in such cases is probably due to hyperæmia of the abdominal viscera, rendering the blood-supply to the rest of the body, including the brain, less abundant. Patients with Ménière's disease should therefore have a rather generous diet, and should have their meals at regular intervals. Where there is arterio-sclerosis, iodides should be exhibited. Gout and syphilis, if present, should be treated. Of all the drugs which benefit the giddiness, bromides, by their sedative action on the nerve centres, are the most efficacious. Any aural defect must of course, if possible, be remedied. Charcot recommended quinine in aural vertigo, others have recommended salicylates, both of these latter to be pushed until their physiological effects are produced upon the hearing. In neurasthenic cases, general measures should be employed for the constitutional condition, and may advantageously be combined with counter-irritation in the form of fly-blisters behind the ear.

Verumontanum. — A mesial ridge (Lat. *veru*, a spit; *montanus*, like a mountain) in the posterior wall of the prostatic part of the male urethra; the crista urethræ. *See* URETHRA, DISEASES OF (*Anatomy, Prostatic Portion*).

Vesalius, Foramen of.—A small canal occasionally found near the root of the great wing of the sphenoid bone; it transmits a small vein into the scaphoid fossa.

Vesalius, Ligament of.—Poupart's ligament (*q.v.*).

Vesania.—Madness or fury of mind (Lat. *ve*, privative; and *sanus*, sound). *See* INSANITY.

Vesical.—Relating to a bladder (Lat. *vesica*, the bladder), more especially the urinary bladder; *e.g.* the vesical arteries, the vesical ligaments, the vesical sphincter, etc. *See* BLADDER, INJURIES AND DISEASES OF.

Vesicants. — Irritants which produce blisters, *e.g.* cantharides. *See* PHARMACOLOGY; PRESCRIBING (*By the Cutaneous Surface, Irritants*).

Vesicle.—A small bladder or bladder-like structure; *e.g.* the umbilical vesicle of the embryo, the cerebral vesicles of the developing brain, the vesicles of various skin diseases, and the seminal vesicles. *See* ECZEMA (*Lesions*); EMBRYOLOGY; FOOT AND MOUTH DISEASE (*Symptoms*); HERPES (*Characters*); PEMPHIGUS (*Definition*); TYPHOID FEVER (*Complications, Cutaneous System*).

Vesico-.—In compound words *vesico-* (Lat. *vesica*, a bladder) means relating to the bladder (usually the urinary bladder); e.g. *vesico-cervical* (relating to the bladder and cervix uteri), *vesico-rectal* (relating to the bladder and rectum), and *vesico-vaginal* (relating to the bladder and vagina). *See* LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Prolonged Pressure, Vesico-Vaginal Fistula*), etc.

Vesicula. — A vesicle or bladder-like structure; *e.g.* the *vesicula fellis* (gall-bladder), *vesicula umbilicalis* or *omphalo-mesenterica* (umbilical vesicle), etc.

Vesiculæ Seminales.

ANATOMY AND PHYSIOLOGY . . . 448
DEVELOPMENT 449
ABNORMALITIES 450
INFLAMMATIONS 450
Acute 450
Subacute and Chronic . . . 451
TUBERCULOUS DISEASES 454
CYSTS, ETC., ETC. 455
OPERATIVE PROCEDURES 456

See also PROSTATE GLAND; URETHRA; etc.

ANATOMY OF THE SEMINAL VESICLES.—The seminal vesicles are two membranous receptacles, situated one on each side, beneath the base of the bladder, between it and the rectum, close to the angle formed by the bladder and the pelvic wall. When distended they form two long sacculated bodies, widened above and narrow below. Their length is usually about 50 mm., their greatest breadth about 18·5 mm., and their thickness about 10 mm., but they vary both in shape and size in different indi-

viduals on the two sides as do the testes, the right being usually the larger. Their upper obtuse extremities are separated widely from each other, but anteriorly they converge so as to approach the two vasa deferentia, which run forwards to the prostate between them. With the vasa deferentia thus interposed they occupy the two divergent sides of the triangular portion of the base of the bladder, which lies upon the rectum, and is bounded behind by the line of reflection of the peritoneum at the extremity of the recto-vesical pouch. The seminal vesicles themselves also rest upon the rectum, but are separated from it by a layer of recto-vesical fascia, which attaches them to the base of the bladder. The sacculated appearance of the seminal vesicles is owing to their peculiar formation. Each consists of a tube somewhat coiled and repeatedly doubled upon itself, and invested by dense fibrous tissue. When unrolled this tube is found to be from 4 to 6 inches long and about the width of a quill. Its upper extremity is closed, so that it forms a long *cul-de-sac*, but there are generally, if not always, several longer or shorter branches or diverticula connected with it, which also end by closed extremities. Inferiorly the seminal vesicle becomes straight and narrowed, and ends in its own duct—about half an inch in length—which unites (close to the base of the prostate) on its inner side at an acute angle with the narrow termination of the corresponding vas deferens to form a single canal, the common seminal or ejaculatory duct. The seminal vesicles receive branches from the inferior vesicle and middle hæmorrhoidal arteries and veins. The nerves belong to the sympathetic system, and come from the hypogastric plexus. In structure the seminal vesicles resemble very closely the adjoining sacculated portions of the vasa deferentia. Besides an external investment connected with the recto-vesical fascia, and containing vessels of considerable size, lymphatics, and gangliated nervous cords, they have a muscular coat and a mucous membrane. The muscular layers are thin compared with those of the vas deferens, and consist of two layers—an outer of longitudinal and an inner of circular fibres. A considerable amount of plain muscular tissue is found covering their posterior surfaces, and extending transversely so as to cover in the two vesicles. There are also longitudinal fibres traceable over the vesicle from those of the bladder.

In well-developed bodies this plain muscular tissue forms a distinct structure—ejaculator seminis — which is triangular in shape; its blunted apex being continuous with the prostate, where it is thickest. From here it passes backwards, widening and thinning as it proceeds behind the seminal vesicles and their intervening vasa deferentia, and is lost over the posterior surface of the bladder.

Fuller, of New York, in referring to this structure, says: "The muscular fibres which compose the lateral lobes of the prostate have no defined posterior border, but extend backward, and become so blended with the musculo-fibrous tissue constituting the lower portion of the seminal vesicles, that it is impossible to say just where one ends and the other begins. If these muscular bands, which extend backwards from the prostate to the walls of the vesicles, are observed carefully, it will be seen that these fibres are so arranged that, when a prostatic muscular contraction takes place, there would be exerted a strong pull in the lower half of the vesicle such as would give the whole seminal vesicle a strong squeeze."

The mucous membrane of the vesicles is traversed by very many fine rugæ, which form an alveolar structure resembling that seen in the gall-bladder, but deeper, and enclosing much finer meshes. The epithelium lining the vesicles is columnar, with a deeper layer of small polyhedral cells. It is probable that the seminal vesicles secrete a peculiar fluid, which is incorporated with the semen. The common seminal or ejaculatory ducts, two in number, are formed on each side by the junction of the narrowed ducts of the corresponding vas deferens and vesicula seminalis close to the base of the prostate gland. From this point they run forwards and downwards between the lateral prostatic lobes, and after a course of nearly an inch end in the floor of the prostatic urethra by two slit-like orifices placed in the verumontanum, one on each prominent margin of the prostatic utricle. Sometimes the ducts from either side unite together and open into the floor of the sinus pocularis. An ejaculatory duct is about three-quarters of an inch in length, and a seminal duct about half an inch, so that the urethral lining is little more than one inch distant from the lining of the vesicle, and it is not surprising, therefore, that inflammation so readily travels from one to the other.

Development.—The seminal vesicles are developed from that part of the lower portion of the Wolffian duct which goes to form the vas deferens. At the lower and outer part of this canal, towards the end of the fourth month of development, there appears a little button—a simple diverticulum of the deferent, which enlarges little by little, and is the rudiment of the seminal vesicle. At birth they form two distinct projections at the terminations of the deferent canals, more horizontal than vertical in their directions; their upper extremities not reaching the level of the ureters, and their surfaces being quite smooth. Their length is about 9 mm., and their breadth about 6 mm. Their cavity is relatively small to the thickness of the walls.

Until puberty the vesicles have only slowly and slightly enlarged. They are not more than

25 to 30 mm. long at the age of ten years, but as soon as the spermatic function is established, the activity of development which affects the testes, the penis, the larynx, and other organs affects equally the vesicles, which acquire very rapidly the volume they maintain during the whole genital life of the individual. Their infantile aspect may persist. The seminal vesicles have no analogue in the woman.

Contents and Function.—The normal vesicular fluid is white with a bluish-white tinge like skimmed milk, and becomes quite viscid and sticky when exposed to the air. It is alkaline on reaction, with a peculiar heavy odour, and coagulates on heating. It is saline, and on standing numerous groups of crystals appear in it (Böttcher's crystals), consisting chiefly of phosphates. Besides spermatozoa the fluid contains a few leucocytes, numerous epithelial cells slightly larger than leucocytes, a few round refractive cells, and considerable granular material.

The function of the vesicles is to store, temporarily, the testicular secretion, and also to provide means to preserve its vitality during that period. The semen is driven into the vesicles by a pumping action exerted upon it by the ampulla of Henle, and is expelled therefrom by a strong simultaneous contraction of the muscular fibres of the prostate and of the walls of each seminal vesicle.

The amount of testicular and vesicular fluid secreted in a given time varies greatly, and depends much on the mode of life and conditions of the individual. The amount of fluid ejaculated at a given instance varies much, depending, in a measure, on the frequency of the act. When the act is solitary and infrequent it averages from half to a drachm.

ABNORMALITIES. — No observations are yet recorded on the congenital deficiencies and irregularities of the seminal vesicles alone, but their condition in cases of retained or absent testis have been noted.

The seminal vesicles either unilaterally or bilaterally—according to whether one or both of the testes have failed to take up their positions in scrotum—show evidence of want of development, and appear as if they were almost absent in some instances. The vesicle on the side on which a testis has not descended is small, very saccular, and unfilled by semen.

It is in connection with the seminal vesicles that most of the pathological changes occur which cause disturbances in the sexual function; and the amount of disturbance in this function which these pathological processes cause seems to depend largely at least on the extent to which they interfere with the act of ejaculation. The changes due to inflammation are the chief pathological factors in their consideration.

INFLAMMATION OF THE SEMINAL VESICLE.—*Spermatocystitis. Seminal Vesiculitis or Vesiculitis.*—Vesiculitis is the term applied to inflammation of the seminal vesicle, and may be acute, subacute, or chronic. The first is usually gonorrhœal, the second frequently simple, and the third either tuberculous or gonorrhœal. Inflammation depending on syphilis, gout, influenza, etc., probably occurs also.

Acute Vesiculitis.—Acute vesiculitis is almost always excited by some affection of the membranous or prostatic urethra, usually by gonorrhœal, but occasionally by other forms of urethritis. The inflammation is the result of direct extension of the pre-existing inflammatory process, and is neither due to metastasis, sympathy, nor reflex congestion. Its analogies with acute epididymitis are striking, and are well worth attention. In the first place, both affections are rarely primary, but are almost always secondary to urethral inflammation. They occur in the third or fourth week of a gonorrhœa. The inflammatory process in both affects also the surrounding connective tissue, and is characterised by a considerable amount of swelling, giving rise to the well-known ovoidal mass filling one-half of the scrotum in the one case, and to the diffused brawny swelling felt through the anterior wall of the rectum—mistaken for the acutely inflamed prostate — in the other. In epididymitis it is well known that it is not the testicle which swells, but the epididymal connective tissue; and in seminal vesiculitis it will be found, if a careful examination is made, that it is not the prostate which is swollen, but the vesicular or perivesicular tissue beyond. Suppuration in both cases is the exception, and resolution the rule.

When acute inflammation invades the seminal vesicle that organ becomes swollen to the feel and approaches the size of a goose-egg. On touching it through the rectum, pain almost unbearable is experienced. The cavity of the sac may be distended with pus, and its walls, together with the perivesicular tissues, are swollen and œdematous. Sometimes this perivesicular inflammation is extensive enough to involve the neighbouring pelvic peritoneum, thus setting up a localised process which has been known to have eventually become general and destroy life. In a percentage of these cases the inflammation does not penetrate along the canal beyond the vesicle, but oftentimes it does extend so as to involve the epididymis, but rarely, if ever, reaches the testicle.

Vesiculitis ending in suppuration usually discharges itself by way of the ejaculatory ducts, but may form deep-seated abscesses in the pelvic tissues, which may open into the rectum, perineum, urethra, or bladder.

Symptoms.—In acute seminal vesiculitis the symptoms presented are almost wholly those due directly to the inflammatory process. There is well-marked fever, often with shiverings or distinct rigors. In the early stages of

the inflammation priapism and other evidences of excitement of the sexual centre are often present; but as the disease progresses these generally disappear. There is often a great throbbing pain in the perineum or rectum, increased if the patient sits or walks, and partially relieved if he lies down, especially on his abdomen or sides. Pain is often complained of in the sacro-iliac region. There is sometimes tenderness in the suprapubic area, corresponding to the side on which the vesicle is situated, and in very severe cases there may be a localised peritonitis at this part. The bladder functions are generally disturbed, especially during the early stages of the inflammation, the symptoms being frequent and painful micturition, together oftentimes with evidences of difficulty or retention. The painful symptoms increase as the bladder distends, and are felt most acutely at the end of the penis at the close of the act of micturition, after which a brief period of relief may follow. Defecation is frequently painful; the urethral discharge is lessened, and may disappear entirely. When the disease is on the increase, as indicated by the unabated continuance of fever, the urine voided is usually clear or nearly so. Then as the fever drops and the acute pain decreases, the urine may be found to be loaded with pus, caused by a natural discharge along the ejaculatory duct. This purulent condition of the urine continues for some time, until the sac is pretty well drained. Should the patient have in convalescence an involuntary emission, as sometimes happens, the ejaculated mass is apt to be tinged more or less with blood, and the act is accompanied with a sensation of pain.

The diagnosis of acute seminal vesiculitis is made by rectal examination alone. The normal prostate can be distinctly marked out, and its limits defined, although it is usually somewhat tender and often a little swollen; but beyond the prostate, occupying the whole bladder base, is a hard convex resistant tender area, extending upwards beyond the reach of the examining finger. In its early stages, swelling may be confined to a vesicle which can easily then be made out as a pyriform tumour with its thick end upwards; but soon this vesicular characteristic becomes lost in the larger "perivesicular" inflammation which follows.

The diseases which simulate seminal vesiculitis, and are indeed usually mistaken for it, are the acute varieties of prostatitis and inflammation of the neck of the bladder. Fuller says that stricture, peritonitis, acute appendicitis, acute epididymitis, acute pyelitis, kidney colic, and acute inflammation appertaining to the rectum may also be mistaken for it.

Treatment.—Rest in bed, the patient lying flat on his back, is a most important requisite in the treatment of this condition. The testicle should be supported with a Baer's triangle (described in section on "Scrotum and Diseases of Testicle") even although the epididymis is not tender. Large poultices or hot fomentations may be applied over the suprapubic or perineal areas, or ice may be similarly applied. Opiates should be administered to control the paroxysmal pain, and leeches to the perineum are often useful. Careful attention should be paid to the bowels, and the rectum kept empty by enemata. After the pus begins to discharge itself freely through the ejaculatory duct, poultices may be omitted. If, while the pus is discharging, urethral and vesical symptoms become annoying, balsamics may be given. Neither urethral injection nor instrumentation, nor vesicular stripping is allowable. The progress of the affection may be followed by occasional careful rectal examination. Resolution generally takes from three weeks to a month, during which time a patient should be confined to bed. In case suppuration should occur outside the vesicle, resulting in abscess formation, or an intravesicular abscess should fail to discharge itself *per vias naturales*, a perineal incision should be made after the manner described below, and drainage established.

Subacute and Chronic Vesiculitis. — These varieties of vesiculitis may be dealt with together. The former is usually of the nature of a simple inflammation, whilst the latter is most often due to gonorrhœa.

Simple inflammations such as may follow the passage of a catheter or sound along a healthy urethra, or may result from simple injuries, vesical calculus, or prostatic hypertrophy, may extend from the urethra to the seminal vesicle.

The vesicle becomes moderately swollen, and its contents semi-purulent. Its walls, however, are but slightly involved, and it is very rare that the perivesicular tissues are involved. The inflammatory process generally stops at the vesicle, but occasionally extends to the epididymis, setting up a light grade inflammation there which renders the part extremely sensitive, but which is not usually productive of much scrotal swelling.

Chronic vesiculitis is usually gonorrhœal. The common form of gonorrhœal invasion, and the one more to be dreaded owing to the lesion it leaves, is that which progresses slowly along the ejaculatory duct and into the vesicle, causing many functional disturbances, but comparatively little acute direct pain. Under the influence of this chronic gonorrhœal process the epithelium lining the part of the genital tract involved loses largely its columnar qualities, and becomes hard and pavement-like, and the subserous structures grow hard and sclerosed by interstitial proliferation. The lining membrane of the ejaculatory ducts may be altered by a chronic gonorrhœal process, but no stricture of their canals results. The interstitial changes oftentimes affect the tissue throughout the

entire thickness of the vesicle wall, and are then prone to invade also the perivesicular connective tissues. When such is the case the surrounding tissues are first invaded by effusion of plastic lymph, which in extreme instances may fill up the post-prostate rectovesical space. These tissues then show to the feel the presence of hard œdema. This is a condition which the unskilled in the rectal feel almost invariably diagnose as prostatic hypertrophy. After a time this plastic lymph becomes organised into hard sclerous tissue. This hard sclerous tissue is much less bulky than the plastic effusion. Sometimes, generally in strumous subjects, gonorrhœal perivesiculitis, be it chronic or acute, may be suppurative. This chronic gonorrhœal process generally does not extend along the genital tract beyond the seminal vesicle. It may, however, involve the cord and epididymis. In such cases the cord is not apt to appear much if at all enlarged. The inflammation of the epididymis is less than in the acute gonorrhœal condition, while at the same time it is more severe than would ordinarily be expected from a simple inflammation.

Symptoms.—In subacute and chronic seminal vesiculitis, during periods of exacerbation there may be painful sensations of a mild grade, such as have just been described as associated with the acute form of the disease. In very many of these chronic forms of inflammation, however, the associated painful sensations are slight, and in a certain percentage of them pain is absent altogether. Oftentimes symptoms of pain and discomfort caused by the disease may be so reflected as to be associated in the patient's mind with other parts. Thus, painful sensations in one or both testes, scrotum, cord, along the urethra, end of penis, bladder, and kidney may be complained of. The rectum, coccyx, and small of the back may likewise be the seats of such pain. Instances are recorded where pain due to seminal vesiculitis has so closely simulated intestinal colic and sciatica as to mislead able practitioners. Numerous and varied bladder and urethral symptoms may be hereby caused. Thus micturition may be frequent, urgent, and painful, sometimes difficult; the urine escaping drop by drop. After the urinary act has been thus inhibited for several hours the spell may all of a sudden pass off and the urine be passed easily. If these cases are carefully watched it will often be found that the vesical symptoms, such as have been described, are most intense after sexual excitement. Then again, inflammatory symptoms may exist in connection with the urethra chiefly, less often with the bladder, which are dependent entirely on an associated seminal vesiculitis. In such cases an urethral discharge of vesical pus will be found, there being no lesion in the urethra or bladder to account for the inflammatory evidences, and no amount of treatment directed toward the urethra and bladder will be found of any avail in curing the inflammatory evidences. As soon, however, as a cure of the vesiculitis may be effected, then these associated symptoms will be found to disappear permanently. Rarely the urethral discharge in such instances may be of such a severe grade as to be bloody. In some instances, through the agency of germ infection, the bladder (bacteriuria) may also become infected; when such is the case it will be found impossible to rid the bladder of the germ infection until after the focus of infection in the seminal vesicle has been eliminated. Another fairly common complaint in chronic seminal vesicular disease is that after straining at stool, specially over a hard bulky movement, there is apt to appear at the meatus a mass of glairy, sticky material.

In this class of affections, however, derangements of the sexual function are most marked, and furnish the most important group of symptoms. When the disease is not of very long standing the active evidences of irritation usually result in symptoms of sexual excitement, such as an undue amount of erections associated with an increase in sexual desire. Anything the least suggestive will be sufficient to excite the sexual centre. Involuntary emissions are in such conditions frequent, often occurring by day. These emissions are generally followed by a feeling of discomfort locally, and by depression generally. They may sometimes cause acute perineal pain. They lack force, and the amount of material ejaculated at a given instance is small. In some extreme instances there may be a feeling of ejaculation without any result, no fluid appearing at the meatus. If, in such cases, sexual intercourse be attempted, there is usually a premature ejaculation, accompanied sometimes by pain, and followed by failure of erection. In all such cases, however, whether there be premature failure or not, little satisfaction is obtainable from the act, the sexual craving still persisting undiminished by the sexual attempts. As the case becomes more chronic, feelings of sexual weakness grow more pronounced, and after a time there may be positive impotency. As the erections grow weaker and finally fail, so also do the sexual desire and craving, together with a cessation from emissions. In a small number of instances potency may persist apparently unimpaired, although sexual desire and satisfaction may be lessened. In a few cases the sexual function remains undisturbed.

The character of the seminal fluid is altered in chronic seminal vesiculitis. It is often purulent and stained with blood. In other instances, where the inflammation is very quiescent, it may be thick and jellified. In most cases the spermatozoa which it may contain are dead on ejaculation. Neurotic sensations, some of them curious and indefinite, are often caused by

seminal vesicular disease. Thus complaint is frequent that the penis is shrivelled, cold, bloodless, and numb. The testes are often retracted and supposed to be atrophying. Bad feelings in the head and sometimes sharp headaches may occur after all. Sexual excitement, mental depression, and melancholy are common in aggravated and long-continued cases of this disease. The disposition may change, a placid person becoming irritable and quarrelsome.

The study of mental conditions dependent on seminal vesiculitis is very important, and a clear distinction should be made between such cases and those in which a psychological derangement is accompanied by sexual symptoms, there being no disease with the sexual apparatus.

Diagnosis of Subacute and Chronic Vesiculitis.—The symptoms and evidences derived from the digital feel per rectum are most important, not only in determining the grade and nature of the existing seminal vesiculitis, but also in differentially diagnosticating it from other affections. To be skilled in this feel the finger of the surgeon requires much education. To practise the rectal feel the bladder must be wholly or partially distended, and the patient assume a kneeling position—best upon the front of an ordinary chair, with his chest lying upon the top of the chair back. The forefinger of the surgeon is introduced as far as possible into the rectum, considerable force being used by the remaining closed portion of the hand to overcome the resistance of the perineal muscles when these structures are rigid. During this time a counter-pressure is maintained with the free hand over the pubes, in order to bring the seminal vesicles into easier reach of the forefinger. By so doing the various inflammatory conditions of the vesicles can be mapped out and appreciated. In case the finger detects evidences of vesicular disease, then Fuller's method of "stripping" the vesicle should be carried out, in order that a specimen of the contents of the sac may be obtained (*see* p. 454). As a result of this stripping the pathological contents of the sac are forced out along the ejaculatory duct into the prostatic urethra, from which part, if the expressed fluid be abundant, it flows along the urethra, and finally drops from the meatus. After the stripping the urine should be voided into a glass, and will show evidences of the expressed seminal material. It is a good plan, before the stripping, for the patient to pass a little urine in a glass, so that thus a natural specimen can be compared with the one containing the expressed seminal fluid. When the contents sac are largely purulent, the specimen of urine passed after the stripping will appear more or less cloudy, while that passed before the manipulation may be wholly clear or much less purulent. Where the vesiculitis is quiescent or very chronic there may be no purulent elements in the contents of the sac, in which cases the expressed material will appear in the urine as a partially transparent jelly-like mass, some of which may be globular and some cylindrical. Such cylinders are moulded in the ejaculatory duct. This jelly-like condition of the expressed seminal fluid lasts, however, but a few moments in the urine, since the acidity of that liquid quickly serves to dissolve it. There are many grades in the quality of the expressed seminal fluid, between the purulent and the jellified conditions. Blood may be mixed with the vesicular material, and may be readily recognised. If the bleeding is recent the colour is red; when not recent the tint is brownish. Degenerative changes in connection with pus may give a greenish colour; oftentimes also, especially after purulent conditions, shreds made up of vesicular epithelial desquamation occur among the expressed material. A microscopical examination should be made for spermatozoa. Sometimes in light forms of simple inflammation of the ejaculatory duct there will be seen in the urine passed after stripping the vesicle a transparent flake, which floats just under the surface of the urine.

The experienced digital rectal feel, together with the process of stripping the vesicles, enables one to diagnosticate seminal vesicle diseases from those of the prostate, bladder, kidney, appendix, rectum, etc. Thus also psychological conditions—functional impotence, hysteria, and sexual weakness due to general debility—can all be differentiated from seminal vesiculitis. Persistent urethral discharges or gleets, when simply symptomatic of a vesiculitis, are frequently mistaken for localised urethral disease, stricture,—posterior urethritis, granular urethritis, and not infrequently for chronic cystitis; and the mistake of diagnosing chronic vesiculitis for chronic pyelitis is not uncommon.

TREATMENT.—*Subacute and Chronic Vesiculitis.*—These conditions should be treated, according to Fuller, by "stripping" the diseased sacs at intervals of five days or a week. It is a safe rule always to begin gently with a new case, where the parts are usually very tender, and to get the patient accustomed to the manipulation before regular and complete strippings are attempted. If the stripping treatment is accomplishing the desired results, the parts should get gradually less and less tender, besides becoming more and more normal to the feel, and at the same time the subjective symptoms should show gradual improvement. Where the treatment has been employed too vigorously or too often, then the finger will detect evidences of fresh engorgement; there will be a reappearance of tenderness together with, oftentimes, an augmentation of subjective symptoms.

The duration of the treatment may be for weeks or months to a year or over. In simple non-gonorrhœal cases in youthful subjects,

where the vesicular walls are atonic and no perivesicular inflammation exists, the cure is generally prompt, while in cases where the sac is bound down by extensive perivesicular inflammation or the individual is advanced in years, the relief from subjective symptoms may be slow, and a positive cure of the pathological process can never be said to occur. Still, even in the worst cases, time and attention will generally suffice to rid the patient of all local distress and inconvenience. During the active stage of treatment coitus should not be practised, and everything tending te produce sexual excitement should be avoided.

The stripping process effects a cure in seminal vesiculitis by aiding and strengthening the mechanism of ejaculation, and by promoting a reabsorption of inflammatory exudate. In all forms of seminal vesiculitis the mechanism of ejaculation is impaired, and, in some instances, it is rendered wholly ineffective. The stripping process, in large measure, accomplishes the results of ejaculation, while the muscles concerned in the act remain passive; and it does in many instances what the weakened mechanism has been unable to do—that is, it expels the jellified pathological fluid from the sac. The muscular apparatus by the passive exercise recovers its tone; it becomes better nourished, and the inflammatory adhesions which may have bound it down grow elastic and disappear.

During the local treatment the patient should live well, and take regular exercise—horseback riding and bicycling should be prohibited. In very chronic cases where progress towards recovery is slow, an entire rest from treatment for a month or so at a time is often of benefit.

The technique of stripping diseased vesicles is thus described by its originator (Dr. Eugene Fuller of New York):—

To accomplish the treatment, the patient presenting himself with a full bladder, should, while standing with his knees straight, bend the body forward at right angles. Then the operator should introduce the forefinger of one hand well into the rectum, the fist of the other hand exercising firm counter-pressure over the pubes. By these means the end of the forefinger will, in all ordinary cases, reach well beyond the posterior margin of the prostate. The bodies of the vesicles can thus be detected, one on each side, beyond the posterior prostatic border (only the lower half of the body of the vesicle can be felt ordinarily by the finger, the rest being beyond reach). After the forefinger has been so introduced, firm pressure should be made with its tip on the body of the vesicle to be treated as far back as it is possible to reach. Then the finger-tip, the pressure being maintained, should be slowly and firmly drawn forward along the line of the vesicle. The manœuvre is aided by the counter-pressure over the pubes with the free hand. This process may be repeated several times in connection with each vesicle. In this manner some of the vesicular contents, provided the sac be diseased and distended, can be pressed out along the ejaculatory duct, and into the prostatic sinus. After the manipulation the urine should be voided in order that the surgeon may see how much has been expressed. This treatment should be repeated not oftener than once in four days, and in most cases under active treatment as often as once a week. If it is done too frequently, or too severe pressure with the finger is employed, acute symptoms may be stirred up, which may leave the patient worse off, apparently, than before treatment was commenced, besides at times causing an acute epididymitis. The active stage of treatment lasts from four to six weeks in the most favourable cases, to eight or nine months, and possibly longer, in severe and chronic ones.

The stripping process is productive of good results not only by reason of its expressing pathological material from the cavity of the vesicle, but also by stimulating, in connection with the vesicle walls and surrounding tissues, a reabsorption of inflammatory congestions and exudations.

Tuberculous Vesiculitis.—The seminal vesicles are occasionally the seat of primary tubercle, but in the majority of cases the disease is associated with similar affections of other parts of the genital organs, most frequently the testicle and prostate. Of 36 cases of disease of the seminal vesicles collected by Dreyer, in 18 the affection was of a tuberculous nature, and of these cases one-half occurred in persons over forty years of age. It is often associated with tubercle in distant regions of the body, and is not infrequently secondary to gonorrhœal inflammations of the vesicle.

Primary Tuberculosis of the Seminal Vesicle.—In primary or acute tuberculosis of the seminal vesicle the inflammation rarely confines itself to the sac and its walls, but shows a marked tendency to involve the perivesicular tissues. Such perivesicular inflammation may contain purulent foci, and may extend backward and involve the peritoneum. The disease often spreads along the cord to the epididymis.

In a subacute form there is a milder degree of inflammation, and fewer subjective symptoms. The walls of the seminal vesicle become moderately thickened, and little but local tenderness is revealed by palpation. Such cases may end a resolution, and rarely, unless aggravated by some exciting cause, extend beyond the seminal vesicle.

Secondary Tubercular Seminal Vesiculitis.—Secondary or chronic tubercular inflammation extending from the urinary tract represents a severe pathological disturbance. The chronic form, although not very painful, except when

subjected to irritation, is always accompanied by subjective symptoms. In connection with the seminal vesicle, the inflammation sometimes shows a marked tendency to involve the perivesicular tissues, filling in the post-prostatic rectovesical space with a hard inflammatory extravasation, so that by the rectal feel it is impossible to define the posterior prostatic border. In severe cases this perivesical effusion may contain pus foci. This form of inflammation generally extends along the genital tract, beyond the vesicle to the cord and epididymis.

A certain number of cases here, as elsewhere, under favourable circumstances terminate in recovery either by resolution, by fibrosis, or by the disappearance of the tuberculous focus altogether. Sometimes the process comes to a standstill, softening areas calcify, and a calcareous nodule, which may remain quiescent, results. More often, however, the disease is continuously progressive, and abscesses form which may open into the bladder, the urethra, the peritoneum, the perineum, or the groin.

The symptoms of tuberculous vesiculitis are those of the other forms of inflammation, but the existence of hard nodules in different parts of the organ which are not very tender on pressure, is very suggestive of the tuberculous nature of the disease; and when these are associated with similar nodules in the prostate or epididymis, the diagnosis is certain. Recurrent urethral discharges are sometimes seen.

The treatment of tuberculous vesiculitis should be by general hygienic measures, but where the disease is primary, removal of the affected organ by operation is called for; and when the epididymis is also affected operative measures may still be adopted. "Stripping" should not be practised in tuberculous disease, except for purposes of diagnosis, and then only in the gentlest manner.

Little is known of the syphilitic affections of the seminal vesicles. G. Lewin once found the walls of the seminal vesicles indurated; in another case a cartilaginous induration of the walls was present to such a degree that there was hardly room for the head of a pin inside.

Dr. Fuller has seen a case of mumps where the seminal vesicle shared with the epididymis and testicle in the inflammatory process; and Comby, in a case of acute mump orchitis and epididymitis, was able by rectal examination to determine the existence of marked tenderness of the corresponding half of the prostate and of the seminal vesicle on this side.

Cysts.—Cysts are occasionally found either due to localised distensions from the vesicle itself or from obstruction of the ejaculatory duct. Their diagnosis can be made by needle puncture, and they can easily be emptied by aspiration. Should more radical measures be called for, they may be excised by the method described below for the removal of the seminal vesicles.

Injuries.—Traumatisms in connection with the seminal vesicles are rare, owing to the protected position of the parts. They may be injured by a perforating wound involving the rectum and bladder, such as occurs occasionally when one is thrown against or sits upon a sharp body. They are also occasionally involved, one or both ejaculatory ducts being cut across or lacerated in surgical procedures, as in vesicle puncture per rectum, for the relief of retention, in extensive prostatectomies, in median perineal sections, where the cut is very deep, and in attempts to draw a stone too large in size through a perineal wound. From such injuries inflammation may result.

Calculi.—Calculi of the seminal vesicles are rare. Guelliot says that these bodies are usually small, and bear a strong resemblance to prostatic calculi. They have a nucleus of spermatozoa, epithelium, and mucus, and are usually small and white. On analysis they are found to consist of seven parts of phosphate and lime and one of animal matter. Keyes removed a calculus from the ejaculatory duct by perineal section, and Fuller succeeded in discharging one the size of a grain of wheat by his own method of "stripping."

Malignant Disease of the Seminal Vesicle.—It is not uncommon for cancerous affections of the prostate, bladder, rectum, or testicle to extend to and involve the seminal vesicles. Primary cancer of the seminal vesicle, however, is apparently quite rare, although, were digital rectal exploration of these organs more commonly and systematically practised, it probably would be found to be of much more frequent occurrence than the present statistics indicate. Guelliot, who has analysed 14 cases, in which one or both seminal vesicles have been affected by cancer, finds but one in which the growth was distinctly primary in the seminal vesicle Gay, of Boston, reports a case of primary cancer of the seminal vesicle in which he made an unsuccessful attempt to eradicate the disease by extirpating the organ.

Sarcoma of the seminal vesicle, either primary or secondary, is of very rare occurrence. Zahn reports the only case of primary sarcoma of this organ.

Malignant growths of this part disturb the sexual function, and, if at all extensive, interfere with urine drainage, and to a less extent with the functions of the lower bowel and rectum. They are also accompanied by pain more or less localised. The recital of clinical symptoms has been so meagre in the few cases reported, that an attempt at detail in this particular is impossible Where disease is confined to the seminal vesicle, speedy extirpation of the organ is advisable. Rydygier's incision, which is a modification of Kraske's, is the method which

should be employed, since that cut gives the operator the space necessary to make the removal most thorough and complete.

Operative Procedures.—The operative procedures which have been performed in connection with the vesicles are (1) aspiration; (2) incision and drainage; and (3) extirpation.

Aspiration is only applicable to certain cases of non-suppurative distension, and may thus be aseptically performed. A long stout needle is entered about an inch in front of the anus, and a little to one or other side of the middle line, and is pushed on through the perineal tissues guided by a finger in the rectum until it enters the cavity of the distended sac. It has been chiefly employed as a means of diagnosis.

Incision and Drainage.—This operation is applicable to acute suppurations in and about a seminal vesicle, and is performed similarly to aspiration. A long narrow-bladed knife, with its edge directed outwards, is entered to one or other side of the middle line, an inch in front of the anus, and is guided by a finger in the rectum to the fluctuating area below. The blade of the instrument is gently rotated on its long axis to and fro, as it sinks into the tissues; when the abscess is "struck" the pus escapes along the side of the blade, and the knife may then be withdrawn, enlarging the opening in a direction obliquely downwards and outwards as it is withdrawn. A drainage tube may be inserted on a probe or with Lister's sinus forceps to the bottom of the wound.

Extirpation.—The perineum is shaved and prepared in the usual manner. A large gum elastic catheter is passed and retained in the urethra, and if the bladder is empty it should be filled with ten or twelve ounces of fluid. The patient is placed in lithotomy position, and having determined that the rectum is empty, a median incision is made from a point half an inch in front of the central tendon downwards to within half an inch of the anal margin. If the patient is stout and the perineum deep, the incision may be carried a quarter round the anus to either side, and about half an inch from it. The wound is deepened layer by layer, the rectum being pushed backwards, until the bulb, covered by its muscle, is seen at the front of the wound and the anterior fibres of the levator ani muscle looping up the prostate. These fibres must be divided mesially, and the under surface of the prostate clearly made out. The rectum must still further be repressed by means of long, deep retractors, and the special layer of fascia and muscle passing backwards from the base of the prostate over the vesicles divided laterally downwards and outwards in the direction of the vesicle. When the vesicle is clearly defined, it must be enucleated from its bed with the fingers, aided by a suitable blunt dissector if necessary. A finger introduced high into the rectum may be utilised to press the organ down into the wound, where it can be more easily grasped and drawn forward preparatory to being excised (Roux). Care must be taken not to damage the rectum in the early stages of the operation, nor the bladder or peritoneum during the enucleation of the vesicle. The cavity must be drained by iodoform gauze drainage, and the angles only of the outer wound closed by a suture. The wound gives rise to very little trouble, and heals rapidly if the whole disease has been cleanly removed. Nodules can be enucleated out of the prostate at the same time if necessary.

This is the plan I have several times followed in the removal of diseased vesicles, and have not felt the want of any other procedure; but other methods have been described and must be referred to.

Kraske's Method.—An incision commences near the posterior superior spine of the ileum on the right side, and extends along the border of the sacrum and coccyx to the middle line of the perineum below. All tissues are divided down to the bone, and the coccyx and sacrum up to the third foramen are taken away. The deeper part of the operation proceeds on the lines of the one just detailed.

Rydygier modified Kraske's operation in the following manner:—An incision commences just behind the posterior superior spine of the ileum, extends obliquely downwards, along, and half an inch from the border of the sacrum, and on to the coccyx to the middle line, along which it is continued towards the rectum as far as may be necessary. A transverse cut is then made inward across the sacrum just below the third sacral foramen, and the exposed bone divided across with chisel and mallet. The lower triangular flap is then raised without difficulty, and bent back, thus affording much exposure of the pelvic contents. After the completion of the operation the flap of bone and skin is replaced.

Von Dittel's Method.—A catheter is tied in, so that the urethra may be easily appreciated, and shielded from injury in operating. For like reasons the rectum is stuffed out with gauze, the ends of which are left hanging out so that the whole can be easily removed. The patient is placed lying on his belly, with his legs hanging down. A cut is then made extending from the tip of the coccyx down on the middle line almost to the rectum. It then deviates to the right or left as the case may be, just avoiding the rectum, making a half-moon-shaped curve around that opening, and terminating in the middle of the perineum. In completing the dissection the rectum is pushed inward and to one side. If more room is needed the coccyx can be removed, and the lower end of the incision, instead of terminating in the middle of the perineum, can be continued laterally and upward to the tuber ischii, thus

combining the Zuckerkandl incision with that of von Dittel. This combination gives very free access to the rectovesical space.

Villeneuve claims to have removed a seminal vesicle through the inguinal canal, by laying this structure freely open in its entire length, and exerting upward traction in the cord (in a manner similar to that in the round ligament in Alexander's operation) until the vesicle is brought into view and excised. This operation is inapplicable where a vesicle is bound down by adhesions, as is usually the case.

Zuckerkandl. — The patient occupies the "lithotomy" position, and an English catheter of large calibre is introduced into the urethra. The operator sits in front of the patient, and guides the knife with his right hand, while the index finger of the left hand is introduced into the rectum, in order that in the progress of the deep dissection the anterior wall of the rectum shall be avoided. A large perineal flap-incision is made, whose posterior extremity on either side extends to the tuberosities of the ischium, and whose anterior boundary lies in front of the rectum. After division of the skin and the subcutaneous connective tissue the perineal septum is divided transversely, and blunt dissection is made between the rectum and the urethra upward toward the prostate. The detachment of the rectum is continued through a considerable extent, until the seminal vesicles and the base of the bladder become visible in displacement of the rectum towards the sacrum. The altered seminal vesicles are peeled out of their surroundings, dissected from the fundus of the bladder, and removed. At the same time morbid collections in the prostate gland can be excised.

Resection and Extirpation of the Vas Deferens. —Resection of the vas deferens in its continuity has been performed in numerous cases. The vas is palpable through the skin, as a round firm strand, and it may thus be separated from the remaining structures of the spermatic cord. The cutaneous incision for the isolation of the vas deferens, 3 or 4 cm. long, may be made either in front of the external inguinal ring or at the neck of the scrotum. The structures forming the spermatic cord are forced out of the wound, and the incision is extended upwards as far as the level of the internal ring, the inguinal canal is laid open throughout, so that the vas deferens is exposed in its course through this canal, and is thus rendered accessible to surgical removal. The pelvic portion of the vas would be accessible by this means only after extended detachment of the peritoneum, entailing injury of disproportionate degree. This portion of the duct is therefore to be reached by the perineal route, as already described, for removal of the seminal vesicles. In place of extirpation, divulsion of the vas deferens is recommended. The duct is isolated and exposed throughout its extent as far as possible, and by gradually increased traction until the vas gives way, about four-fifths of the duct can be removed.

Extirpation of the seminal vesicle has been practised in a number of occasions, generally for tubercular conditions. The results from such operations have not so far been very brilliant. Occasionally, however, the operation may be required. The Zuckerkandl, the von Dittel, and the Rydygier incisions, or combinations of these, represent the surgical methods to be employed.

Vesicular.—Bladder-like or made up of vesicles, or produced by vesicles; e.g. *vesicular eczema* (eczema characterised by the formation of vesicles), *vesicular emphysema* (emphysema in which the ultimate alveoli of the lungs are distended), *vesicular murmur* (the sound heard on auscultation over normal lungs), and vesicular mole (hydatidiform degeneration of the chorion). *See* DERMATITIS HERPETIFORMIS; FOOT AND MOUTH DISEASE (*Vesicular Eruption*); MENSTRUATION AND ITS DISORDERS (*Metrorrhagia, Causes, Hydatid Mole*); MORPHINOMANIA AND ALLIED DRUG HABITS (*Antipyrin, Cutaneous Eruption*); PREGNANCY, OVUM AND DECIDUA (CHORION, AFFECTIONS OF, *Vesicular Degeneration*).

Vesiculitis.—Inflammation of the vesiculæ seminales. *See* VESICULÆ SEMINALES (*Inflammations*).

Vesiculo-.—In compound words *vesiculo-* means vesicular in its various significations; e.g. *vesiculo-bronchial breathing* (a type of respiration combining vesicular and bronchial qualities), *vesiculo-tubular*, etc.

Vessels. *See* ARTERIES, DISEASES OF; ANEURYSM; AORTA, THORACIC; APHASIA; BRAIN, PHYSIOLOGY OF; BRAIN, AFFECTIONS OF BLOOD-VESSELS; BRAIN, SURGERY OF; CAPILLIARIES, DISEASES OF; EMBOLISM; HEART, PHYSIOLOGY OF; PHYSIOLOGY, CIRCULATION; PULSE; TOXICOLOGY (*Ergotism*); VEINS, DISEASES OF; etc.

Vestibule.—In anatomy the term vestibule is applied (1) to a part of the internal ear, (2) to a triangular space in the vulva below the meatus urinarius, and (3) to part of the left ventricle of the heart at the origin of the aorta. *See* GENERATION, FEMALE ORGANS OF (*External*); HEART, EMBRYOLOGY; PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Internal Ear*).

Vestigial. — Of the nature of a relic; rudimentary; *e.g.* Meckel's diverticulum, the hyaloid artery, etc.

Vesuvin.—A stain used in histological work; Bismarck-brown or triamidobenzol.

Vetch. *See* TOXICOLOGY (*Lathyrism*).

Veterinary Medicine.—The science of medicine as applied to the domestic animals, especially to the horse and cow.

Veternositas. *See* Coma Vigil.

Via.—A way, *e.g.* primæ viæ or viæ naturales (the intestines).

Viability.—Capability of living apart from the mother; the possibility of extra-uterine as compared with intra-uterine life; an infant is said to be viable when it possesses this power; according to Scotch Law an infant may be viable at six lunar months or 168 days.

Vibices. — Cadaveric lividities, sugillations, or post-mortem stains; in the singular the word is *vibex* (Lat. *vibex*, a weal). *See* Purpura (*Character of Cutaneous Hœmorrhages*); Post-Mortem Methods (*External Examination, Discolorations*).

Vibration. *See* Constipation (*Treatment, Massage, Vibration*); Nose, Chronic Inflammation (*Atrophic Rhinitis, Treatment, Vibratory Massage of Turbinated Bones*).

Vibrio.—One of the schizomycetes among the bacteria. *See* Micro-organisms.

Vibro-massage.—Vibratory massage. *See* Massage.

Viburnum Prunifolium. — Black Haw; it is official in the U.S. Pharmacopœia and in the Indian and Colonial Addendum to the B.P. for 1898, and has been used in threatened abortion, dysmenorrhœa, and menorrhagia (dose, 1 to 2 fl. dr. of the *fluid extract*).

Vicarious Menstruation.—A sanguineous discharge from some part of the body other than the uterus, believed to represent the menstrual discharge (which fails or is diminished in quantity); xenomenia; literally, *vicarius* is that which takes another's place. *See* Menstruation and its Disorders (*Vicarious*); Mammary Gland, Diseases of (*Mastitis of Adolescence*).

Vice.

Definition and General Considerations 458
The Alimentary System 460
Childish Eating — Drinking — Drug Habits—Smoking—Constipation . 460
The Neuro-Muscular System . . 462
Neglect of Exercise—Excessive Movements 462
The Sexual System 462
Physiological Psychology of the Sexual Function — Apathy — Excess—Unnatural Vice 462

See also Alcoholism; Masturbation; Morphinomania; etc.

Vice—Some Medical Aspects of

The term under which we have decided to write this article is probably not the best that might have been chosen, for it carries with it too much ethical import, and our first step must be to repudiate the moral imputation which the word vice conveys. There is no need for us to attempt philosophical accuracy, much less to aim at a contribution to ethics. Yet there is some resemblance between the view which the wise physician must take of certain practices to which some of his patients are addicted, and that with which the pastor or the teacher regards sin. From a moralist's point of view, vice must have a negative definition as a failure to achieve virtue, and so, to the physician, certain practices must appear as a failure to achieve the normal. For our purpose it is sufficient to define vice as a voluntary neglect or abuse of normal functions. We have to consider what the habits and actions are which interfere with health; and, in every relation of the organism, these may be considered as acts of neglect or as acts of abuse. For instance, it is vicious to neglect one's teeth, and it is vicious to chew tobacco; to take no exercise, and to "scorch" on wheels; to decline to eat, to eat too much, and to eat rubbish; not to make love, to make too much, and to make it in the wrong way.

At the outset we are confronted with the difficulty of determining what is too much or too little of anything, because we cannot ever expect to agree as to what is just enough even of a good thing; and still more difficult is it to agree as to what those things are which, in the vague phrase of some, are "harmless in themselves." Every case must be a law unto itself in the simple and physiological sense that anything is good which promotes the fitness of the whole organism, and anything bad which detracts from it. That at once debars us from social considerations, and sets aside the whole problem of motive. Many things which are good for the organism must, at least at times, be denied for the sake of society; and many things which are bad for the organism men and women pursue out of a desire to benefit their fellows. It is enough to suggest these, and a host of other considerations which we propose to ignore, to amplify the confession that this is anything but a contribution to ethics. We propose to discuss baneful actions simply as such.

And when we try to discover what is good for the organism we come face to face at once with the question of what the organism is for. And we had best assume that the organism does not exist for its own pleasure or satisfaction, or for the approbation of any critic; or at least that it is not judicious for physicians to admit any such doctrine as a sober truth. The or-

ganism is for work; and it is our business to make it as efficient a mechanism as possible—fit to evolve the utmost energy it may, and for as long as possible. Vices, then, are acts and habits which depreciate the organism as a working machine.

Yet we have now no simpler a conception of the problem before us, but have only shifted the difficulty to another side of it. For instance, the duration of the organism's efficiency is a moot point; for one must choose between premature cessation of usefulness and superannuation; we must decide whether we shall encourage a short life at full pressure, or a long life of mediocrity. Here again we must have regard only to utility, and ignore the claim which even earnest workers advance to survive their usefulness, enjoy a senile leisure, and earn a respectable epitaph. Others, no wiser, would urge youth onward precipitately to a middle life of distinction, crowded and hurried with an excess of effort, and would regard with complacency an early death or even deliberate suicide. We can only repeat that every case must be a law unto itself. Each life should achieve its best possibilities, and should not be curtailed by excess of early effort; and it must be a matter of opinion how such an ideal is to be furthered. Yet it seems a wise maxim to enjoin, in conformity to our point of view, that no mode of life is justifiable, however brilliant its rewards and however full of zest, which depletes more than it replenishes the organism. For we must recognise that there is a normal limit of durability in all the organs of the body, and excessive wear and tear must be considered vicious.

As we consider this subject it becomes more and more clear that the practical problem in most cases is to ensure the proper subordination of the various systems and functions to the whole organism. It is nerve-work after all that counts; the evolution of our race has been towards a mode of activity whereby we project our personality upon the world by art and handicraft, by brain and nerves, which convey, in words and in deeds, the return which each man makes to the world in which he finds a living. The organism then is a hierarchy, of which the nervous system is the head and centre, not only for what we call brain-work, but for all effort such as constitutes skilled and unskilled labour, and also domestic activities. Each organ and system therefore must be developed and yet held in check, so that it may make its greatest possible contribution to the nervous system—to quicken the mind so as to promote its capacity for perceiving, thinking, and doing; and the difficulty must always be to determine when the indulgence of any one part of the organism, say the palate, becomes disproportionate, and takes from the whole man more than it gives. In considering that difficulty we should bear in mind two different kinds of value which attach to many activities. One of these is the nutritive value, as when one eats and feeds the blood, or walks abroad and refreshes it; the other is the dynamical value, as when a meal "gladdens the heart," or a noble prospect stirs the imagination. Although these two may be described in common terms, they have essential differences, one of which is that the blood system is the vehicle of nutritive values and the nervous system the chief vehicle of the dynamical; and there are probably characteristic differences in the chemistry and in other aspects of these two kinds of processes. It is the latter, the dynamical, utility which we are most apt to depreciate, especially if we share the view of the moralist, who regards all men and most women as of the same nature, and demands from every one the same virtues and the same degree of each virtue. The physician, however, must recognise constitutional differences in his patients—not only for the purpose of nutrition, but also as factors in determining character and conduct. Probably the only physiology of mind which will enlighten us will, if it ever comes to be written, treat of the subject from this point of view; will describe the brain as a congregate of all the bodily functions as well as of the mental; and will show how it develops in relation to visceral and systemic and special sense stimulation, and how, utilising the forces of nutrition, it translates these energies into projective or practical activities. And in such a description there must be constant reference to the personal equation, to the fact that every organism is peculiar, inasmuch as the proportions of its several activities vary from those of all its fellows.

This much it has been necessary to say in a general and preliminary manner, for it is clear that an article such as this can only suggest a few aspects of so vast a subject, and leave the reader to work them out in individual cases. And though convention demands that, when we descend to the particular, we must discuss only a few flagrant kinds of misconduct, it is clear that mankind suffers far more from errors of omission which most people never dream of regarding as vice. That is the reason why we should focus our attention upon neglect of every function as a vice and not confine ourselves to the idea of the abuse of function. No greater service could be rendered than to suggest to the practitioner how much the ordinary man loses by simple neglect of the possibilities of his nature. If a man has an eye for colour, he robs himself of an important source of energy if he does not satisfy it; a refined palate, an ear for music, a delicate touch, an aptitude for construction, a capacity for sports, and every other gift of the body that we can think of, all have a contribution to make to the organism which should be duly collected. And the same

is true of our mental constitution. It is pitiful to think what scant consideration is given to the development of the endowments of the intelligence, with what complacence men and women who are most censorious concerning sins of the flesh pass over their own and their neighbour's mental indolence, and how carefully our schools cram information into young brains that are not taught even the elements of mental skill. And the whole organism suffers for lack of the appropriate zest. A man who neglects his gift for logic, for mathematics, for imagination and invention, or any other, not only loses the kindred result, the idea or project, which he might have reaped, but misses also the general inspiration derived from such exercises; for a prudent use of the mind's parts has the greatest of all values of that kind which we have called dynamical.

In pursuit of this physiological view of our subject it would be right that we should consider every function of the body and observe how its neglect or abuse reacted upon the whole organism and especially upon the nervous system, both as regards nutritive and dynamical values; then a second and larger volume should be devoted to a similar treatment of vices of the mind. But it is enough to suggest that course of study, and we must content ourselves with a few illustrative items. Other aspects we must also omit—etiology, for instance, which in every case is a relation between constitutional endowment and experience; pathology, which must refer to the secondary effects of vice, and becomes the pathology of the diseases which it has occasioned; and therapeutics, which can only be an elaboration of very elementary and often impossible advice. Nor shall we append a bibliography, which must needs include more that is misleading and uninformed and prurient than what is wise and appropriate.

The Alimentary System

Vices of the palate alone would suffice for a topic which, if exhaustively discussed, would occupy many pages, and we must restrict our attention to a few points. One of these, and an important one, is the mistake of *childish eating*, a phrase which may serve to point the moral for the elderly, while it directs attention specially to the errors of youth. For it is beyond doubt that the great majority of children of the middle and upper classes are overfed. Indeed, it is ludicrous to remark how complacently parents look on while their children obviously dilate their stomachs. The habit is one to which young people are prone because they can only conceive of the immediate value of food and drink; that is, they fill themselves for the sake of the immediate satisfaction which follows. It is in part an over-indulgence of the palate, but the more important factor is probably a mechanical one. The child is happy when he swells with food, unless a wise nature sends griping to spoil the pleasure of it; and he becomes so used to a degree of abdominal distension that he is not happy without it. This question of quantity is as important as that of quality in diet. The chocolate and candy vice is of course a stupid error; but, on the whole, the food of the better classes is not bad if only they would take a good deal less of it. For, if you let a careless boy of nine or ten take his will of a tempting meal, he will put as much into his stomach as would fill a football. Parents are therefore well advised who appreciate the capacity of a normal stomach; and when we are consulted about children who are not gaining enough weight, or whose complexion is unsatisfactory, so that, as the old saying goes, they "put their food into a bad skin," improvement will almost certainly follow if we prescribe only four meals daily, each of which can be contained in a breakfast cup. That lays upon the patient, and suggests to the child, the duty of having regard to the nutritive value of each meal; and small boys, who dislike the cure, will find some relief in a tightening of the waistband until the involution of their stomachs and abdominal walls is complete.

That suggests the interesting reflection that perhaps the crusade against tight-lacing may explain why we now see fewer "starving girls" than formerly. Only rarely, in these days when athletics are in vogue, do we meet with the misguided young woman who is too tired of life, or too pious, or too genteel to enjoy her lunch. There is a type of woman, however, often spirited and surprisingly energetic, gay and charming in society, but in her private moments pale and tired, nervous, peevish, and depressed, who cannot and will not eat a wholesome meal. Good food and healthy exercise are her aversions; but she counts upon her tea in bed each morning, and when she reclines upon the lawn, or in her boudoir of an afternoon; savouries have a great attraction for her, liqueurs, dainty confections, and cigarettes; and her unfortunate physician is periodically called upon to sanction phenacetin or antipyrin for a complication of nerves, dyspepsia, and menstrual headaches. That kind of vice must be put down with a strong hand in girlhood: and when it has become established, washing out of the stomach and the enforcement of the elementary rules of hygiene will either effect a remedy or rid the physician of an unsatisfactory patient.

This brings us to the very difficult subject, the vice of *drinking*, concerning which it is almost impossible to speak wisely. One aspect of it, not generally understood and appreciated, we may first consider, because it is important in all serious vices. It may be described as the reaction of the conscience, an interesting physiological conception. Very often, perhaps usually, drinking dulls the conscience early in a career

of drunkenness; but a great many men and women who drink too much, and who know they drink too much, yet who are not to be called drunkards, illustrate this reaction. For convenience we may use the term conscience in its loose and popular sense, and take it to embrace not only a sense of right and wrong, but also the sense of what is proper and expedient. Now even an elementary conception of the physiology of mind enables us to understand that the mechanisms of this great group of functions must be widespread and strong, that is, capable of intense and far-reaching activity. The group is, of course, one which subserves emotion and sentiment; and in the evolution of the human mind, from the beginning of social life onwards, the instinct for approbation has been about the most important of the springs of conduct. In the life of the individual the same importance manifestly attaches to these functions, as when the child refers every question of conduct to the criterion of a parent's praise or blame, and as when the man develops a sense of the approval or disapproval of God. What the nature of this fabric may be we can only conjecture; it is enough for us to recognise that there is such a mode of activity which obtains as an accessory to nearly every self-conscious act. And the important fact is that, when the reaction of this group of functions is opposed to, instead of being in harmony with, conduct a great disability is imposed upon the whole mind. As yet, we are not in a position to explain how one set of cerebral mechanisms inhibits or impedes another, and there may be no direct opposition of forces, but this much is clear, that when a certain act or habit fails to achieve the concurrent activity of so important a function, there must be a great loss of force, a much weaker production than would otherwise have resulted. Psychology, however, suggests something like actual conflict and corresponding waste of power; no mind can be quite efficient which is dogged by the consciousness that its course of action is unworthy. And in these days, when the fashion is to interpret all pathology in the crude terms of nutritive values always conceived of as pertaining to the blood-stream, it is needful for us to emphasise this other aspect of the problem. Worry certainly is as potent in the production of cerebral exhaustion as any other single factor that we can name, and it is our business to find some physiological conception for it. For this reaction of the conscience is often excessive and becomes in itself a vice; in women especially self-consciousness is often exaggerated, and the mind suffers from a too frequent and undue criticism of its performances. Many people suffer in this respect in regard to drinking.

It is useful to discern this type, which we may call the timid drinker, for it is necessary to deal with him in a manner quite different from our management of the obdurate drunkard. And, to begin with, honesty and a frank recognition of the truth about drinking is urgently called for. It is time, for instance, that the untruthfulness of the teetotal fanatic should be exposed, and the fact brought home to him that he has been a prime influence in retarding both private morality and legislative progress. The total abstinence injunction is obeyed, for the most part, by persons who have the least need of it; it has admittedly saved many reckless and confirmed drunkards; but it has also hastened the ruin of many young people who, tempted to depart from the custom of their parents, have been impelled to do their drinking alone or in undesirable company, and have added shame and fear to their weakness. Another result of the disrepute in which all drinking is regarded in certain communities is a failure to appreciate good and bad stuff. Taken all over, the drinking practices of the people are very gross and crude; a wise parent will be at some pains to educate both his own palate and his sons', and will encourage a taste for the lighter and purer liquors; and even some who habitually exceed are improved by more cultured selection of their drink. And the time at which people drink is of importance in this same connection, for the habit of drinking largely at meals is a pernicious tradition; much of the ill-effect of alcohol is due to incomplete digestion; and it is therefore probably wise to have a large and dilute drink not less than half an hour before dinner, and let that meal be as dry as possible.

The suggestion that people should be taught how to get the utmost value out of both food and drink is part of a general increase of attention to physical culture, which is having a marked influence upon the practices of the rising generation. For the rest, there is not much to be said about excessive drinking that has not been too often said already. We should be as careful, however, not to err on the side of leniency in our dealings with the obdurate drunkard, as to be not too severe on the timid patient. The latter will respond to peaceable and enlightened measures, and especially to the frank confidence of a friend. The thick-skinned sot requires coercion and intimidation. And that brings us to the everlasting demand that the law should deal boldly with these cases. Even short of compulsory detention in a Retreat, great good would be accomplished if, as has been suggested, powers were granted for proper authorities to farm the wages or salary or other income of the drunkard.

Drug Habits and Smoking.—Indulgence in narcotics is dealt with elsewhere, and upon all such practices there is nothing new to say. The whole problem simply demands courage and frankness, common sense and firmness. If, for example, it is true that cigarette-smoking

among boys is greatly on the increase, and that it is very harmful, it should be easy to rapidly diminish it. Any practitioner of parts could easily stop such a habit in a boy who was not an imbecile by some plain speaking, and by putting him under the informal control of a strong companion. If, as is suggested, physical training is to become an important feature in our educational system, one of the effects will likely be to diminish smoking and other vices. This is a kind of question, however, in which exaggerated opinions are apt to come to the surface.

For drinking, for drug habits, and for other vices, it is surprising that so little good use has been made of the therapeutic voyage. Sea voyages, as a rule, are of a minus value for the vicious; but that is probably because the régime is not explicitly arranged to suit such cases. It would be an interesting and probably a valuable experiment if some one would organise a series of therapeutic voyages during which every passenger would undergo a course of systematic physical training, with an experienced physician to prescribe and a hard-hearted captain to enforce the discipline necessary in every case.

Constipation.—Neglect to evacuate the bowels is a very prevalent vice, especially in women. It is indeed surprising that there is not more widespread knowledge of the importance of the excretory functions; perhaps the majority of women in the poorer ranks, and a considerable number of the well-informed, believe that two or even less evacuations in the week are sufficient; and it would seem that many physicians are not much more enlightened. The reason is, that, in some cases, the system becomes habituated to the intestinal poisoning, and suffers only a slow and perhaps postponed injury. In such cases, however, improvement in general health is obvious if a daily and complete evacuation is achieved, though it is common for the patient to feel worse for a short period if the habit of constipation is suddenly broken and intestinal activity too suddenly induced.

The Neuro-Muscular System

It is quite impossible to even suggest the various vices of nerve and muscle which the physician may have to deal with. It may be useful, however, if we would induce a patient in ill-health to make the most of himself, to repeat that abuses and neglects of nervous activities are both prevalent and important. Restlessness and excitability in all its forms—from the habit spasms of the choreiform to the garrulity of the illiterate and the anxious worrying of the too self-conscious—are amenable to common-sense treatment, and will repay it. But the neglect of the muscular system is a more frequent and an important fact in present-day life. Our men are perhaps busy enough in working hours, but, in hours of leisure, indolence is the rule. It is astonishing how seldom one meets a patient who can do anything at all except the business by which he earns a living, or who has any interests and tastes which stir his intelligence. And young women suffer still more in this respect. One of the most striking characteristics of their class is an over self-consciousness consequent upon a sense of uselessness. The amount of time and energy devoted to the learning of certain accomplishments which rarely have any tangible result is simple waste. The mind requires an outward effort, and something to show for it. There are few activities which yield so much zest in life as those in which there is something to be seen actually growing before one's eyes. The fashion which at present encourages young people to develop their muscles severally and assiduously, depends to some extent upon this pleasure which we normally have in a palpable demonstration that virtue is rewarded. But it is difficult to understand why the same energy should not be devoted to more useful pursuits. Gardening is perhaps the ideal industry, in that it gives exercise to great masses of muscle, encourages a love of open air, promotes hardihood, and forms a contact for the mind with primitive and natural forces. But there are endless forms of handicraft which are easy to learn yet persistently neglected, and which would repay much culture. For one of the most useful and fertile elements in a human mind is the consciousness of skill and the stimulus which comes from the mere success of making something. Our race has developed along lines in which the formative or constructive activities were of the utmost importance; the exigencies of city life have led us away from them; and they are wise who, even under great difficulty, reach back to them. This is a therapeutic which physicians, as a rule, are much too slow to utilise. For patients who are subject to nervous ailments are invariably too self-conscious, at least in the early stages of their illness, and in many cases that stage would not be reached if men and women were seriously interested in projects other than personal.

The Sexual System

The physiological excuse for the tendency of civilised nations, and especially, perhaps, of the British, to make a fetish of chastity is to be found in the monopoly of the attention which sexual indulgence is apt to establish. The same is true of gambling and of gluttony, though not to so great an extent. For the sexual function is woven intimately into the whole nature, and its activities swamp and inhibit, or at least suborn, all else. Only a few men, and still fewer women, can make an art of loving and yet retain their interest in affairs. Love makes

a fool of a man; to be enamoured is to neglect all but one aim; and to make a study of sexual practices of necessity debauches the mind. And this, be it observed, is especially true of illicit and perverted sexual vice, for then the mind is weakened and predisposed to calamity by the reaction of the conscience, focussed in a fear of discovery and disgrace.

In this connection it is well to dissociate ourselves from the purely spiritual aspect of the subject, and to have special regard to the physiology of emotion; for we have, in the psychology of sexual indulgence, a vivid example of the importance of cortical arrangements and their relations with visceral ganglia in determining diseased processes. The sexual mechanism, as has been observed, must be very widespread in its connections; from the organs of generation tracts, obviously capable of intense activity, reach inwards to the sensorium, and are presumably adapted both for the reflex activities which occasion trophic changes in various organs and vague systemic effects and also, probably by special fibres, for the direct stimulation of the cortical mechanisms, whose activities coincide with appropriate emotion. For this sexual function we must remember the unlimited avenues of stimulation from without as well as from within, from vision, hearing, smell, and touch; and, on the other side, the expressive or outward relations which are the characteristic effects and acts of this function, such as those which permit of interference with respiration and circulation, with vaso-motor functions, and even with gastric and renal activities, and the other more voluntary relations which become translated into ideas and purposes and acts. We are justified in assuming that, at least in some people, this mechanism is the most powerful—fit for the most intense activity and having the most widespread relations; while in others it would appear to be insignificant. And we cannot ignore the corresponding differences in the dynamic value of two such natures. For it is absurd to deny the importance of such a function in stimulating the whole organism, or its importance at other times in depleting it; and again, if it is at variance with what we call the conscience, the disturbance and instability which such a conflict occasions.

Apathy.—The first vice which we have to consider in relation to this group of functions is the feeling and practice, often due to constitutional defect, which finds expression in the view that virginity is of itself a good thing. It is almost unnecessary to say that women are more commonly defaulters in this respect than men. It may be hazarded that in every such case, when the apathy is genuine, there is a structural reason for it, most commonly perhaps in the cortex—a kind of sexual imbecility. In men it is commonly associated with cowardice and selfishness—a foolish bashfulness or an undue subservience to a false idea of modesty, and, very often, an exacting fear lest to share another's emotions should curtail the opportunities of other forms of self-indulgence.

But this vice of sexual neglect is commoner in women—so common indeed as to be alarming, for its prevalence bodes ill for the race, and it is moreover an important factor in many cases of conjugal infelicity. One even hears of women long married who do not seem to have ever experienced any great sexual awakening, though perhaps from a sense of duty they permit intercourse with the husband. In some instances it would appear that this function has never been reached, because it has been fenced off by constitutional barriers which prohibit any state of mind approaching to abandon, and which predispose to a constant watchfulness or determine an exaggerated idea of dignity. Such cases are probably hopeless unless the hidden potencies can be exploited by some ingenious and subtle suggestion which associates love-making with mystic ideas under circumstances likely to induce ecstatic feeling. Perhaps the majority of cases of feminine apathy, however, are occasioned by the aggressiveness of the husband, which creates in the wife a revulsion against a practice which would make her merely the instrument of an appetite which seems both senseless and beastly. In many instances, unless this adverse relationship is of long standing, a felicitous result follows when the husband learns to regard his wife's feelings before his own. In any case, apathy is not a state which the physician should ignore; for we must recognise how profound and beneficial an alterative effect sexual awakening induces, how married life promotes healthy metabolism if pregnancies are not too frequent, and how it enlarges and revives the mind. That effect, it is interesting to surmise, may depend in women partly upon the dynamic value of opening up the sexual tracts in the nervous system, and partly upon the quickening effect of germinal infusion upon the general metabolism.

Sexual Excess.—Sexual excess is probably not commoner than sexual neglect, though a perverted public opinion metes out to it a more ready condemnation. Its evil effects upon the organism are both obvious and manifold. We must specially bear in mind the claims which an excessive sexual indulgence makes upon the attention, how hard it is for the sexualist to do good work, and how likely he is to miss many of the most desirable intellectual and emotional interests; and also the probability that his health will be impaired and his disposition demoralised. Here again we have to note two types: one of them unashamed, and even careless of detection, and who consequently suffers less; the other, under the reaction of fear, apt to become furtive and morose.

Perhaps no form of vice illustrates better the

maxim that every man must be a law unto himself. For it is clear that what is excess for one person may be of no bad effect upon another. Each case must determine to what extent the indulgence can be permitted, and at what point the whole constitution loses more than it gains by it. The gross evidences of such an injury would be sought for in the skin and in muscle and nerve—pallor, flushings and pimples, tremor and restlessness going on to loss of sleep, with perhaps the addition ef perverted appetite for food and drink.

Nothing can be expected from a restatement of the appropriate treatment in such cases. Even if the physician detects signs of this vice in any of his patients, he will probably not feel justified in interference. When the patient comes with a frank confession and a sincere desire for a reform of his habits, the battle is half won, and nearly every such case should have a successful termination. The management of such a patient is a mere matter of common sense. Above all, the patient's attention should be directed away from his habit, and not focussed upon it; that is, he must be encouraged to develop his spiritual and his physical nature to the highest pitch of fitness of which they are capable; and all the effort and enthusiasm that can be evoked should be explicitly directed to that end, with the least possible reference to the vice that makes the effort necessary. That is true of most habits. We must remember that vice is supremely interesting, and especially to the vicious; that every victim of a depraved habit has an "inverted gusto" in discussing it, even though it be in tears and with every token of repentance; that the best chance for every one is to keep his mind fixed upon, and order his life so as to achieve, the other activities which he has been neglecting; and if, after all, the vice persists, it is a great gain if the patient, notwithstanding his lapses, occupies his mind in his "lucid intervals" with things that are worthy and above reproach. To such an end wise companionship is almost indispensable. And for this and other vices we should remember the usefulness of the idea of postponement; that, while it is often impossible to persuade a patient to total abstinence, and very hard to get him to stick to a prescribed discipline, it is always useful, and appeals to his most manly instincts, to induce him to practice daily the trick of putting off and off the hour or the day of his indulgence. Many men are interested and even amused at testing their endurance, and have an added zest in expectation of the added pleasure which their postponement of it ensures, who could not be brought to even attempt total abstinence.

Sexual Perversions.—We may affirm, probably without fear of refutation, that the great majority of the boarding-schools in this country are infested with sexual vice. The evidence of masters, and especially head-masters, is of the least possible value in this connection. We must go for evidence to "old boys" who have reached years of discretion, and who have learned to regard their sexual organs as wholesomely as they regard their other members, yet who have no interest in boasting or other exaggeration. Any one who is in a position to acquire the facts, and who takes pains to probe the subject, can convince himself that there is hardly a boarding-school in the country which is not, at least at times, a hotbed of sexual vice. For obvious reasons, it occurs in epidemics, for it is of necessity contagious. This applies even to masturbation; the self-taught masturbator is about as rare as the self-taught drinker, for it is characteristic of the vice that its victim has a strong desire to communicate it to his fellows. And so at times in large boarding-schools the state of affairs is not only very bad, but, at least among the boys, quite notorious, and a few ringleaders, even openly jealous of each other's conquests, renew their barely-concealed efforts to add to their train of followers.

In such epidemics there comes to be something like a cult of sexualism, and, if it were profitable to pursue the subject minutely, we should find many resemblances to the mystic rites of savage tribes. Not only do the boys vie with each other to discover new and attractive methods of sexual stimulation, but we hear also of the outcropping of the curious perverted instinct to inflict wounds upon themselves and upon each other; of the bestowal of, for them, princely gifts upon the objects of their affection, as upon a mistress; and even of the placing on a pedestal of some chosen child, perhaps more comely in appearance and in spirit than the rest, whom one of these leaders in vice will champion, and whose innocence he will protect with the most jealous care against both his own and his rivals' blandishments.

On the other hand, it is only fair to say that at times the spirit of those same schools may turn from such vices, and show a corresponding zest in observing a clean and wholesome life. It is also most necessary to point out that what has been said refers only to boarding-schools, and that apparently the day-schools of the country are very rarely infested.

The most difficult and perhaps the most important part of this problem is to determine to what extent such vices prevail among girls and women—difficult, because its investigation is one of insuperable delicacy in cases which it is most worth one's while to try to save; and important because it is next to impossible for a woman to persist in such practices with anything like an even mind. Many vicious men are comparatively happy, and live fairly cheerful and successful lives. Women cannot be so,

partly because their sexual nerve-tracts are less detached than in men, more intimately and widely related to other functions, and partly because the conscience-reaction is almost invariably greater and more disastrous in women. It is enough to say that masturbation by women is much commoner than is generally suspected by physicians, and that it is probably our duty, in neurasthenic and allied cases which baffle us, to overcome the difficulty of determining whether this is or is not a factor in the case. The treatment is not more difficult than in men, but we must bear in mind this important difference, that, while many men may underestimate the evil effect of bad habits, a woman almost invariably exaggerates it, and one's attention in her case should be explicitly directed towards a more confident and unself-conscious state of mind.

Nothing is to be gained by a more elaborate study of perverted sexual practices. It may be admitted that the subject is an interesting one; but the interest of it does not seem to be wholly scientific. The most that can be done is to suggest that these evils are much commoner than is supposed, and that physicians should be alive to them, and talk the subject over not only with boys but with parents, many of whom are in a state of crass ignorance on the subject. And a word of warning is necessary in dispute with certain writers who attribute most cases of bad habit to perverted instinct, and who assume a congenital constitution whereby the mind of the patient—both in idea and in feeling—is naturally attracted by the person of some one of the same sex. Such cases are of extreme rarity. Nearly every case is one of perverse habit, not one of perverse instinct. To put the matter plainly and by the method of bald illustration, when a case comes before us of a man who shuns women and makes love to men, it is safe to assume that his sexual mechanisms were fairly normal in childhood, but that long association in immorality has given him an irresistible taste for the wrong thing. There are, however, genuine cases of congenital perversion, and it is hard to conceive any appropriate treatment of them short of segregation or castration.

Vichy. *See* Balneology (*France, Alkaline*); Mineral Waters (*Alkaline*).

Vicq d'Azyr's Bundle.—A band of nerve-fibres passing from the median nucleus of the corpus mammillare of the brain to the optic thalamus (anterior tubercle).

Vicq d'Azyr's Operation.—Incision of the crico-thyroid membrane of the larynx.

Vidago. *See* Balneology (*Portugal*).

Vidian Canal.—A canal in the sphenoid bone passing from the foramen lacerum to the spheno-maxillary fossa, and named after Guidi (Lat. *Vidius*), an Italian anatomist; the *Vidian artery* passes through the canal.

Vienna Paste or Caustic.—A mixture of slaked lime and caustic potash, used to destroy small growths, such as warts and caruncles.

Vierordt's Method. *See* Blood (*Methods of Clinical Examination, Estimation of Total Quantity*).

Vieussens, Annulus of. *See* Physiology, Circulation (*Intra Cardiac Nervous Mechanism, Sympathetic Fibres*).

Vieussens, Valve of.—The anterior part of the roof of the fourth ventricle of the brain.

Vigilambulism. Sleep-walking or somnambulism. *See* Hysteria (*Vigilambulism*).

Vigilium or Vigil.—Absence of sleep. *See* Coma Vigil.

Villefranche. *See* Therapeutics, Health Resorts (*Riviera*).

Villi.—Hair-like projections growing from a mucous surface (*e.g.* the intestinal villi), or from the outer aspect of the chorion (chorionic villi), or on synovial membranes; the singular form is villus (Lat. *villus*, a shaggy hair). *See* Fœtus and Ovum, Development of (*Chorion and Placenta*).

Vina.—Wines; pharmaceutical preparations in which sherry is used to extract the drug; examples are found in Vinum Ipecacuanhæ, Vinum Ferri Citratis, and Vinum Quininæ (in the two last named orange wine is used instead of sherry). *See* Prescribing; Antimonium; Colchicum; Ferrum; etc.

Vinadio. *See* Balneology (*Italy, Sulphur Waters*).

Vincent's Angina.—A pharyngitis resembling diphtheria, characterised by the presence of a fusiform bacillus; there is a pseudo-membranous inflammation; the symptoms are like those of diphtheria. *See* Diphtheria.

Vincula Accessoria Tendinum.—Additional insertions of the flexor sublimis and of the flexor profundus digitorum (Lat. *vinculum*, a band).

Vinegar.—Dilute and impure acetic acid ($C_2H_4O_2$); in Pharmacy a solution of a drug in vinegar or in acetic acid; there are two official preparations of Acidum Aceticum, viz. *Acidum Aceticum Dilutum* (dose, ½ to 2 fl. dr.), and *Oxymel*, containing 1 part of acetic acid and 1

part of water to 8 parts of clarified honey (dose, 1 to 2 fl. dr.). *See* ACETA.

Vinum. *See* VINA.

Violation. *See* MEDICINE, FORENSIC (*Rape*).

Violet.—One of the colours of the spectrum; also the name given to several staining agents (methyl-violet, gentian-violet, etc.) used in histological investigations. *See* COLOUR VISION; SPECTROSCOPE IN MEDICINE; POST-MORTEM METHODS (*Examination of Tissues, Staining*).

Violin-Player's Cramp. *See* NEUROSES, OCCUPATION (*Etiology*).

Viperidæ. *See* SNAKE-BITES AND POISONOUS FISHES; SNAKE-BITE IN THE BRITISH ISLES.

Virchow's Disease.—Leontiasis ossea or megalocephaly. *See* LEONTIASIS OSSEA.

Virginian Prune Bark. *See* PRUNES.

Virginity. *See* MEDICINE, FORENSIC (*Rape, Evidence*).

Virility.—The power of procreation as an attribute of the male after the age of puberty; the penis is sometimes called the virile member.

Virus.—A poison (microbic or chemical) which causes a disease or pathological change; it can be deprived of some of its virulence by culture and inoculation, and is then called *attenuated virus*; the virus of hydrophobia when propagated through a series of rabbits increases in virulence till it reaches a stage when its incubation period is seven days—it is then called *virus fixe*; the lymph taken from the vaccine pustule of a human being is called *humanised virus*.

Vis.—Power of force; e.g. *vis formativa* (formative or reparative energy, as shown in the tissues), *vis medicatrix naturæ* (the natural tendency to recovery or healing in contrast to that due to medicines or to operations), *vis a tergo* (force acting from behind, *e.g.* the uterine contractions in labour in contrast to the *vis a fronte* as exhibited by the forceps).

Viscera.—The plural of *viscus*, any one of the organs of the four body-cavities (head, thorax, abdomen, or pelvis); *e.g.* the abdominal viscera, such as the intestines, stomach, liver, spleen, etc.

Visceral Pain.

I. DEFINITION 466
II. NERVE-SUPPLY OF THE VISCERA . 467
III. EVIDENCE OF THE INSENSITIVENESS OF THE VISCERA—
(*a*) *Experimental* . . . 467
(*b*) *Clinical* 467
IV. NATURE OF THE PAIN EVOKED BY STIMULATION OF THE VISCERA . 468
V. THE SENSORY SUPPLY OF THE TESTICLE 468
VI. THE SENSORY SUPPLY OF THE ORIFICES OF THE BODY . . . 469
VII. CLINICAL EVIDENCE OF THE REFERRED NATURE OF THE PAIN . 469
(*a*) *The Radiation of the Pain* 469
(*b*) *The Distribution of the Hyperæsthesia* . . 469
VIII. THE CHARACTER OF CUTANEOUS HYPERÆSTHESIA 470
IX. MUSCULAR HYPERÆSTHESIA AND MUSCULAR CONTRACTION . . 471
X. THE SPREADING OF SYMPTOMS . 472
XI. TENDER NERVES AND TENDER VERTEBRAL SPINES 472
XII. VISCERAL AND CUTANEOUS MUSCULAR REFLEXES 472
XIII. SENSORY SYMPTOMS OF INDIVIDUAL ORGANS AND STRUCTURES . . 473
(*a*) *The Hollow Muscular Organs — Digestive Tube, Gall-Ducts, Ureter, Bladder, Uterus, Heart* . 473
(*b*) *Serous Membranes* . . 477
(*c*) *Parenchymatous Tissues* . 478
XIV. OUTLINE OF THEORY CONCERNING THE RELATIONSHIP OF THE SYMPATHETIC SYSTEM TO THE CEREBRO-SPINAL 479

See also under the various organs, BLADDER, BRAIN, LIVER, LUNGS, etc.; POST-MORTEM METHODS (*Examination of Body Cavities*); TERATOLOGY (*Heterotaxy*); etc.

I. DEFINITION.—*Pain is a disagreeable sensation due to stimulation of some part of a cerebro-spinal sensory nerve or its centre, and referred to its peripheral distribution in the external body wall.*

The term cerebro-spinal used here has reference solely to the nerves arising from the brain and spinal cord, and distributed to the external body wall. Although the tendency is at present to regard the division of the nervous system into cerebro-spinal and sympathetic as obsolete, yet there are good grounds, to be shown in the following argument, for maintaining the distinction.

In the above definition it is contended that wherever the stimulation reaches the cerebro-spinal sensory nerve, whether in the brain, in the spinal cord, in the trunk of the nerve, or in its peripheral endings, the sensation of pain is referred to the peripheral distribution of the nerve. Thus in certain diseases of the brain, and in rare cases of epileptic aura, where the stimulation affects a part of the cortex of the brain, which is in connection with the sensory nerve, the sensation is referred to the peripheral distribution of that nerve. In diseases of the

spinal cord, as in locomotor ataxia, the characteristic lancinating pains are referred to the peripheral distribution of the nerve. So also are the pains of herpes zoster, a disease of the ganglia of the posterior spinal roots. In this article the subject is limited to the study of the pain and certain other associated symptoms arising from disease of the thoracic and abdominal organs. From the study of the facts that will be detailed later the following conclusions have been arrived at: that pain is only felt in the peripheral distribution of a sensory cerebro-spinal nerve; that there is no evidence of sensory cerebro-spinal nerves being distributed to abdominal or thoracic organs; that the stimulation proceeding from those organs, which gives rise to pain, is conveyed to some part of the brain or spinal cord, there the stimulation affects some portion of a cerebro-spinal sensory nerve, and the pain thus arising is referred to the peripheral distribution of the sensory nerve so affected.

The view expressed in the above statement implies that the organs themselves are incapable of evincing pain or other sensation, as touch, sensibility to cold and heat, but that there is originated in them an impulse which passes by their nerves to the centre of one or more sensory nerves in the brain or spinal cord. The stimulation of these nerve centres evokes pain which is referred to the peripheral distribution of these nerves in the body wall.

II. Nerve-Supply of the Viscera.—The chief nerve-supply of the thoracic and abdominal organs comes from the sympathetic system. Nerve fibres pass out from the sympathetic ganglia to the various organs, and from the organs to the sympathetic ganglia. Nerve fibres pass from the spinal cord to the sympathetic ganglia, and from the sympathetic ganglia to the cord.

The functions of these nerves passing to and from the sympathetic ganglia are various, but are mainly concerned in regulating the functions of the various organs to which they are distributed. It is frequently assumed by experimental physiologists and others that certain afferent nerves in the viscera are "sensory" in function, and that certain ganglia are also "sensory." Except that these nerves run from the viscera to the cord, practically no proof has been adduced to support this assertion. Observations and experiments directed to ascertain the degree of sensibility of the viscera have demonstrated their insensitiveness. Pain, when evoked, has invariably been a referred pain.

The other nerves distributed to the viscera are the vagus and phrenic nerves. Branches of the vagus pass to most of the thoracic and abdominal organs. The functions of the vagus are numerous, but there is no evidence that it is a sensory nerve, yet there are grounds for assuming that it contains afferent fibres, which convey impressions that stimulate the centres of cerebro-spinal nerves. The phrenic, though mainly motor in its functions, has been demonstrated by Sherrington to contain afferent fibres. These convey impressions which stimulate the roots of sensory nerves in the spinal cord. Evidence of these statements will be given later.

If there be no sensory nerves distributed to the viscera, the question arises, by what mechanism is the symptom of pain brought about? That pain is evoked from affections of the viscera is a matter of which there can be no doubt. It is scarcely conceivable that a special arrangement of nerves should exist to cause pain and never be employed during a whole lifetime. Were the pain of angina pectoris, for instance, evoked through stimulation of cardiac sensory nerves, these nerves in the vast majority of people would never be exercised. The means by which this is brought about is probably by the excessive stimulation of the sympathetic nerves in visceral disease. The performance of the visceral functions passes unnoticed by the individual. From the brain and spinal cord to the viscera, and from the viscera to the brain and spinal cord, a flow of nervous impulse is nevertheless constantly maintained. When from any cause the strength of the afferent impulse is increased the stimulation arising in the brain or cord affects neighbouring cells which are in connection with nerves supplying skin and muscle of the external body wall. The individual then becomes conscious of this by feeling pain in the peripheral distribution of these nerve centres thus stimulated, and certain muscles likewise become tonically contracted or more readily liable to contraction, in order to protect the underlying viscera. Thus peristalsis of the bowel takes place, and the individual is unconscious of the process. But when the peristalsis becomes violent he becomes conscious of the fact, but it is revealed to him as a severe pain in the nerves of the skin and muscle of the abdomen.

III. Evidence of the Insensitiveness of the Viscera.—(*a*) *Experimental.* — Numerous experiments have been made on animals, demonstrating the insensitiveness of the viscera. Haller, in 1752, published a series of observations where he had exposed the various internal organs and submitted them to a great variety of injuries without evincing the symptom of pain or other sign of discomfort in the animals operated upon. These observations have been in a great measure corroborated by later experimentalists.

(*b*) *Clinical.*—The clinical evidence is equally convincing, although the almost universal testimony of medical experience is seemingly opposed to this view. Medical men are daily finding evidence of what they assume to be sensitive organs. Pressure over an enlarged liver causes

pain, or gentle pressure over the abdomen in peritonitis is felt as pain by the patient. This is too often supposed to be conclusive evidence of the tenderness of the visceral structures. This is, however, not evidence that the viscera are tender; the pain, as a matter of fact, being evoked by the stimulation of the hypersensitive external body wall covering the diseased organs.

Numerous instances occur scattered through medical literature where it has been observed that organs exposed in the human subject have shown no signs of sensation when stimulated. For many years I have taken every opportunity afforded me to test the sensibility of the viscera when the organs have been exposed through accidents or operative procedures. In the case of the heart and lungs the observations have been conducted on patients from whom a piece of rib has been removed for the relief of an empyema. The visceral layer of the pleura has never shown any sensation when pinched or pricked. The parietal layer has been equally insensitive, but when hard pressure has been made a sensation of pressure has been felt, evidently because the structures of the external chest wall were being pressed upon. In a few cases I have been able to reach the pericardium and exercise pressure against the heart. In no case has any sensation resulted.

In numerous cases I have been able to test the insensibility of the bowel. In one unique case I had an excellent opportunity of demonstrating this insensibility. The patient, a man aged fifty-six, had suffered from an umbilical hernia for many years. He had attempted to keep it back by tightly applied bandages. Ulceration of the skin followed, and this extended so deep at one place that the wall of the bowel (being adherent to the parietal peritoneum) was ulcerated through, and a fistula established. When I saw him, in consultation, I found in addition that an area of skin about three inches in diameter around the fistula was completely gone. It was resolved to attempt to suture the gut. The area denuded of skin being nearly insensitive, and as the patient objected to chloroform, we made the incision into the abdomen through the denuded surface. When the contents of the abdomen were exposed a portion of the bowel was drawn out, old and recent adhesions were broken down, a piece of the ulcerated bowel and underlying mesentery were removed, and the cut ends of the bowel sutured. All this was done while the patient was conscious and intelligent, and repeated inquiries during the operation elicited the statement that he felt no pain. Opportunity was also taken to demonstrate the insensitiveness of the liver. But there were times when he groaned and complained of pain. This occurred whenever the upper end of the cut bowel (small intestine) passed into peristalsis. This portion of the bowel was treated first and laid to one side of the abdomen. At varying intervals it would change from a large flaccid tube to a narrow fleshy rod. Whenever this occurred he groaned. Asked where the pain was felt, he passed his hand over the umbilical region, although the contracting piece of bowel was at least twelve inches away.

IV. Nature of the Pain evoked by Stimulation of the Viscera.—Although no evidence of sensation is discoverable by direct stimulation, yet in some instances pain can be evoked when the stimulation is adequate. In the above instance pain was seen to occur when the non-striped muscular fibres contracted vigorously, but the pain evoked was not felt in the contracted bowel, but was referred to the region of the umbilicus. As I shall show later, this is the region to which the pain of the small intestine is usually referred. An adequate stimulus can be employed by causing non-striped muscles to contract, as by giving a distending enema and so stimulating the lower end of the great bowel to violent spasm. The pain then is always referred to the front of the abdomen below the umbilicus, and is not situated in the bowel itself. A pain in the middle of the forehead in many people is evoked on swallowing a piece of ice; the stimulation arising in the œsophagus, passing probably by the vagus, and stimulating a portion of the fifth cranial nerve.

One occasionally meets with other instances where the stimulus is adequate. Thus, Mr. Caird tells us that once, when incising the peritoneum above the pubis in a conscious patient, severe pain was felt but was referred to the point of the penis.

Pain of a referred character can sometimes be roused by exercising pressure on a glandular organ. It is difficult to differentiate between a pain actually due to an affected organ and the stimulation of the sensory nerve in its protective covering. It is for this reason that clinical observations are apt to be misleading. It may, however, be stated broadly that the sensibility of an organ or tissue to direct pain depends upon the amount of nerve supply from the cerebro-spinal system. So far as I am aware, apart from the vagus and phrenic nerves, no such supply has been traced to the abdominal or thoracic viscera. I have in several instances carefully dissected intercostal and abdominal nerves from their origin in the spinal cord to their peripheral distribution, but could detect no twig entering the pleura or peritoneum.

The demonstration of the truth of these assertions (the referred pain due to the stimulation of the parenchyma of an organ, and the direct pain due to the stimulation of the sensory covering of an organ) can best be shown in the study of the sensory relationship of the testicle.

V. The Sensory Supply of the Testicle.—For a long time I was hampered in the enunciation of the view that all pains arising

from the viscera were referred pains, by the demonstrable fact that pressure on the testicle, as well as the pain in inflammation of that organ, called forth a direct pain. One day, while tapping a hydrocele, I laid hold of the testicle and touched the organ very lightly with the end of the canula. The patient immediately called out that it hurt, and referred the pain unerringly to the site touched. Shortly after I was demonstrating this symptom in another case of supposed hydrocele, where, however, the stimulation evoked no sensation whatever, and I concluded that this could not be a case of hydrocele. I subsequently operated on the patient, and found a small omenta hernia lying in a sac distended with fluid. What I stimulated in this case by the canula was the inside of the hernial sac, whose insensitiveness was therefore in striking contrast to the extremely sensitive testicle. The explanation is to be found in the fact that a branch of the genito-crural nerve is distributed to the testicle and its coverings. As the tunica vaginalis is in reality of the same origin and nature as the peritoneum, this is the only instance in the body of a serous membrane being supplied by a sensory spinal nerve. I do not think it would be straining the argument to state that the difference between the sensibility of the testicle and other viscera lies in the fact that the testicular covering alone is supplied by a spinal sensory nerve.

A pain of a different character and situation can sometimes be demonstrated on applying pressure to the testicle. If we take a patient with a long pendant testicle and apply pressure to the organ, the individual is sometimes cognisant of two pains, one at the point of pressure and another situated in the groin. In people with short cords it is not always easy to distinguish between the two pains, but with careful observation on an intelligent individual the distinction can be made out. This pain in the groin is evidently a referred pain. It is somewhat similar in situation and in character to the aching one finds in some cases of varicocele. It is also felt very severely in orchitis.

It is possible that pressure on the kidney and ovary may elicit a similar referred pain, but I have not had the opportunity of observing with sufficient exactness the nature of these pains on account of the confusion arising from the stimulation affecting the nerves of the external body wall.

VI. The Sensory Supply of the Orifices of the Body.—A difficulty arises in investigating the orifices of the body. The sensibility of mucous membranes appears to be distinctly limited to the areas supplied by spinal sensory nerves. Beyond the limits of the distribution of the spinal sensory nerves the sensation of pain when evoked is referred to some part of the body wall.

VII. Clinical Evidence of the Referred Nature of the Pain.—Confirmatory evidence of the referred character of the pain in visceral disease is shown in the distribution of the area of the pain in certain affections and in the presence of other symptoms associated with the pain, as hyperæsthesia of the external body wall.

(*a*) *The Radiation of Pain.*—If we consider the area in which pain is felt in certain forms of angina pectoris, we shall find that only by viewing the pain as referred can a suitable explanation be arrived at. Many patients complain of the pain in angina pectoris as starting over the præcordium. The pain, increasing in severity, passes outwards towards the left axilla, and strikes down the left arm, usually on the inner surface of the upper arm and ulnar border of the forearm. In some cases the pain is almost entirely limited to the arm. The area of the external body wall here described is one supplied by a series of spinal nerves whose spinal roots are contiguous, *i.e.* the fourth, third, second, and first dorsal nerves. The pain does not radiate up the chest as high as the clavicle, for the skin over the clavicle and on the chest as low as the second rib is supplied by the third and fourth cervical nerves.

The radiation of the pain in renal calculus affords a similarly striking evidence of the referred character of the pain. The pain in this complaint frequently begins in the back in the lumbar region, and passes round to the front, slanting downwards over the lower part of the abdomen into the testicle. Because the ureter runs in a somewhat similar direction for a short distance, it has been assumed that the pain was due to the stone scratching along that tube. But the course here described corresponds to the distribution of the twelfth dorsal nerve, and evidence of a hyperæsthetic condition of the tissues supplied by this nerve can often be demonstrated. A branch of the genito-crural nerve from the first or second lumbar nerve supplies the tunica vaginalis, and hence the extension of the referred pain to the testicle.

(*b*) *The Distribution of the Hyperœsthesia.*—A still more striking evidence of the referred character of the pain is to be found in an exaltation of sensibility of the tissues of the external body wall in the area where pain is felt. This hyperæsthesia or hyperalgesia is not present in all cases. When it does occur it corresponds in area with the situation of the pain. If we take the instances of heart pain or the pain of renal colic given above, we find most convincing evidence of the referred character of the pain. Thus in a patient suffering from disease of the aortic, mitral, and tricuspid valves, the pain was first felt in the outer part of the left chest. This pain was followed by a tenderness of the skin which showed cutaneous hyperæsthesia well marked in the area shaded

in Fig. 1. After several attacks the pain was felt over a more extensive area in the chest, and extended into the left arm. This area became also very hyperæsthetic, and corresponded to the shaded area in Fig. 2.

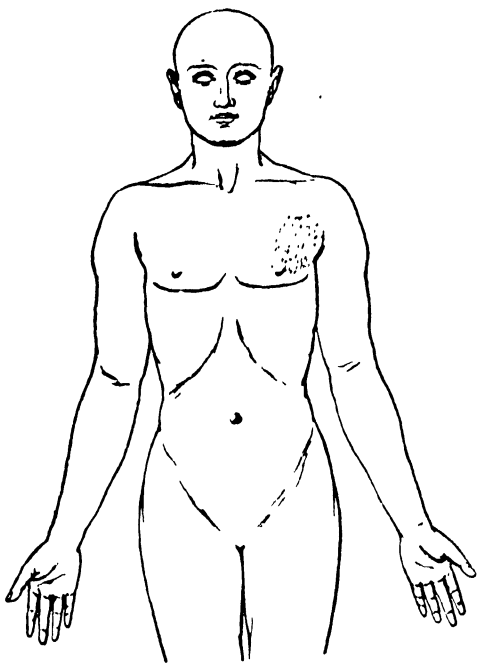


FIG. 1.—The shaded area shows the situation of the pain and hyperæsthesia after the first attack of angina pectoris

The hyperæsthesia in renal colic shows a similar relationship of cutaneous tenderness with the area of pain. In Fig. 3 the shaded

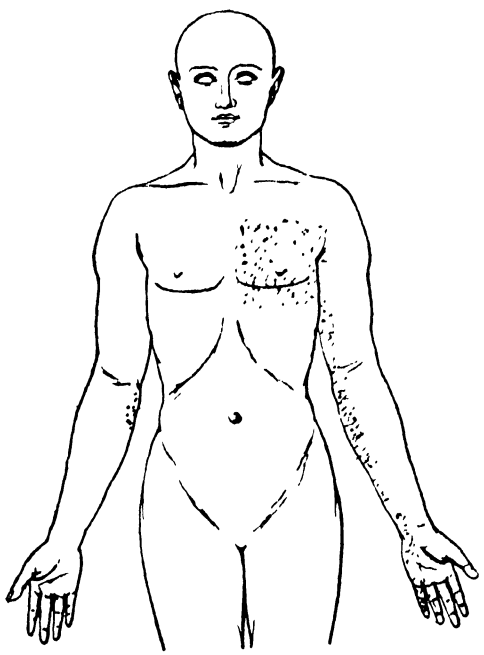


FIG. 2.—The shaded areas show the distribution of the pain and hyperæsthesia after repeated attacks of angina pectoris. Note that the inner surface of the right elbow was also hyperæsthetic, and the pain was also severely felt here.

area shows the hyperæsthesia in a patient suffering from renal colic, who passed a small calculus a few days after the onset of the pain. The pain radiated to the testicle, and this organ was extremely tender on light pressure. It will be observed, however, that the shading showing the cutaneous hyperæsthesia stops short at the groin, the skin of the scrotum is not altered in its sensibility, while, as I have said, the testicle was tender to light pressure. The reason for this is that the shaded area represents roughly a portion of the area of cutaneous distribution of the twelfth dorsal and first lumbar nerves, and as the latter nerve also supplies the deep covering of the testicle, the testicle is tender to pressure. The skin of the scrotum receives its nerve-supply from distant nerves, namely, the third and fourth sacral, and their centres in the spinal cord are therefore unaffected by the stimulation.

VIII. THE CHARACTER OF THE CUTANEOUS HYPERÆSTHESIA.—In a certain number of cases after an attack of pain due to some visceral

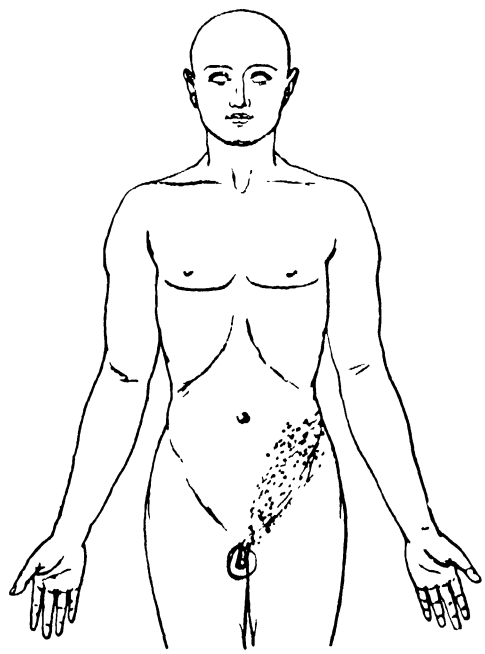


FIG. 3.—The shaded area shows the distribution of the hyperæsthesia in a case of renal calculus.

disease, the tissues forming the external body wall become hyperæsthetic. This hyperæsthesia can be demonstrated to affect not only the skin but the deeper structures of the external body wall, more particularly the muscles. Glands such as the mammary also become tender to pressure. In addition to the tenderness of the muscles there is a great tendency for the muscles to become tonically contracted.

The hyperæsthesia of the skin shows itself in two forms. Sometimes the skin is so tender that the patient cannot bear the clothing to touch the part. At other times the light stroking of a pin calls forth the sensation of pain instead of that of touch. This form of hyperæsthesia may be called epidermic or superficial cutaneous, because it appears when the stimulus applied affects the surface of the skin.

Another form of cutaneous hyperæsthesia can be demonstrated when the skin is gently

pressed between the thumb and forefinger. By this procedure we can elicit exalted tenderness, when the surface of the skin shows no increased sensitiveness. This deep cutaneous tenderness is always present with the superficial, and extends over a greater area than the superficial. Thus if one first map out an area of hyperæsthesia by gently stroking with a pin-head, then map out the area by gently pinching the whole skin, the latter area will be found to be more extensive and to embrace the former area.

Another point to be made out is the fact that the borders of these areas are ill defined. In mapping out the borders one finds that the patient has sometimes difficulty in saying whether the skin tenderness is increased or not. Sometimes, again, while the patient has no doubt as to the increased sensitiveness, on again testing the same place after a few minutes the patient confidently asserts that the same place is not hyperæsthetic. While the edges of a given patch of hyperæsthesia are variable, the centre is usually invariable in its manifestation of increased sensibility. This same variability of the edges of a hyperæsthetic area is also present in the hyperæsthesia due to disease and to injury of the spinal cord itself. The reason is probably due to the fact that, as has been shown by Sherrington, neighbouring nerves overlap in their distribution, so that the same piece of skin is supplied by more than one nerve, and it is only when the centres of two neighbouring nerves are affected that an area of skin shows lasting hyperæsthesia.

The areas of hyperæsthesia, besides being of doubtful extent, are also of very irregular distribution. It is seldom that one gets the same exact distribution of hyperæsthesia in two individuals suffering from the same complaint. Since I first drew attention to the occurrence of hyperæsthesia as a diagnostic sign in visceral disease, an attempt has been made to map out the greater part of the body into areas supposed to represent the peripheral distribution of individual sensory nerves, by means of the hyperæsthesia present in visceral disease. But if these areas be carefully studied they will be found to afford no safe guide for such accurate delimitation. Hyperæsthetic areas undoubtedly occur in the field supplied by spinal nerves whose centres are associated with the sympathetic or other nerves supplying the viscera affected. But it is doubtful if a single nerve in its entire distribution is ever alone affected. The stimulation from the viscera seems to affect portions of the centres of several nerves that supply the same or contiguous areas of skin.

IX. Muscular Hyperæsthesia and Muscular Contraction.—It is only in recent years that muscles have been demonstrated to be provided with sensory nerves, notwithstanding the fact that hyperæsthesia of the muscles is a common and readily demonstrated symptom. In nearly all cases exhibiting cutaneous hyperæsthesia, muscular hyperæsthesia is also present. Muscular hyperæsthesia in such cases is not usually easily demonstrated, because pain is at the same time evoked by pressure on the skin when the muscle is stimulated. But a large number of cases which show no cutaneous hyperæsthesia readily show extreme tenderness when the muscle is stimulated. This muscular tenderness is often present with a readiness to contract when stimulated. The symptoms of muscular hyperæsthesia and muscular contraction are of much more frequent occurrence and of more clinical value than the cutaneous hyperæsthesia. They appear after an acute attack of pain, as after an attack of biliary colic, or they may be present in the absence of severe pains, where the patient complains only of a soreness when the muscles are pressed upon or when they are called into action, as in coughing or on taking a deep breath. In pleurisy the severe stabbing pain that is occasioned by deep breathing is due to the violent or spasmodic contraction of the hypersensitive intercostal muscles. Examination of these conditions of the muscles is best carried out in the broad muscular sheets that form the abdominal wall. These muscles possess the faculty of tonically contracting in their entirety or in sections. When all the muscles are contracted one feels the abdomen hard like a board. When only a small portion of the muscle is contracted, as in the case of a portion of the rectus muscle, between two intermuscular septa, the contracted muscle feels like a hard tumour in the abdomen. A similar deceptive feeling occurs when a piece of the internal oblique in the iliac region is contracted.

In addition to these contractions, the muscle is always more or less tender. One can often observe that a muscle may be but slightly contracted when very gentle pressure is applied, and it may at once become hard and board-like on increasing the pressure. The muscle being at the same time hypersensitive, the increased pressure calls forth the symptom of pain. It is really this painful stimulation of the abdominal wall that observers so frequently mistake for the tenderness of the underlying viscera. Thus an enlarged liver, such as occurs in advanced heart failure, seems extremely sensitive to gentle pressure. If, however, one is able to lift gently a piece of the muscle away from the organ in suitable cases, by inserting the finger into the umbilicus, and gradually compress the muscle, the tenderness will be found to be in the muscle. Or, after carefully mapping out the lower border of the liver, the tenderness will be found to extend over a greater area than the liver.

This tenderness of the muscles of the abdominal wall is often mistaken for local peritonitis. One finds frequent statements to that effect in recent text-books. I have known a very experienced surgeon diagnose a local peri-

tonitis after an attack of gall-stone colic because of this muscular tenderness. Yet this tenderness was present not only in the muscles of the abdominal wall, but extended to the muscles in the thorax, as high as the pectoralis major. So tender, indeed, may the muscles of the right side of the thorax become after an attack of gall-stone, that I have seen it ascribed to an attack of pleurisy.

X. The Spreading of Symptoms.—The spreading of the stimulus to nerve centres in the spinal cord has been incidentally alluded to in the case of heart and renal pains, and illustrated in Figs. 1, 2, and 3. This spreading may affect other than sensory nerves. Thus muscular spasm may be set up in parts more or less remote, and give rise to misleading symptoms. In a severe case of renal colic due to a calculus in the pelvis of the kidney or in the ureter, the whole of the abdominal muscles may be thrown into strong contractions. Not only may the muscles forming the anterior abdominal wall be affected, but the sphincters may become strongly contracted, the result being that neither flatus nor fæces can be passed. After a few days the abdomen may become so distended that there is presented a variety of symptoms closely resembling those of obstruction of the bowel. Careful attention to the history of the case and to the site of the pain will aid in arriving at a correct conclusion.

The spreading of the stimulus to the sensory nerves may be revealed in other ways than by cutaneous and muscular hyperæsthesia. So sensitive do the centres of the spinal nerves become, that however the stimulus reaches them pain is evoked. Thus after an attack of gall-stone colic, ingestion of the food into the stomach sometimes causes great pain referred to the epigastrium. The explanation seems to be that the centres of the sensory nerves in the spinal cord are so extremely sensitive that any stimulus reaching them evokes the sensation of pain. The ingestion of food sends a stimulus, probably by the sympathetic, to the spinal cord, and this, which under ordinary circumstances gives rise to painless stimulation, now produces the symptom of pain, just in the manner that a light touch to the hyperæsthetic skin gives rise to a sensation of pain. It is a matter of common experience to find organs remote from the part stimulated affected in their functions. Thus nausea and vomiting are common in labour, in renal and hepatic colic. The secretion of glands may be affected, as in the intense salivation sometimes present in angina pectoris. While the symptoms are often limited to one side of the body, the stimulus may affect the spinal roots supplying the opposite side. Thus the heart pain sometimes radiates to the right arm (*see* Fig. 2).

XI. Tender Nerves and Tender Spines.—The small nerve-trunks running to the regions in which pain is felt may become very tender to pressure. As several of these have a permanent situation and are readily accessible to pressure, the points of tenderness have sometimes a diagnostic significance. Thus a branch of the ninth dorsal nerve as it pierces the sheath of the right rectus muscles two inches from the middle line, and a little higher than the level of the umbilicus (Fig. 4), is frequently sensitive in gall-stone colic. In cases of doubt I have come to look upon it as almost diagnostic of that disease. So also a twig of the twelfth dorsal nerve as it crosses the iliac crest three inches behind the anterior superior iliac spine is frequently very tender in renal colic due to a calculus. A small twig from the first dorsal nerve (the internal anterior thoracic) is sometimes tender in heart affections as it courses over the second and third left ribs in the nipple line.

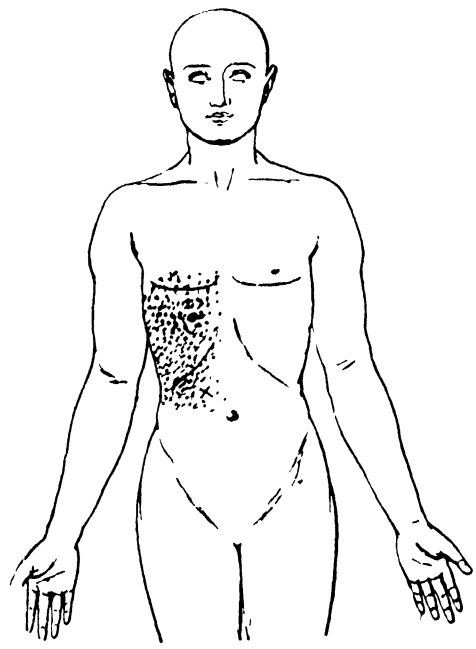


Fig. 4.—The shaded area shows the distribution of hyperæsthesia in a case of gall-stone colic. The X indicates a spot very commonly tender to pressure in this complaint.

Certain vertebral spines have a tendency to become tender on pressure, and to resemble the tenderness due to diseases of the vertebra. This spinal tenderness, when associated with a band of hyperæsthesia, is sometimes mistaken for spinal disease. I have found the upper dorsal spines tender to pressure in heart disease, the fourth to seventh dorsal vertebra tender in affections of the stomach, and the sixth to the ninth tender in hepatic colic. This tenderness is not due to the pressure applied to hyperæsthetic skin covering the spinous processes, for it is present when pressure is applied when neither the skin nor deeper tissues are hyperæsthetic.

XII. Visceral and Cutaneous Muscular Reflexes.—The muscles of the external body wall are influenced in a twofold manner by

visceral affections. In Paragraph IX. attention was drawn to the muscular contraction in visceral disease. This may be spoken of as a *viscero-muscular reflex*, inasmuch as the stimulation passes from the affected viscera by the sympathetic to the centre of the muscle nerves in the cord. It is characterised by long-con-continued contractions of the muscles, and is in striking contrast to the short, rapid contractions of the cutaneous muscular reflex.

Another peculiarity is that the muscular contraction due to visceral stimulation does not readily relax under chloroform anæsthesia. Thus we may push the anæsthetic till all the other voluntary muscles are relaxed and sensation abolished, and still the visceral-stimulated muscle remains contracted. This is particularly the case with the abdominal muscles, where the rigid recti sometimes interferes with the surgeon's freedom. In a case of stone in the bladder I found the lowest segment of the left rectus muscle firmly contracted and tender on pressure. It remained so during complete anæsthesia and till the bladder was opened (supra-pubic operation). The patient became sick, and the muscles relaxed and did not again contract.

A number of causes produce the muscular contraction, as contraction of hollow muscular organs, inflammation of the peritoneum, affections of the parenchyma of organs, etc. When more is known of the exact relationship of the sympathetic nerve supply to the cerebro-spinal nerves, this symptom will be of great diagnostic value.

With hyperæsthesia of the skin there is also increase in the activity of the muscular reflexes. This is well illustrated in gastric affections with hyperæsthesia of the skin of the epigastric region. A slight stroking of the skin here will call forth strong, rapid contraction of the upper portion of the left rectus muscle. Not only is the muscular response abnormally easy to elicit, but it may also be called forth by stimulation of the non-hyperæsthetic skin at a remote distance, as, for instance, by stimulating the skin in the axilla. This would point to the fact that the centres of the muscular nerves are abnormally sensitive to stimulation.

XIII.—Sensory Symptoms of Individual Organs.—Before dealing with the sensory symptoms of individual organs, it is necessary to state that it is often impossible to locate with certainty the cause of many pains. It is difficult to tell, for instance, whether any given pain may not arise from affections of the body wall itself. A pain over the lower part of the back may be a referred pain from some internal organ, or it may be due to some rheumatic affection of the muscles, as lumbago. Severe pains in the arm may be of peripheral origin, as in some forms of neuralgia, or they may be due to an affection of the heart. Again, there may be diseases which affect more than one tissue. Thus appendicitis may set in with severe peristalsis of the bowel, giving rise to the characteristic cramp-like pains. With extension of the disease a peritonitis may be set up with the characteristic sensory phenomena. Suppuration may follow in the tissues of the abdominal wall, producing pain and other symptoms of a purely local character.

There are often indefinite pains which are difficult to locate, particularly as many patients describe the sensations in very vague language. It often happens that the pain is felt in a somewhat diffuse fashion. In speaking of the spread of the hyperæsthesia, I drew attention to the fact that it spread along districts supplied by portions of nerves whose roots were contiguous, and did not embrace the whole distribution of any one nerve. As visceral pain often occurs without any hyperæsthesia being present, we are dependent entirely on the patient's description for discovering where the pain is felt. The patient frequently gives a totally wrong description of the site when describing it from memory. But if asked to note carefully the place where the pain is felt during a subsequent attack, much more reliable information can usually be obtained. But even after that there are a great many pains which, in the present state of our knowledge, it is impossible to locate with certainty.

In describing the pains and other sensory phenomena associated with visceral disease, it will be better to consider the subject from the standpoint of the tissues affected, rather than from the point of view of the organ as a whole. Certain kinds of tissue give rise to pains that bear a somewhat similar resemblance in character. Those organs containing non-striped muscles present a certain similarity of symptom, while affections of the membranes present a different kind of symptom. For this reason the subject will be considered according as the pains are associated with (*a*) hollow muscular organs, (*b*) serous membranes, (*c*) parenchymatous tissue.

(*a*) *The Hollow Muscular Organs.*—In a great number of instances there is a certain resemblance between the character of the pain in affections of the hollow muscular structures. It has been pointed out in describing the peristalsis of the bowel in Paragraph III. that coincident with the contraction of the bowel a pain was felt. The character of the pain was typical. It gradually increased in intensity, reached its height, and gradually subsided. The character and duration of the pain agree with the character and duration of the muscular contraction. The muscle contracts slowly, increasing in force till fully contracted, and then gradually relaxes. Such is the character of the pain to be found at times in all hollow muscular organs—the digestive tube, gall-ducts, ureter,

bladder, uterus. In the heart a pain of a similar character may occur, but there is no reason to suppose that the muscle remains spasmodically contracted, seeing that there may be no interference with the rhythm of its contraction. In other instances the pain may not have this definite wavy character. The contraction of the tube may continue for a long period, and pain of the most intense character may persist for hours. Or the contraction may be less severe and the pain less intense.

There is a tendency for organs that are single to have the pain referred to the middle line, though it may radiate to one side. Thus the cramp-like pains due to severe peristalsis of the bowel are almost invariably situated across the middle line. So also are the uterine pains due to contraction of the bladder. The pain of the stomach is also most often felt in the middle line, but generally radiates to the left, while the pain in gall-stone colic, also frequently situated in the first instance in the centre, radiates generally round the right side of the body. The pain due to renal calculus is always one-sided. The pain from the heart is often felt across the breast bone, but usually radiates towards the left side.

The Digestive Tube.—It cannot be demonstrated in all cases what it is that actually causes the pain in affections of the digestive tube, but in a great many cases it can be reasonably inferred that strong peristaltic contraction of the muscle is the exciting cause. Thus, if we study the symptoms of obstruction of the bowel, it will be found that the portion of intestine above the obstructed point is at times thrown into strong peristaltic contractions, and at the same time severe pain is felt. If the situation of the pain be noted, it will be found to bear a distinct relationship to the piece of bowel in which the increased peristalsis occurs. Thus the spasm of the muscular wall of the stomach causes pain to be felt in the epigastrium, spasm of the small intestine about its middle causes pain in the umbilical region, and spasm of the lower end of the great bowel causes pain above the pubis. Pain felt in places between these points would indicate peristalsis of a portion of bowel higher or lower than those parts mentioned, according as the site of pain is higher or lower in the abdomen. Apart from cases of obstruction, one finds evidence of the truth of this view in cases of severe abdominal pain, when the contents of the bowel set up violent peristalsis. Thus the pain may be felt higher than the umbilical region. A desire to have the bowels relieved may be felt by the patient, but no relief is obtained. The pain continues, but gradually descends, is felt in the umbilical region, then gradually lower, till it is felt above the pubis, when the necessity for relief becomes at once urgent, and with relief of the bowels the pain ceases. Here it is evident that the peristalsis has been driving the contents of the bowel lower down, and as parts of the bowel passed into violent peristalsis the pain was felt lower in the abdomen.

While the above statement holds true for the digestive tract as a whole, it also holds true for more limited portions. Thus, if the situation of pain in gastric ulcer be studied, an idea can be formed of the site of the ulcer. In the majority of cases of gastric ulcer there is a localised pain referred to the middle line in the

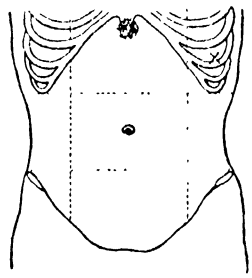


FIG. 5.—The shaded area shows the site of pain; X corresponds to the position of the ulcer.

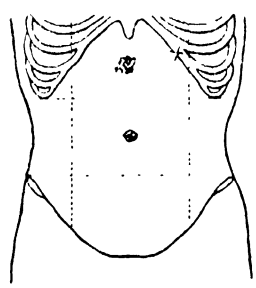


FIG. 6.—The shaded area shows the site of pain; X corresponds to the position of the ulcer.

epigastrium. The patient can often put the finger on a certain spot and say with decision that here the pain is most intense. There is often found at the same time a limited area of hyperæsthesia. If the situation of this pain be high up in the epigastrium, as in the dotted area in Fig. 5, the site of the ulcer will be near the cardiac orifice of the stomach. The **X** marks the position of the ulcer in relation to the external body wall in one particular case. If the site of pain be felt in the middle of the epigastrium, as in the dotted area of Fig. 6, the ulcer will be found near the middle of the stomach. Fig. 6 is from a patient who had a ruptured gastric ulcer, and the site of the ulcer was reasoned out from the position of the pain previous to the rupture. The patient was operated upon within fifteen hours of the rupture; the perforation was found in the middle of the stomach on the lesser curvature, was sewn up, and resulted in a good recovery. I mention this, as the knowledge of where to look saved the risk of blindly exploring, whereby the contents of the stomach might be squeezed out of the perforation. When the site of pain is low in the epigastrium, as in the dotted area of Fig. 7, the ulcer will then be at the pyloric orifice of the stomach. The above reasoning may not be absolute, nevertheless I have found it invariable in over a dozen cases, where the position of the ulcer has been verified by post-mortem examination or by operation. It occa-

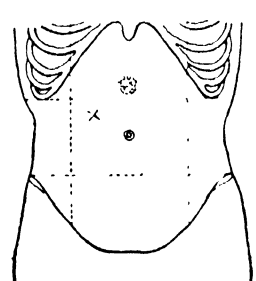


FIG. 7.—The shaded area shows the site of pain; X the site occupied by the ulcer at the pyloric orifice of the stomach.

sionally happens that the localised pain is not situated in the epigastrium, but when we know more of the exact relationship of the stomach and the skin areas the localised pain will become a guide to the site of the ulcer. While there may be a localised pain in gastric ulcer, the pain and hyperæsthesia may radiate very widely. Usually it passes round the left side. A band of hyperæsthesia passing from the epigastrium round to the back, with the seventh and eighth spines tender to pressure, may simulate disease of the vertebræ. The hyperæsthesia may extend widely up into the chest and down into the abdomen. The epigastric reflexes in such cases are always greatly exaggerated. It is not easy to decide on the immediate cause of the pain in gastric ulcer and indigestion. There is absolutely no evidence of sensitiveness of the mucous membrane. Contraction of the muscle does give rise to pain, and it is possible that it is the muscular coat that is affected, being more readily stimulated to contraction on account of the disease. In Beaumont's experiments on the man with the gastric fistula, the only record of pain arising from his stimulation of the internal surface of the stomach occurred when he produced strong contraction of the pyloric end.

Gall-Ducts.—The first onset of pain in gall-stone disease may suddenly become very severe. It is often referred to the middle line, in the lowest part of the epigastric region and adjoining part of the umbilical region. From here it may radiate widely across to the left side, but more usually it passes round the right side over the lower ribs to the back to the right of the eighth, ninth, and tenth dorsal vertebræ, and here the pain may also be felt with great severity. The attack of severe pain may last for many hours, and be accompanied by severe nausea. The attacks may gradually pass off and not recur for some lengthened period, or they may recur with such frequency that the patient is seldom free from pain for weeks and even months. After a severe attack of pain there is often extreme hyperæsthesia of the external body wall. It was in a patient suffering from this complaint that I first made the observation of hyperæsthesia in visceral disease. A few days after an attack of colic he called on me, holding his right arm stiffly away from his body. Asked why he adopted this attitude, he replied that the contact of his clothes to his skin caused him great pain. On stripping him, I found an area of cutaneous hyperæsthesia corresponding to the shaded area in Fig. 4. The hyperæsthesia in cases of gall-stone colic may extend more widely, and be present in the muscles not only of the abdominal wall but as high as the right pectoralis major. I have already mentioned that ingestion of food may also cause pain, and for this reason such ailments may be mistaken for diseases of the stomach.

Many cases of gall-stone colic are difficult to diagnose because of the vagueness of the symptoms. In many such doubtful cases I have found almost convincing evidence in the presence of a spot peculiarly tender to light pressure, exactly in the position of the X in Fig. 4. Here a twig of the ninth dorsal nerve emerges from the muscle and is readily affected by the pressing finger. This observation I find has been independently observed by Mayo Robson, whose practical knowledge of this disease is very extensive.

The cause of the pain in this disease I assume to be due to the strong contractions of the muscular coat of the gall-bladder or gall-duct upon the stone, the latter acting as the exciting cause. The hyperæsthesia results from over-stimulation of the centre in the spinal end of the nerves supplying the lower part of the chest and upper part of the abdomen, and these centres remain abnormally excitable after the muscular spasm has relaxed.

The cutaneous and muscular tenderness are often stated to be evidence of an accompanying peritonitis, but it is not necessarily so. While doubtless a localised peritonitis may accompany gall-stone disease, and hyperæsthesia be a symptom of peritonitis, yet hyperæsthesia is such a constant accompaniment of muscular spasm that we are not justified in diagnosing peritonitis from its presence.

Ureter.—The pain arising from a stone either in the pelvis of the kidney or in the ureter is usually very well defined in its distribution. It begins in the back in the lumbar region, radiates round the side, and descends the front of the abdomen in a slanting direction to the level of the pubis. In the male it generally strikes into the testicle. There is also frequently present a good deal of hyperæsthesia of the external body wall. The cutaneous hyperæsthesia when present has a distribution corresponding to that shaded in Fig. 3. But it may be more extensive, passing down over the upper part of the thigh. On the other hand, only a small patch in the shaded area may be present. The deeper structures, and particularly the muscles of the back and front of the abdomen, are often very tender. This muscle tenderness is usually limited to the side affected, but, as I have already said, it may affect all the muscles of the abdomen. This tenderness of the muscles in the lumbar region is often assumed to be evidence of a tender kidney, as patients wince when pressure is applied. Careful examination of the muscles reveals the tenderness to be in them. A branch of the twelfth dorsal nerve passes over the iliac crest three inches from the superior anterior iliac spine, and light pressure applied over it sometimes causes pain. The testicle itself is often very tender to light pressure, due to the hyperæsthesia of its tunica vaginalis. It will

be noted that the skin of the scrotum is never hyperæsthetic, as its nerve supply is from the remote sacral nerves. This testicular tenderness is often diagnostic in those obscure cases where one has some difficulty in deciding from the patient's account whether the pain is really due to renal calculus.

Although the pain here described is most commonly associated with renal calculus, yet any other condition that gives rise to spasm of the muscle wall of the ureter will cause these symptoms. Thus, I have seen it associated with a collection of pus in the pelvis of the kidney or ureter, and it may be present with a movable kidney.

The Bladder.—The pain due to the bladder is not easy to localise. The functions of the bladder are so intimately associated with structures that are supplied with sensory nerves (as the sphincters and urethra) that it is difficult to tell whether they may not be affected. I have seen intense backache felt over the top of the sacrum in retention of urine due to spasm of the sphincter in women at once relieved on emptying the bladder. In spasm of the bladder due to cystitis or due to a stone in the bladder, the pain is commonly referred above the pubis, or in the perineum, and in the male along the urethra to the end of the penis, and the perineum may be hyperæsthetic. The sympathetic supply of the bladder is twofold, from the hypogastric plexus and from the third and fourth sacral nerves. The pain over the sacrum and above the pubis is probably due to the nerves associated with the hypogastric plexus, while the pain in the perineum and penis is due to the stimulation affecting the sacral nerves.

The Uterus.—The symptoms connected with disease of this organ are also complicated by its association with structures (as the cervix and vagina) supplied by sensory spinal nerves. In scraping the uterus, for instance, local pain is felt in stretching the cervix, but the scraping of the interior of the uterus is painless. The pain in labour is characteristic of the pain due to the contraction of non-striped muscles. Its gradual onset, increasing in severity, lasting for a shorter or longer period at its fullest intensity, then its gradual relaxation, corresponds in character and duration to the pain felt in other hollow muscular organs. That this pain is usually supposed to be in the uterus is easy to understand, as the coincidence of the pain with the contraction of the uterus is readily demonstrated. But there are other points that lead one to a different conclusion. Thus the pain is often entirely limited to the back, about the top of the sacrum. This has hitherto been explained as being due to pressure of the child's head upon nerves, as the lumbar plexus, or to stretching of the cervix. But as pressure upon nerves gives rise to pain in the peripheral distribution of the nerves, and as the nerves of the lumbar plexus are not distributed in this region, that cannot account for the pain. Further, the same pain is felt in this region when the uterus is small, with no child's head to complicate matters, as during the after-pains, as it is when the uterus is large and extends high in the abdomen.

The pain in labour is also felt across the abdomen, referred usually to the lower part, whether the uterus is full and large, containing a full-time child, or when it is small and situated entirely in the pelvis. In a few cases I have seen extreme pain result from the entrance into the womb of water while the patient has been syringing the vagina. The intense pain over the lower part of the abdomen, induced evidently by spasm of the uterus, has been accompanied by great hyperæsthesia, and such tenderness of the abdominal muscles that the condition resembled what one finds in acute peritonitis. On rare occasions I have found the pains in labour almost entirely referred to the upper part of the thigh.

The Heart.—There are two very striking points in connection with the pain in heart affections: first, that hearts similarly diseased may exhibit a great difference in regard to the sensory symptoms. Thus, in cases of aortic disease, pain may never be present, while in other cases pain of the most intense description is a characteristic feature. In degeneration of the heart muscle and in disease of the coronary artery the same uncertainty in regard to the presence of sensory symptoms is found. The second point is that pain and the other sensory phenomena when present do not afford of themselves a clue to the nature of the disease, as one finds a similarity of symptom in the most diverse forms of disease.

The consideration of these points suggests that the pains may have a common origin, and so far as I have been able to reason out the subject, I am disposed to view them as being essentially due to muscular stimulation. In the great majority of cases with heart pain, the attacks come on when the weakened organ is exposed to a strain beyond its strength. Thus hill-climbing is a fruitful source of heart pain in all predisposed subjects. Vaso-motor contraction is another frequent cause, as when a susceptible patient is exposed suddenly to cold, as on going out of a warm house on a frosty morning. Vaso-motor contraction also causes the pain in the attacks of increased arterial pressure seen often in the elderly, the gouty, and the subjects of Bright's disease.

The similarity of the attacks of pain in heart affections to the pain of other hollow muscular organs is also very striking.

The pains are usually felt in two fields, namely, in the areas of distribution of sensory nerves whose roots are associated with the

nerves supplying the heart, viz. the sympathetic and the vagus. The sympathetic nerve-supply is associated with the upper dorsal spinal nerves, and it is in the field of distribution of these nerves that the most typical sensory symptoms are shown. Thus the most common site of the pain is over the chest, across the middle line, or in the left side, sometimes as far out as the shaded area in Fig. 1. From here the pain radiates usually upwards to the axilla and down the left arm. The shaded area in Fig. 1 shows the site of pain and hyperæsthesia of skin after the first attack of angina pectoris in one case. The shaded area in Fig. 2 shows the area of pain and hyperæsthesia after the attacks had increased in frequency and in severity. It will be noted that the inner side of the right elbow had become also hyperæsthetic and the patient felt the pain here also.

On the whole, hyperæsthesia is not common after attacks of angina pectoris, some of the most severe cases showing no tenderness of any part of the chest wall. But the distribution of the pain has a very striking resemblance to the areas found hyperæsthetic in certain equally typical cases. In rare cases the pain may be entirely limited to the left arm.

The pain is usually assumed to be in the heart when it is limited to the chest. Seeing that it is continuous with the spreading of the pain to remote parts, as the left arm, and that it is associated at times with demonstrable hyperæsthesia of the external body wall, it seems more reasonable to assume its situation to be in the nerves thus rendered hyperæsthetic. It is sometimes entirely limited to the arm, and one of the most typical cases of angina pectoris that I ever saw had the pain entirely limited to the left forearm. Again, the pain may be limited entirely to the epigastrium. The pain may be situated in other portions of the field of supply of these nerves, as the side or back of the chest. In certain heart cases a field of pain and muscular tenderness is found to occur in the neck, the top of the shoulder, and the back of the head on the left side. When the sterno-mastoid or the upper border of the trapezius muscle is highly pinched, tenderness, sometimes exquisite, is complained of by the patient. I have observed cases where this tenderness was almost the only symptom beyond weakness in cases of heart failure, and the patient's recovery was coincident with a diminution and ultimate disappearance of this tenderness. This field of pain and tenderness is probably occasioned by a stimulus passing upwards by the vagus nerve. The origin of the vagus and spinal accessory nerves are so closely associated that one is forced to conclude that the stimulus proceeding by the one has affected either the spinal accessory nerve itself or the sensory nerves associated with it in its distribution.

(*b*) *Serous Membranes.* — I have already pointed out that the tunica vaginalis is the only serous membrane that is supplied by a spinal sensory nerve. Serous membranes lining the great cavities of the abdomen, thorax, or skull are insensitive to the ordinary means of stimulation. Pain and other sensory symptoms when evoked can be demonstrated to be present only in the tissues forming the external body wall exclusive of the serous membranes.

Peritonitis.—The pain and tenderness in this complaint can readily be demonstrated to exist either in the skin or muscles. In acute peritonitis the patient assumes a characteristic attitude—the abdomen is kept rigid by the contracted muscles, the legs are drawn up, and the respiration is almost entirely thoracic. There may be the most extreme tenderness on touching the abdomen. On careful examination this is found to be due to superficial cutaneous hyperæsthesia, and it may extend some distance beyond the limits of the peritoneum. Thus Fig. 8 shows the distribution of this hyperæsthesia in a patient suffering from acute peritonitis following upon the rupture of a gastric ulcer into the peritoneal cavity. It is seen that the hyperæsthesia not only extends up into the chest, but invades to a slight extent the skin of the thigh. In the absence of the superficial cutaneous hyperæsthesia one can readily demonstrate that the tenderness on pressure in peritonitis is either in the deeper skin or in the muscles. If one gently lifts a portion of either of these structures away from the peritoneum by deftly inserting the finger into the umbilicus, and then applying pressure, the characteristic pain is felt. In other cases where the muscle is not strongly contracted it can be

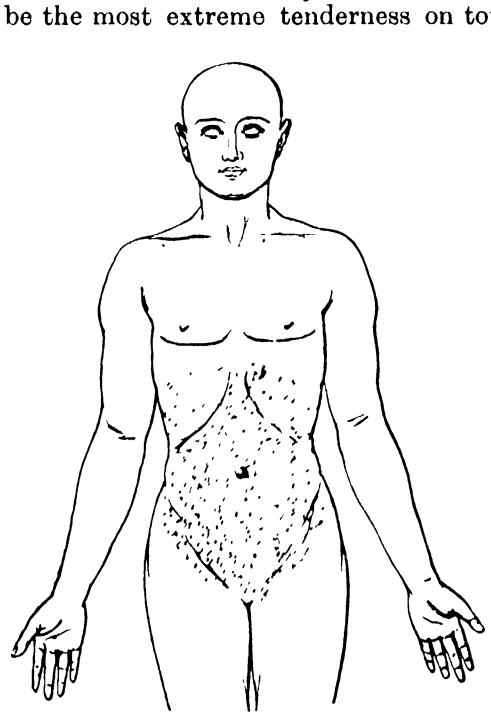


FIG. 8.—The shaded area shows the distribution of the superficial cutaneous hyperæsthesia in a case of acute peritonitis.

made to do so by gently irritating with the point of the finger.

This hardness of the muscle is often mistaken for a thickened peritoneum, and its presence on the other hand is often assumed to be evidence of peritonitis. It is necessary to insist on the fact that hardness and tenderness of the abdominal muscles may exist entirely independently of any sign of peritonitis, but may be due to affection of other tissues, as in simple gastric ulcer, renal and hepatic colic.

Pleurisy.—The pain in pleurisy may be felt immediately over the inflamed pleura, or it may be referred to a portion of skin some distance off. When its situation is immediately over the area of friction it is difficult to prove whether after all it may not be situated in the pleura. Frequently, however, it is accompanied

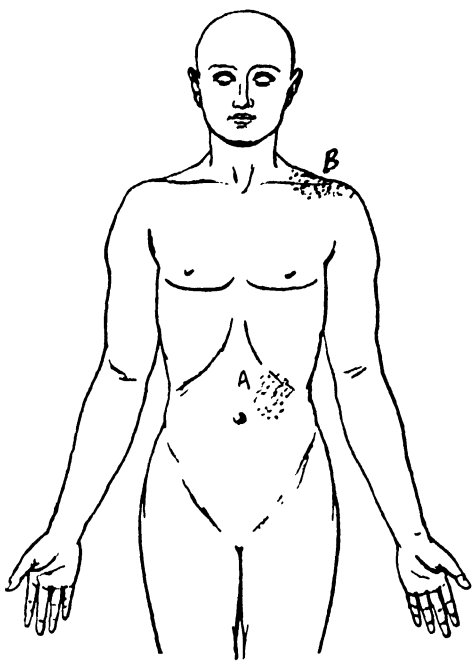


FIG. 9.—The shaded areas *A* and *B* show the situation of pain and hyperæsthesia in diaphragmatic pleurisy.

by a hyperæsthesia of the external body wall, either of the skin or muscles, and the fact that the pain and hyperæsthesia correspond in situation leads one naturally to the conclusion that the pain is in the tissues that are hyperæsthetic. This conclusion is all the more feasible when the pain and the tenderness are remote from the inflamed area. When the pleurisy affects the diaphragmatic pleura the pain is usually situated in the abdomen, sometimes as low down as the umbilicus, as in the shaded area in Fig. 9. The pain in this site is not infrequently associated with hyperæsthesia of an irregular patch of skin and a certain amount of muscle tenderness and contraction. This would suggest that the stimulus has passed by the sympathetic nerves to the spinal cord and irritated the roots of the spinal nerves supplied to the abdomen. In rare cases this diaphragmatic pleurisy is associated with great pain on the top of the shoulder, and I have on rare occasions found an area of hyperæsthesia here (B in Fig. 9). Ross drew attention to this shoulder pain and explained it by reference to the fact that this area is supplied by the fourth cervical nerve, and the phrenic nerve also comes from the fourth cervical. It may be that as the phrenic contains afferent fibres these convey the stimulation to the root of the sensory nerve, or it may be that the stimulus proceeds by way of the vagus. But there is no doubt as to the close association of this area of skin with the diaphragmatic pleura. The most severe pain in pleurisy is that due to the spasmodic contraction of the intercostal muscles. Just as the abdominal muscles contract on the slightest stimulation in peritonitis, so these hyperæsthetic intercostal muscles are stimulated to violent contraction by the movements of respiration in pleurisy.

Meningitis.—I have not sufficient data to establish satisfactorily all the sensory symptoms in this disease, but there is no reason to suppose that it differs from the general characteristics of disease of the other serous membranes. That it is, like the pleura and peritoneum, insensitive to stimulation I have been able to demonstrate in wounds of the skull. That it is characterised by the same tendency to muscular contraction is evidenced by the rigid way in which the contracted muscles of the neck hold the head.

The Pericardium.—In the great majority of cases pericarditis is not associated with any sensory symptoms. In the rare cases that I have found them, they seem to resemble the symptoms present in heart affections generally, due probably to an extension of the inflammation to the muscle wall. Thus in one well-marked case there was great pain in the left chest, extending down the inside of the arm.

(c) *Affections of the Parenchyma of Organs.*—I am extremely doubtful how far disease of organs, apart from their serous coverings, can exhibit sensory symptoms. It is well understood, for instance, that the lungs may be deeply excavated with phthisical cavities and pain may never be felt. It might seem that even in inflammation of the lungs there is no pain unless there be an accompanying pleurisy. At all events the pain and other sensory symptoms that we associate with pneumonia are identical with those observed in attacks of pleurisy, and one occasionally meets with cases of pneumonia with absolutely no pain, probably due to the fact that the pleura is not invaded. The pain and hyperæsthesia in the areas in Fig. 9 are sometimes associated with pneumonia of the base of the left lung. It is also a matter of common experience that affections of other organs are usually painless. Thus the bowels may be largely ulcerated, the kidneys acutely inflamed, and the patient never complain of pain. I have observed pain, muscle contraction,

muscle tenderness, and cutaneous hyperæsthesia accompany acute enlargement of the liver due to heart failure. But I could not convince myself that the symptoms may not have been due to the sudden stretching of the capsule, just as in acute orchitis the pain, local and referred, may be due to the stretching of the tunica vaginalis.

In ovarian affections great caution has to be exercised in demonstrating the symptoms present. A widespread hyperæsthesia, sometimes of a purely hysterical nature, is so liable to occur that no safe deduction can usually be drawn. I may, however, point out that ovarian tenderness is often described because of the pain felt on pressure being applied "over the ovaries" in the iliac region. If one studies the position of the ovaries in the pelvis it will be recognised that pressure applied here is not likely to directly affect the ovaries. In ovarian disease pain and hyperæsthesia are often present in the iliac region, and the application of pressure here calls forth the pain because it is the hyperæsthetic tissues that are being pressed upon. It often happens that the muscles here are slightly contracted as well as tender, and this tenderness is not infrequently assumed to be due to inflammatory adhesion.

The only instance where I have found what seems to be pain due to stimulation of an organ is on applying pressure to the testicle. In some cases of long pendant testicles the local and referred pain can be very instructively illustrated. Thus a man who in his employment had to lift heavy weights, complained of aching in the groin and down the inside of the thighs, particularly on the right side, had a long scrotum with the testicles at the bottom. The right testicle was small and sensitive, and on applying pressure he felt at once pain in the testicle, and then a few seconds after he groaned as if in great pain and said he felt another pain come on in the groin and down the inside of the thigh. The first pain is evidently due to stimulation of the genital branch of the genito-crural nerve, as it supplies the tunica vaginalis; the second is a referred pain due to the stimulation passing by the sympathetic to the centre of the first lumbar nerve in the spinal cord producing the pain in the region of the distribution of that nerve. This latter pain was not only delayed, but gradually grew in intensity and gradually subsided, and was accompanied by the peculiar sensation of depression characteristic of visceral pain.

XIV. Outline of Theory concerning the Relationship of the Sympathetic System to the Cerebro-spinal. — The view expressed above implies that the functions of the cerebro-spinal system of nerves and of the organs supplied by these nerves are primarily for the defence and protection of the organs necessary for the maintenance of life. The joining together of the muscles and nerves in the visceral and cutaneous muscular reflexes is evidently purposed for the protection of the economy. This view of the matter further suggests a theory of development which explains the reason for the division of pains into local and referred. In the lowest scale of animal life digestion is the principal function, and in the early stages of evolution a digestive cavity is the first semblance of an organ to appear. This is followed by a rudimentary circulatory system to carry the digested fluid to the different regions of the body. After this comes a respiratory system to purify the blood. These various organs are co-ordinated by a rudimentary system of nerves which are manifestly the antecedents of the sympathetic system. So far the external body wall is not differentiated, but is mainly a protective encasement. In further development this covering gives place to contractile and sensitive structures, so that the economy is protected from external injuries by a sensitive covering which, on painful stimulation, causes reflex action of certain muscles, and the animal is thereby removed from the offending neighbourhood. Herbert Spencer elaborates a similar idea to account for the vertebral segmentation. The nervous supply of the protective covering and that of the viscera are co-ordinated in the cerebro-spinal nervous system.

The view that the sympathetic nerve system originates independently of the cerebro-spinal system is supported by other considerations. It may be accepted as an established dictum that the more recently evolved structures are more liable to change and to disease. The period of mental growth in the human being, for instance, as well as the gradual acquisition of faculties, as speech and dexterity in manual acts, point to an increase in the functions of the brain and spinal cord, whereas the functions of the sympathetic system are as efficiently performed at birth as during later life.

It is a matter of common experience that disease is extremely frequent in the cerebro-spinal system, with a tendency to affect the most recently acquired functions, whereas disease of the sympathetic system is of rare occurrence.

The same distinction between the two systems is seen in their susceptibility to the influence of drugs. In the administration of chloroform the higher intellectual centres are the first to be affected, and after the loss of consciousness follows the abolition of sensation and muscular movement. A distinct interval elapses before the next symptom appears, which is the invasion of the sympathetic system, and is shown by the gradual abolition of respiration. Following on this comes the abolition of the functions of the heart.

This view of the independence of the sym-

pathetic system is at variance with the accepted view that the sympathetic system is but an outgrowth from the cerebro-spinal. The evidence from the latter view is based entirely on the histological examination of the embryo. Sufficient account may not have been taken in the interpretation of the observation of the effect of condensation where the development of the organs may not have been sufficiently distinctive and separate to give a clear conception of their relationship. A careful study of the functions and of the peculiarities of the two systems as above outlined certainly raises a strong doubt as to the reliability of the embryological view.

Visceroptosis. *See* ENTEROPTOSIS; LIVER, DISEASES OF (*Hepatoptosis*).

Viscosity.—The state of being viscid or adhesive, *e.g.* the state of the blood which precedes coagulation; the "viscosity value" of the blood has been estimated at five times that of distilled water. *See* BLOOD.

Viscum Album.—The mistletoe, recently recommended in arterio-sclerosis and in hæmoptysis for the purpose of lowering the blood-pressure.

Viscus. *See* VISCERA.

Vision, The Field of. *See also* BRAIN, PHYSIOLOGY OF (*Sensory Centres*); BRAIN, TUMOURS OF (*Symptoms, Sight*); CHOREA (*Symptoms, Sight*); COLOUR VISION; ECLAMPSIA; EYE, EXAMINATION OF; GLAUCOMA (*Symptoms, Impairment of Vision*); HYPNOTISM (*Special Senses*); HYSTERIA (*Sensory Disorders*); NEPHRITIS (*General Diagnosis*); OCULAR MUSCLES, AFFECTIONS OF; PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Vision, Cerebrum, Visual Centre*). —For clinical purposes the field of vision is investigated by the separate examination of each eye. Such examination may be carried out by the following methods:—

I. For rapidly testing the periphery of the visual field the fingers of the examiner are used as the test-object. The patient is placed with his back toward the light, while the examiner, standing from one to two feet directly in front of him, carries out the test. If the patient's right eye is under examination, he is asked to cover the left eye, and to do so without either pressing upon the covered eye-ball or interfering with the vision of the other eye. The examiner, having closed his own right, fixes with his left eye the right eye of the patient, while the patient fixes with his right eye the left of his examiner. In this way continued accuracy of fixation on the part of the patient is ensured, and the examiner is enabled to use the periphery of his own visual field as the standard of comparison.

To complete the test the examiner determines the extreme limits of his own field by carrying his moving finger-tips from beyond these limits inwards, in the various meridians, and always in a vertical plane, which bisects the common line of fixation. In each of the principal meridians the presence, or absence, of coincidence in the extent of peripheral vision is ascertained by asking the patient when he first sees the finger-tips. In this way the presence of a defect in the periphery of the field of vision may be rapidly ascertained, but the extent and nature of such defect can only be determined with accuracy by means of the perimeter.

II. The perimeter is an instrument with which the field of vision may be examined throughout, that is to say, both as to its periphery, its centre, and the whole intervening area. The instrument consists essentially of a graduated arc along which the test-object is carried. The arc is capable of rotation through all the meridians.

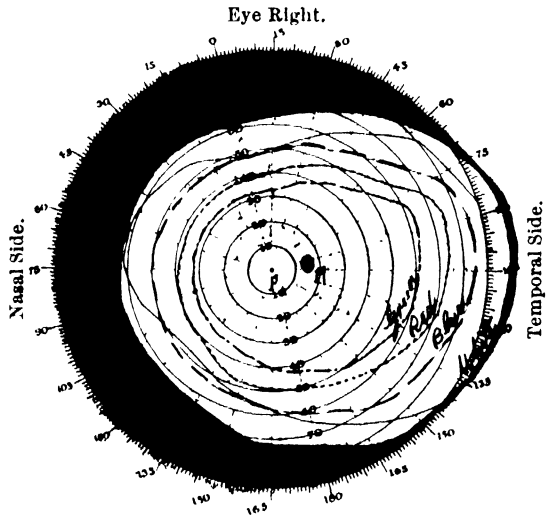


CHART 1.—Test object 20 sq. mm. After Baas. *F*=Fixation point; *M*=Mariotte's blind spot.

By the use of an adjustable chin-rest the patient's eye under examination is maintained in position at the centre of the circle of which the graduated arc forms a part. The fixation object is attached to the pivot on which the arc revolves. As each meridian is examined the position of the test-object is marked upon the chart which is attached to the back of the instrument.

Each chart represents the interior of a hemisphere drawn on the flat. The radiating lines show the meridians in which the examination may be carried out, while the concentric circular lines, which are numbered in degrees from the centre outwards, show the extent or limit of the field in any particular direction. An irregularly oval line, which is traced over the chart, shows the normal average boundary line of the field of vision. The preceding complete chart shows the field of vision for white and also for colours in the normal eye.

III. The examination of the visual field, according to Bjerrum's method, with small white

test-objects, is carried out in the following way:—The apparatus consists of a large vertical screen made of black velvet, of two metres' diameter, and marked out with fine black cord in concentric and radiating lines. The concentric lines represent at regular intervals the distance in millimetres from the centre to the margin of the screen. The radiating lines mark the various meridians. The fixation object is usually placed at the centre of the screen, but may be removed to either margin if a more extended area is required. The test-objects, which are white, vary in diameter from 1 to 20 mm., those in most common use being from three to six. A long rod covered with the same material with which the screen is made, and supplied with a small spring-catch at its extremity—the "carrier"—is used for moving the test-object about over the screen.

The patient is placed at one or two metres from the centre of the screen, according to circumstances, and is supplied with a chin-rest.

The result of the examination is first marked out on the screen with black pins, and afterwards transferred to a chart upon which there has been previously recorded the results of the ordinary perimetric examination.

The transference of the field from screen to chart is most conveniently carried out by reference to a prepared table of equivalents. This table gives in degrees and minutes the angular separation from the centre of each concentric arc marked on the screen, or of the fractions of a metre as in the table here given from Meisling's paper in the *Annales d'oculistique*, December 1900.

Distance from Eye.

Metre.	Two Metres.	One Metre.
1/8	3° 34′	7° 7′
1/4	7° 7′	14° 2′
3/8	10° 37′	20° 33′
1/2	14° 2′	26° 34′
5/8	17° 21′	32°
3/4	20° 33′	36° 52′
7/8	23° 38′	41° 11′
1	26° 34′	45°

The carrying out of this test also entails careful attention to the following points:—

1. Careful correction of refraction.
2. An equal and constant illumination of the screen.
3. Accuracy of fixation on the part of the patient.
4. The tendency to rapid retinal exhaustion from continued fixation, which can only be combated by frequently repeated intervals of rest.

In the normal eye the visual acuity, which is highest at the macula lutea, rapidly falls as we pass towards the periphery of the retina. Thus when the field is tested by Bjerrum's method at two metres' distance, a test-object of 18 mm. diameter, which subtends a visual angle of ½° 1′, gives the field of vision as with the ordinary perimeter. When a smaller test-object is used the normal field becomes reduced in extent accordingly. As can be seen from the chart (No. 2) in which the ordinary perimetric field is shaded grey, while the central part, left white, represents the area responding to the smaller test-object. The chart also shows how

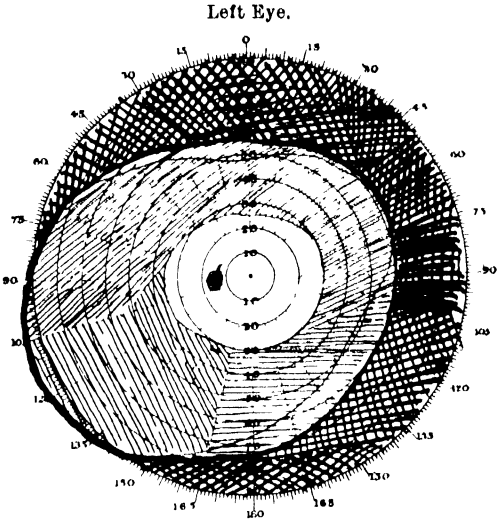


CHART 2.—Normal field for white by Bjerrum's method (Test $\left\{\begin{smallmatrix}\frac{20}{1000}\\ \frac{1}{2000}\end{smallmatrix}\right.$).

the blind spot of Mariotte increases in diameter as the test-object is reduced in size.

The blind spot is surrounded by a zone of retina of relatively reduced visual power, and which fails to respond to the smaller test-objects. The irregularity of outline of the blind spot is

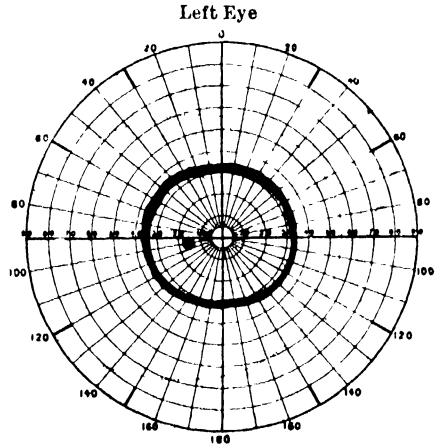


CHART 3.—Peripheral limitation of the field as found in hysteria.

due to the large vessels passing over it (*see* Chart 2).

The clinical importance of morbid changes in the field of vision arises from the fact that certain forms of defect are characteristic of distinct types of disease.

Transitory peripheral limitation of the field of vision is met with in hysteria (*see* Chart 3).

In this condition the results of different examinations may vary widely. If a prolonged examination of the field be carried out, it is

found that increased restriction can be demonstrated as the duration of the examination extends. Further, if the results of examinations in subdued light and in bright light be compared, it is found that the field is more extensive in subdued light. This last point may easily be tested, and is comparatively of constant occurrence.

All the more typical points with regard to the field in this condition depend upon the rapidity with which exhaustion supervenes when sustained functional activity is demanded. Since, however, the hysterical state may coexist with more grave nervous or other disease, further investigation is always necessary. Central vision often remains unaffected, but may also become deteriorated. Lead-poisoning occasionally produces a disturbance of vision similar to that met with in hysteria, though not with the same inconstancy of symptoms. In acute syphilitic choroiditis the field of vision, as

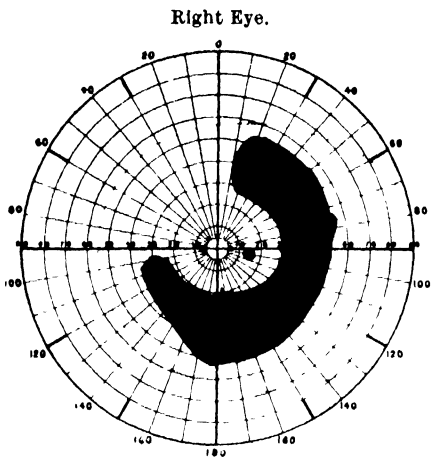


CHART 4.—Partial ring scotoma from a case of retinitis pigmentosa.

well as the visual acuity, are much reduced when examined in subdued light as compared to bright light. This forms a marked contrast to hysteria, and results from the presence of night-blindness in this condition. In the forms of choroiditis, where patches of degeneration occur at various parts of the fundus oculi, corresponding scotomata are found in the field. In some cases secondary optic atrophy plays a prominent part in causing loss of sight. As the ophthalmoscopic signs are sufficient in these cases for diagnosis, it would be of little use to say anything further as to the field of vision in this connection.

Retinitis pigmentosa occurs in both eyes, and is usually associated with night-blindness and concentric limitation of the field. The field may present the peculiarity of a ring-shaped scotomata. Central vision is not, as a rule, full, although often sufficient for reading. The prognosis is unfavourable, inasmuch as no treatment is of any avail, and while the condition may remain stationary for a long time, it tends to become worse (*see* Chart 4).

Any injury to the eye such as might produce commotio retinæ, or hæmorrhage into the retina or vitreous, will cause loss of the field corresponding to the site of lesion. The prognosis is, on the whole, good in these cases, though serious secondary effects may follow. *Detachment of retina* causes loss of vision usually in the form of a sector-shaped peripheral encroachment, often with a history of

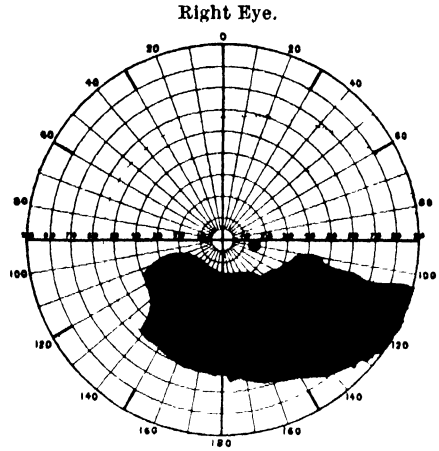


CHART 5.—Detachment of retina.

sudden onset (*see* Chart 5). The prognosis is bad in detachment of retina, as it often extends and seldom recovers.

Central amblyopia is met with in a variety of conditions affecting the retina, choroid, and optic nerve. Central choroiditis can be recognised by means of the ophthalmoscope (*see* Chart 6). Scotomatous amblyopia following exposure of the retina to direct sunlight, and

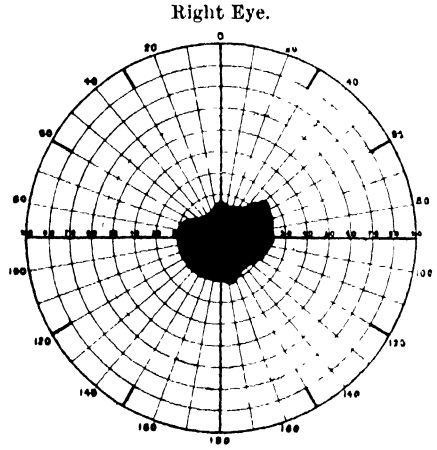
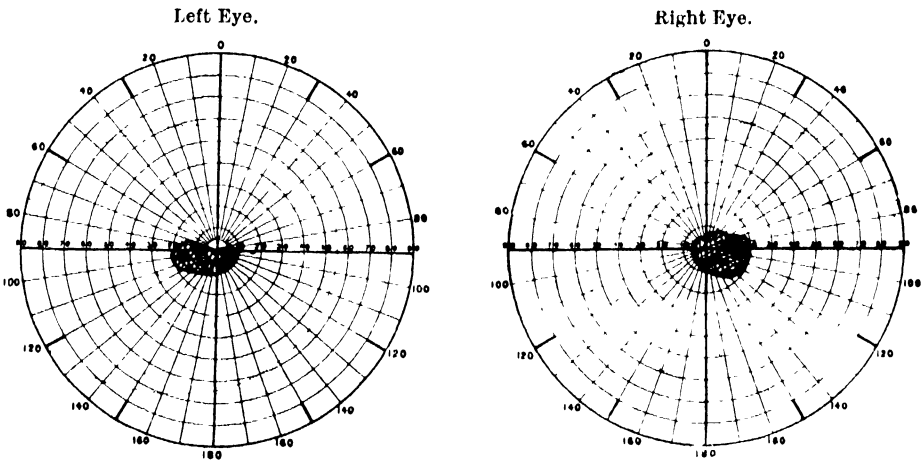


CHART 6.—Central choroiditis.

sometimes caused by electric light, may be recognised from the history of the case and the mode of onset.

Simple optic atrophy, with central scotoma, is often met with; and central scotoma is also, although very infrequently, found in cases of glaucoma. Apart from these conditions just mentioned, the subject of central amblyopia may be generally considered in two divisions or groups of cases—toxic amblyopia and retrobulbar neuritis.

Toxic Amblyopia.—Under this heading are considered these cases of central amblyopia which are of gradual onset, affecting both eyes usually in the same way, and much to the same extent. The absorption of poisonous drugs, usually extending over a prolonged period of time, is the cause in all cases. The well-known tobacco amblyopia is the most common type, but other poisons are known to produce a similar condition. Bisulphate of carbon, alcohol, iodoform, filix mas, quinine, and cocaine may be mentioned as examples. Tobacco amblyopia is most frequent in men over forty years of age. It is common to find visual acuity considerably reduced. Usually it is less than one-third of normal vision. The light-sense is always markedly affected, so that the power of distinguishing degrees of illumination is much reduced. Thus, if Bjerrum's types for testing the "light difference" are used, this defect can be readily demonstrated. The field is of normal extent, and if tested with the usual large-sized white test-object, shows no change throughout. When the test is made with colour (red or green) a colour scotoma is found in each eye, extending from the blind spot, which it includes, to the fixation point, which it often also includes. The scotoma is oval, having its long axis horizontal, and occupies for the most part a paracentral position. It can be demonstrated for white if a sufficiently small test-object is used. It is always negative, that is to say, the patient is not conscious of it as a blind spot in the field (*see* Charts 7). The prognosis is always good, provided the patient gives up the use of tobacco, and recovery may even take place with the continued use of tobacco in reduced amount. Vision usually begins to improve about one month after the use of tobacco has been stopped, and complete recovery follows in three months or more.



CHARTS 7.—These charts are from a case of well-marked tobacco amblyopia. The scotoma is for colour and also for Bjerrum's small white object-test.

Retrobulbar Neuritis. — Under this heading come conditions of the optic nerve, which, while differing in etiology, have certain common clinical manifestations. The clinical characters of this group are almost entirely subjective, and may be stated as follows:—Rapid failure of vision, usually coming on in one eye. Pain and tenderness in the neighbourhood are often present, and especially on pressing the eyeball backwards into the orbit, and on lateral or vertical movements. Amblyopia is always present in the form of a central or paracentral scotoma, but may vary widely and in any degree from a temporary complete loss of sight on the one hand to almost full vision on the other. Ophthalmoscopic changes are absent as a rule during the early stages. A tendency to recovery is also characteristic. A variety of conditions affecting the optic nerve in its course from the chiasma to the eyeball may bring about the above group of symptoms. It is not, however, possible to discuss these conditions here, but only to mention the more important. Firstly, the nerve may suffer from morbid changes arising in the tissues surrounding it, as orbital cellulitis, periostitis, syphilitic disease of the sphenoid, which is apt to affect both nerves, and tubercle of the chiasma. Secondly, the nerve trunk may be the site of lesion, as in gumma of the nerve.

Disseminated sclerosis may attack the optic nerve, either as part of a general condition manifested in other parts of the nervous system, or as a primary condition of the optic nerve. Rheumatic and gouty conditions, with chill as the exciting cause, account for many cases, while menstrual disturbances are also regarded as a cause. Cases have also occurred in connection with peripheral neuritis. The field of vision is affected in a very similar way to that found in tobacco amblyopia, that is to say, there is a negative colour scotoma in much the same position, and the field is not restricted. The following are the distinguishing points in the field:—The scotoma is either in one eye only, or if in both there is more frequently dissimilarity in form and extent. The scotoma is more apt to extend to the inner side of the fixation point, and tends to be more central and less regular in form than that met with in tobacco cases. Though recovery so often takes place, the scotoma may become permanent, and may be associated with loss of peripheral vision. Progressive scotomatous atrophy may follow, or the scotoma may become absolute and remain stationary. The prognosis is on the whole good, though much less favourable than in toxic amblyopia from tobacco (*see* Chart 8).

Optic neuritis when secondary to increased intra-cranial pressure, as in cerebral meningitis, etc., is almost always bilateral, though one nerve may become affected before the other. During the early stage of this form of neuritis vision is often quite unchanged—central vision, the field, and the colour-sense remaining normal. After the lapse of some weeks, when the inflammatory exudation begins to be replaced by

Left Eye.

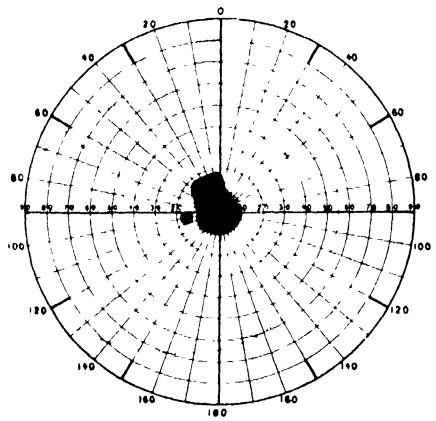


CHART 8.—Absolute scotoma which began with an attack of retrobulbar neuritis and became permanent.

fibrous tissue, there is often progressive loss of vision. The prognosis is on the whole bad, as post-neuritic atrophy is of frequent occurrence, and in a number of instances leads to complete blindness. In some cases, however, good vision may be permanently retained.

HEMIANOPIA. — The field of vision is here limited in a most remarkable way, and varies according to the form of hemianopia present. The most common form is that known as homonymous hemianopia, which may be right sided or left, complete or partial, relative or absolute. The loss of vision to one side of the fixation point, when both eyes are open, often impresses the patient with the idea that he has lost the sight of one eye. The true state of affairs is at once demonstrated by rapidly taking the field of each eye separately with the fingers. When this is done it is found that in each eye the same side, say the right side, of the field has been obliterated. On more careful examination with the perimeter the complete or partial nature of the case becomes evident. In cases of complete homonymous hemianopia the line of demarcation which separates the blind from the seeing part of the field is very sharply defined. It occupies a generally vertical position, and passes through the whole field at or very near the vertical meridian. The dividing line, as a rule, deviates towards the blind side close to the fixation point, and thus usually leaves central vision intact.

In incomplete or partial cases the areas of blindness are less extensive than in the complete form. The blind areas usually correspond in position and extent in the field of each eye. Thus homonymous quadrants may be lost.

Homonymous hemianopia is "absolute" in the great majority of cases, that is to say, colour, form, and light senses are all gone. In some cases the condition is of the "relative" type, when the colour sense may alone be lost, while the light sense remains.

The term hemiachromatopsia is applied to cases with marked colour defect. It has been demonstrated by Bjerrum's method that the form sense is also often affected in hemiachromatopsia. It is considered probable that these relative cases are the result of less severe lesions, which only partially interrupt conduction.

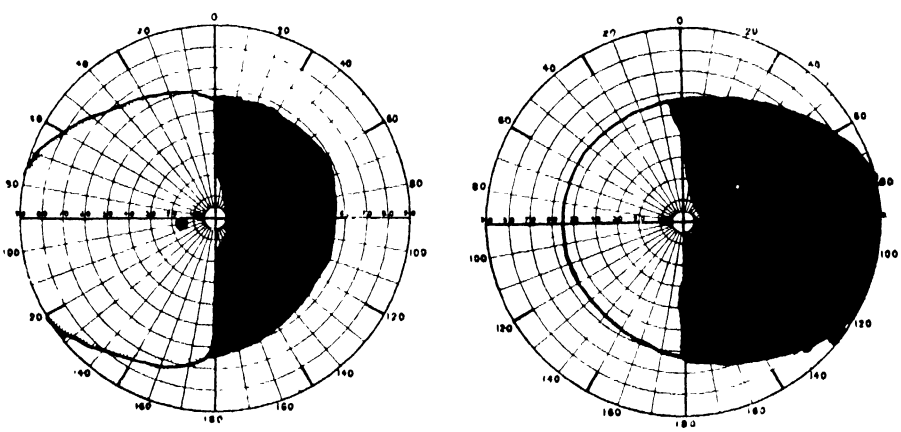
Homonymous hemianopia is often met in association with hemiplegia, but more frequently occurs apart from that disease. Transient forms may exist in connection with hysteria or migraine, and have been noticed prior to the incidence of cerebral hæmorrhage.

The lesion, of whatever nature, is one which interrupts conduction in the course of one optic tract, and may do so at any point from behind the chiasma to the cortical centre.

The following points may assist in the localisation of the lesion :—

"Wernicke's pupil symptom," or the "hemiopic pupil."—To examine for this symptom, the

Left Eye. Right Eye.



CHARTS 9.—Homonymous hemianopia.

patient should be placed in a darkened room with an ophthalmoscopic lamp behind his head. The eye is then slightly illuminated from the front by throwing reflected light from a concave ophthalmoscopic mirror held well out of focus, while the patient is asked to look across the room in order to avoid accommodation. Finally, with a second mirror held to one side a sharply focussed beam of light is thrown on to the amaurotic half of the retina. If, when this

has been done, the pupil fails to react to light thrown upon the blind half of the retina, or does so very little, the symptom is said to be present.

The presence of this symptom is regarded by some excellent authorities as evidence that the lesion involves the optic tract in front of the corpora quadrigemina, thus preventing the effect of light falling upon the amaurotic part

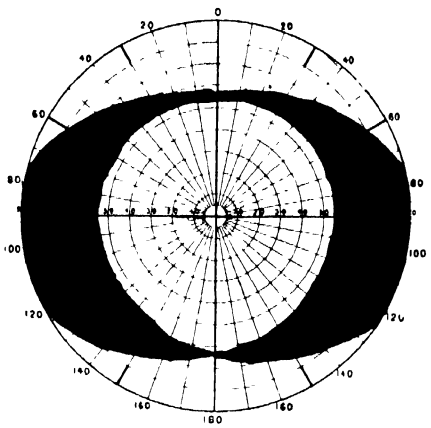


CHART 10.—Showing the binocular field in bitemporal hemianopia.

of the retina from reaching the third nerve nucleus by way of Meynert's fibres.

If the symptom is not present, no conclusions can with safety be based on its absence, because a possible reaction through the normal half of the retina cannot be excluded. It is proper to state that some authorities do not believe that any value can be attached to this symptom.

The presence of word blindness, mind blindness, etc., is suggestive of a cortical lesion, while lesions of the cortex and optic radiations are more likely to produce the incomplete forms of homonymous hemianopia (*see* Charts 9).

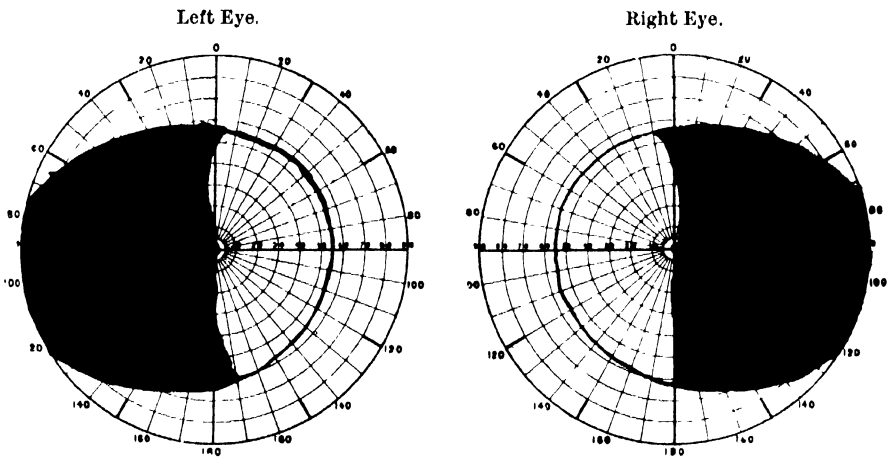
The ophthalmoscopic examination usually shows no change in the disc during the earlier stages, but optic neuritis may be present. In the later stages partial atrophy of the disc is found.

Prognosis is relatively good, as the condition of vision usually remains stationary.

Bitemporal Hemianopia.—The true nature of the visual defect is not so obvious in this as in the homonymous form. When the patient comes under observation with both eyes open, the nasal field of each eye extending about sixty degrees to the opposite side gives a binocular field of such lateral extent as might readily escape notice (*see* Chart 10).

This is more especially the case as the patient is more troubled by his impaired central vision than by the restriction of his field. When the field of vision is examined in each eye separately, it is found to be defective throughout its temporal half in each eye. The dividing line between the normal and the blind part of the field is not so sharply defined, and is less regularly vertical than it is in the homonymous cases. Central vision is usually impaired or lost. Owing to the nature of the lesion, the fields are apt at some period in the course of the case to show inequality. Thus the sight of one eye may be lost by the encroachment of the blind area, while the original half-blindness remains in the other. The condition may be relative or absolute, sometimes beginning as relative and later becoming absolute. Cases not infrequently show a tendency to temporary improvement of symptoms. The lesion is almost invariably—to begin with at any rate—one which affects the crossed fibres occupying the central part of the chiasma, and supplying the nasal halves of the retinæ.

Temporal hemianopia is a common early symptom of acromegaly, here resulting from enlargement of the pituitary body. Aneurysm, periostitis, hyperostoses, basal meningitis, and syphilitic gumma are recognised as causes. Symmetrical cortical lesion is very unlikely. The prognosis is bad as compared with the homonymous form, central vision being always impaired, and many cases passing on to complete blindness from atrophy. Occasionally, however, permanent recovery does take place (*see* Charts 11).



CHARTS 11.—Bitemporal hemianopia.

Binasal hemianopia is exceedingly rare. It results from a chiasmal lesion affecting the lateral parts of the chiasma, where the uncrossed fibres pass which supply the temporal halves of the retinæ.

Altitudinal hemianopia also occurs, and is supposed to be most frequently caused by chiasmal lesion, and sometimes by optic neuritis. Cortical lesions may be the cause.

Optic atrophy, apart from those cases of consecutive atrophy already referred to, is met

with as a primary condition, and frequently occurs in connection with disease of the central nervous system. In tabes dorsalis atrophy may occur early or late in the course of the disease, and often leads to blindness.

In disseminated sclerosis central scotoma is more common, while the deterioration of vision is less uniformly serious than in tabes. Lateral sclerosis and general paralysis of the insane are also associated with atrophy.

The field of vision for white frequently becomes reduced in a concentric manner, though a more rapid encroachment towards one side more often takes place. The field for white takes no typical form, either with the ordinary perimetric test or with Bjerrum's test. When tested with red or green it is found that the field for colour is reduced out of proportion to that for white.

The condition of colour vision varies considerably in different cases of optic atrophy. When

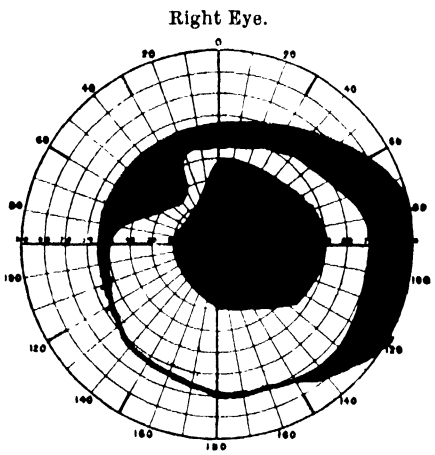


CHART 12.—Scotomatous atrophy with peripheral restriction of the field.

the field for white shows peripheral restriction, and the area over which colours are recognised is much reduced, the prognosis is unfavourable.

When central scotoma is present vision is often very much reduced, or central vision lost, yet if the peripheral extent of the field is full, and the colour restriction slight, the prognosis is relatively good, as such a condition may remain *in statu quo* for years. When optic atrophy is not secondary to disease of the choroid or retina, but has begun primarily in the nerve, examination of the light-sense is of value in distinguishing it from chronic simple glaucoma. In optic atrophy the light difference is reduced, while the light minimum is little affected. The reverse condition obtains in glaucoma.

FIELD IN GLAUCOMA.—The form of the field of vision for white, as recorded with the ordinary perimeter, may vary widely, and although there are certain forms of restriction which are more common, it is often necessary to carry the investigation farther by making the test with colours.

In glaucoma it has been shown that while the restriction of the field for colour is greater than that for white, it is on the whole approximate to the normal relationship.

On the other hand, in optic atrophy the colour restriction is relatively greater than for white, often markedly so. A good idea of the types of the field for white, and their relative frequency of occurrence, may be derived from the investigations of Bange (Berry), who gives the result of examining over 100 cases.

1. Defect in the nasal side alone—27 cases.
2. Defect most marked on nasal side—44 cases.
3. Destruction of field, including the centre, and having only a small temporal portion—9 cases.
4. Remaining field in the form of a peripapillary oval—4 cases.
5. A central or paracentral scotoma, with or

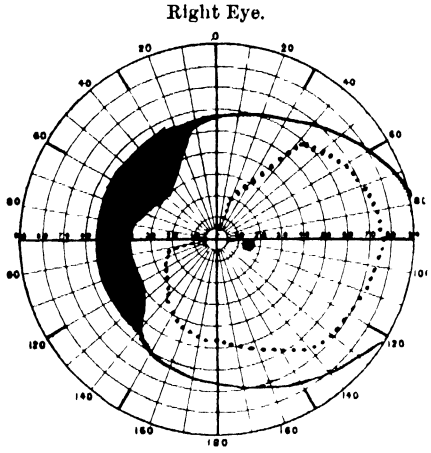


CHART 13.—Giving the field for white and for red as taken in a case of glaucoma.

without slight restriction of nasal margin—4 cases.

6. Concentric restriction—6 cases.
7. Preponderance of defect on temporal side—4 cases.
8. Restriction only upwards—2 cases.

From the above it will be seen that although the form of the field may vary widely, yet in a large proportion of the cases the nasal field is affected most (71 per cent of above).

Difficulties of diagnosis occur chiefly in connection with the simple chronic cases of glaucoma, and as it is in this class of case that the loss of the nasal field is of most frequent occurrence, a preponderance of loss on the nasal periphery of the field may be regarded as the common type.

The chart, taken from an actual case, may prove useful (*see* Chart 13).

When in glaucoma the field is examined by Bjerrum's method (which has been already outlined), certain interesting and important features appear. The final importance of the result to be gained by this method perhaps demands further corroborative proof, yet its

importance is sufficient to justify some notes on it here.

In the December number of the *Annales d'oculistique* for 1900, Mr. Aage A. Meisling of Copenhagen writes on this subject. His paper embodies the results of Bjerrum's former work, together with the additional results of a more recent and extensive investigation of his own on this subject.

The following are the characters of the visual field in glaucoma, as quoted from the above-mentioned paper:—

(1) The visual field as taken with the ordinary perimeter does not correspond with that taken by the "small white object" (Bjerrum's) test.

Defects found in the field of vision by examination with the ordinary perimeter are termed "absolute," while those demonstrated by the "small white object" test are termed relative. The relationship of the area over which vision is most fully preserved to that of relative defect is the point of chief characteristic importance.

(2) The area of most acute vision, as also the area of relative defect, are each in contact with the blind spot, or are said to meet at the blind spot.

(3) Scotomata have frequently a paracentral position. They form a characteristic arc round the macular region, either above or below, or both, the concavity being directed towards the fixation point. Scotomata never reach beyond the blind spot toward the temporal side, but may extend to the periphery on the nasal side.

(4) There are some typical fields which are of semilunar form on the temporal side of the blind spot, the blind spot being situated in the concavity of the crescent.

The form of the visual field is stated to be generally characteristic in glaucoma as opposed to optic atrophy, but may be simulated by fields of vision in retrobulbar neuritis, hæmorrhagic papillo-retinitis, and embolism of a branch of the central artery of the retina.

Visual. — Relating to sight; *e.g.* visual acuity, visual centres, visual field, and visual purple. *See* Eye, Clinical Examination of; Aphasia (*Visual Aphasia or Ataxia*); Brain, Physiology (*Visual Centres*); etc.

Visus.—Light; as in such terms as *visus brevior* (myopia), *visus nocturnus* (nyctalopia), *visus senilis* (presbyopia), etc.

Vital Statistics.

Introduction 487
Population 487
Notification 488
Marriages, Births, Deaths . . . 489
Infantile Mortality 492
Occupation and Mortality . . . 493
Longevity 494
Tests of Health of a Community . 495
Fallacies 496

See also Notification.

Vital statistics is the science of numbers applied to the life-history of communities. Its significance is similar to that of the more recently coined word Demography—though the latter does not necessarily confine itself strictly to the study of life by statistical means. Another term has been frequently used in recent years—"Vital and Mortal Statistics." The continued use of the word "mortal" in this connection is undesirable and objectionable. The term "vital statistics" is comprehensive and complete, as death is but the last act of life.

Of the problems of life with which the science of vital statistics is concerned, population, births, marriages, sickness, and deaths possess the chief importance; and in the following sketch of the subject I shall concern myself chiefly with these. The subject naturally divides itself into two sections: the sources of information, and the information derived from these sources, and both these will require consideration.

The importance of numerical standards of comparison in science increases with every increase of knowledge. The value of *experience*, founded on an accumulation of individual facts, varies greatly according to the character of the observer. As Dr. Guy has put it, "The *sometimes* of the cautious is the *often* of the sanguine, the *always* of the empiric, and the *never* of the sceptic; while the numbers 1, 10, 100, and 10,000 have but one meaning for all mankind." Hence the importance of an exact numerical statement of facts. The sneering statement that statistics can be made to prove anything can only be made by one ignorant of science. In fact, nothing can be proved without their aid, though they may be so ignorantly or unscrupulously manipulated as to appear to prove what is untrue. Instances of fallacious use of figures will be given as we proceed.

An accurate statement of *population* forms the natural basis of all vital statistics. Thus the comparison of the number of deaths in one with the number of deaths in a second community has no significance unless we know also the number living out of which these deaths occurred. Even then our knowledge would be defective without further particulars as to the proportion in each population living at different ages, and the number dying at the corresponding ages. For other purposes we should require to know the number of married and unmarried, the number engaged in different industries, and so on, in order that the influence of marital condition, of occupation, etc., on the prospects of life may be calculated. The first desideratum of accurate vital statistics is a *census enumeration* of the population at such intervals as will not cause the intervening estimates of population to be very wide of the mark. In this country a decennial census is taken, the last occurring in 1901. In the intervals the popula-

tion of the entire country, and of each town or district, is estimated. Various methods of *estimating the population* have been adopted:—

(1) If a strict record of emigration and immigration be kept, then, in a country in which a complete registration of births and deaths is enforced, the population can be easily ascertained by balancing the natural increase by excess of births over deaths, and the increase or decrease due to migration. This is done in New Zealand, but is impracticable in England, as no complete account of migration can be kept.

(2) The increase of inhabited houses in a district being known year by year, the increase of population may be estimated on the assumption that the number of persons per house is the same as at the last census. This may not be strictly accurate. In 1901 it was found that in England and Wales the average number of persons per house was less than in 1891.

(3) It may be assumed that the annual increase during the present decennium will be one-tenth of the increase during the last decennium, 1891-1901. If so, the population, *e.g.* 1905, is the enumerated population in 1901 *plus* $4\frac{1}{4}$ times the annual increase occurring during 1881-1891. (The *fourth* is required because the census is taken early in April, and the population is estimated to the middle of the year.) This method is fallacious, because it makes no allowance for the steadily increasing numbers who year by year attain marriageable age and become parents. It assumes, in other words, simple interest where compound interest is in operation.

(4) The Registrar-General's method, the one generally adopted, assumes that the same *rate of increase* will hold good as in the preceding intercensal period, *i.e.* that the population increases in geometrical progression, and not in arithmetical progression as under (3). The application of this method will be best understood by an example:—

If the census population of a town is 32,000 in 1891, and 36,000 in 1901, what is the mean population in 1905?

(*a*) *Find the rate of increase in* 1891-1901. If P = population at census 1891 and P^1 = population at census 1901,

and if R = rate of increase of population, then

$$P^1 = P + R^n \text{ in the } n\text{th year.}$$

$$\log. P^1 = \log. P + 10 \log. R.$$

$$\tfrac{1}{10}(\log. P^1 - \log. P) = \log. R.$$

$$\frac{4{\cdot}556303 - 4{\cdot}505150}{10} = {\cdot}0051153 = \log. R.$$

(*b*) *Apply this to the increase in the next* $4\frac{1}{4}$ *years.*

$$\text{Here } P_{1905} = P_{1901} R^{\frac{17}{4}}$$

$$\log. P_{1905} = 4{\cdot}556303 + \frac{17}{4}({\cdot}0051153)$$

$$= 4{\cdot}578043.$$

By consulting the table of logs. the population corresponding to this number will be found to be 37,848 = population at the middle of 1905.

Estimates made by the last-named "official" method are liable to error even when made for the entire country, and still more when applied to special districts. Thus the decennial rate of increase of the population of England and Wales in 100 years has varied from 15·8 per cent in 1821-31 to 12·4 in 1891-1901. The anomalies are even greater when the official method is applied to great towns. In one decennium such a town may, owing to brisk trade, have a rapid increase of working population with many children, and in the next decennium, in consequence of emigration or transmigration, there may be little or no increase. The declining birth-rate, which is having a greater effect on the number of population than the declining death-rate, is another cause of disturbance which increases the difficulty in forming a correct estimate of population in intercensal periods. A quinquennial census is highly desirable in order to avoid the doubts necessarily associated with estimates of population in the later years of a decennium, and the birth- and death-rates which are based on these estimates.

The Registration of Births and Deaths.—Civil registration of births and deaths began in 1837, but was not compulsory until 1870. It would be going beyond the scope of this article to give details of the enactments as to registration. It suffices to state that it is the duty of the practitioner to give a certificate stating the cause of death of his patient to the best of his knowledge and belief. There is no registration of still-births in this country. Many deaths are registered of which the cause of death is not medically certified, and the value of our national vital statistics is considerably diminished on this account. Much improvement is desirable in the medical certification of causes of death. Every medical student ought to receive instruction on this subject before the completion of his studies. Names of symptoms, as dropsy, hæmorrhage, convulsions, and obscure names, as abdominal disease, should be avoided. If the patient has recently suffered from injury, or recently passed through child-birth, or had a specific febrile disease, this must not be omitted from the certificate.

The Registration and Notification of Sickness forms another valuable source of information. Various attempts have been made to secure a general registration of disabling sickness, but with only partial success. District and workhouse medical officers appointed since February 1879 are required to furnish the medical officer of health with returns of pauper sickness and deaths. This source of information might, with advantage, be more fully utilised by medical officers of health. Section 29 of the Factory and Workshops Act 1895 requires that every medical practitioner attending on or called in to visit a patient whom he believes to be

suffering from lead, phosphorus, or arsenical poisoning, or anthrax, contracted in any factory or workshop, shall send to the chief inspector of factories at the Home Office, London, a notice stating the name and full postal address of the patient, and the disease from which he is suffering—a fee of 2s. 6d. being payable for each notification, and a fine not exceeding 40s. being incurred for failure to notify.

The Compulsory Notification of Infectious Diseases is enforced by the Act of 1889, which now applies to the whole country.[1] The list of diseases to be notified is as follows:—

Smallpox, cholera, diphtheria, membranous croup, erysipelas, the disease known as scarlatina or scarlet fever, and the fevers known by any of the following names: typhus, typhoid, enteric, relapsing, continued, or puerperal, and also any infectious disease to which the Act has been applied by the Local Authority in manner provided by the Act.

It is the duty of the medical practitioner to ascertain whether in his own district such diseases as whooping-cough and measles have been added to the schedule of notifiable diseases. It is the duty of (*a*) the head of the family to which the patient belongs; in his default, of (*b*) the nearest relatives in the house; in their default, (*c*) of every person in attendance upon the patient; and in default of any such person, (*d*) of the occupiers of the building, as soon as they become aware that the patient is suffering from an infectious disease to which this Act applies, to send notice thereof to the medical officer of health of the district. (*e*) The mere formal duty of sending to the medical officer of health a certificate stating the name of the patient, the situation of the building, and the infectious disease from which in his opinion the patient is suffering, is imposed on every medical practitioner attending on, or called in to visit, the patient, on becoming aware that the patient is suffering from an infectious disease to which this Act applies. He is entitled to a fee of 2s. 6d. if the case occurs in his private practice, and of 1s. if the case occurs in his practice as medical officer of any public body or institution. He is subject to a fine not exceeding 40s. if convicted of failure to notify.

The value of returns of infectious diseases as enabling preventive measures to be taken, is increased by interchange of notification returns of different districts. This is now undertaken weekly for a large number of districts by the Local Government Board, and the Registrar-General publishes quarterly summaries of such returns, as well as weekly returns of infectious diseases for the metropolis.

Marriages are usually stated in proportion to the total population, or the number per 1000 of population; but a more accurate method would be to base the marriage-rate, for comparative purposes, on the number of unmarried persons living at marriageable ages. In England the marriage-rate is always higher in large towns than in rural districts. Thus in 1900 the marriage-rate in London was 17·6 as compared with an average marriage-rate in 1891-95 of 15·2 per 1000 of the estimated population in England and Wales. The higher marriage-rate in towns is chiefly owing to the fact that higher wages and greater scope for remunerative work attract young country people of marriageable ages to towns.

Births are usually reckoned as a rate per 1000 of population. Clearly, however, if one population had a larger proportion than another of women of child-bearing years this method of comparsion would not be free from possible error. Even were the proportion of women of child-bearing ages equal, the comparison might be fallacious if in one population the proportion of single women was much higher than in the other. The possibility of illegitimate births does not materially vitiate this conclusion, as such births do not constitute more than 4 per cent of the total births, and their number is not excessive in the districts in which there is the greatest excess of single women, viz. in districts in which a large number of domestic servants are employed. The only strictly accurate method is to subdivide the births into legitimate and illegitimate, stating the former per 1000 married women of child-bearing years, and the latter per 1000 unmarried women of child-bearing years. I append an example of the relative accuracy of the three methods above indicated:—

Birth-Rate.			
	Per 1000 Inhabitants.	Per 1000 Women aged 15-45 Years.	Per 1000 Married Women aged 15-45 Years.
Kensington . . .	21·8	61·6	215·4
Whitechapel . . .	39·9	172·1	328·3
Percentage excess of birth-rate in Whitechapel over that in Kensington	83%	179%	53%
	A	B	C

Thus, according to the ordinary method (A) of stating the legitimate birth-rate, it is 83 per cent higher in Whitechapel than in Kensington, whereas it is really only 53 per cent higher. Similarly, a statement of the illegitimate birth-rate in the two districts "per 1000 inhabitants" shows an excess of only 6 per cent in Whitechapel, while a statement "per 1000 unmarried women aged 15 to 45

[1] Except London, where similar powers are given by the Public Health (London) Act.

years," shows the real excess of 144 per cent. Both in this and other civilised countries there has been in the last twenty-five years a steady decline in the birth-rate. In England the maximum birth-rate was 36·3 per 1000 of population in 1876, and the minimum 29·3 in 1899. This diminution is only caused to a minor degree by postponement of marriage to more mature years and by a larger proportion of celibacy. Nor is there any reasonable ground for the view that a diminished power of either sex to produce children has been produced by alcohol, syphilis, tobacco, or other causes. The main cause of the diminution of the birth-rate is "the deliberate and voluntary avoidance of child-bearing on the part of a steadily increasing number of married persons."

Deaths are calculated in proportion to every 1000 of the population, the unit of time being a year. This unit is preserved even when death-rates for shorter periods, *e.g.* a week, are stated. Thus the death-rates for the thirty-three great towns published weekly in the chief newspapers are *annual death-rates*; they represent the number who would die per 1000 of the population, supposing the same proportion of deaths to population held good throughout the year. The best plan to obtain the weekly annual death-rate is as follows: the correct number of weeks in a year being 52·17747, if the population of a town be 143,956, and the number of deaths in a given week are 35, then the death-rate is 12·687. Thus

$$\frac{143{,}956}{52{\cdot}17747} = 2758 \qquad \frac{1000}{2758} = 0{\cdot}3625.$$

This is the factor by which the weekly number of deaths must be multiplied

$$35 \times 0{\cdot}3625 = 12{\cdot}6875 \text{ or } 12{\cdot}7.$$

The above is the *crude death-rate.* Various corrections are required, which must now be considered. The most important of these are for public institutions, for visitors, and for age and sex. A public institution, *e.g.* a workhouse, infirmary, or asylum in a given district may consist almost entirely of persons belonging to another district. The rule is to relegate to the district to which they belong all deaths of inmates of an institution, *i.e.* subtract all deaths of outsiders occurring in inside institutions, and add all deaths of inhabitants occurring in outside institutions. The following copy of the official table recently authorised by the Local Government Board for the annual reports of medical officers of health shows the columns giving the necessary corrections.

Note.—The deaths to be included in Column 7 of this table are the whole of those registered during the year as having actually occurred within the district or division. The deaths to be included in Column 12 are the number in Column 7, corrected by the subtraction of the number in Column 10, and the addition of the number in Column 11.

By the term "Non-resident" is meant persons brought into the district on account of sickness or infirmity, and dying in public institutions there; and by the term "Residents" is meant persons who have been taken out of the district on account of sickness or infirmity, and have died in public institutions elsewhere.

The "public institutions" to be taken into account for the purposes of these tables are those into which persons are habitually received on account of sickness or infirmity, such as hospitals, workhouses, and lunatic asylums. A list of the Institutions in respect of the deaths

Table I.—*Vital Statistics of Whole District during* 1902 *and Previous Years.*

Name of District

Year.	Population estimated to Middle of each Year.	Births.		Total Deaths registered in the District.				Total Deaths in Public Institutions in the District.	Deaths of Non-residents registered in Public Institutions in the District.	Deaths of Residents registered in Public Institutions beyond the District.	Net Deaths at all Ages belonging to the District.	
				Under 1 Year of Age.		At all Ages.						
		Number.	Rate.[1]	Number.	Rate per 1000 Births registered.	Number.	Rate.[1]				Number.	Rate.[1]
1	2	3	4	5	6	7	8	9	10	11	12	13
1892 *												

[1] Rates in Columns 4, 8, and 13 calculated per 1000 of estimated population.

* Followed by similar figures for each of the next ten years, and the averages for the ten years.

in which corrections have been made should be given in the table below :—

Thus at ages over 5 and under 45 for males, and under 55 for females, the death-rate is

Area of District in acres (exclusive of area covered by water).	. ..	Total population at all ages	At Census of 1901.
		Number of inhabited houses	
		Average number of persons per house . ..	

[See below.

I.	II.	III.
Institutions within the District receiving Sick and Infirm Persons from outside the District.	Institutions outside the District receiving Sick and Infirm Persons from the District.	Other Institutions, the Deaths in which have been distributed among the several Localities in the District.
Is the Union Workhouse within the District?		

The population as well as the deaths of these institutions should be excluded, in so far as they are derived from outside the district, in order to make the net death-rate approximately correct.

Theoretically, the correction ought to be extended so to apply to visitors who do not die in public institutions. In practice, however, this cannot be effected until a central "clearing-house" is established. The exclusion of deaths of visitors from the district in which they occur is easy; their inclusion in the returns of the district from which they came is more difficult to secure. For the present, they should be included in the death-rate of the district in which they occur.

Death-Rate according to Age and Sex.—To obtain a true conception of the death-rate in a community it is necessary to state the number of deaths in each sex in proportion to the number living at different ages. The importance of this is shown by the following extract from the Registrar-General's report for 1899 :—

lower than is the total death-rate for all ages For females at all ages, except from 5 to 15, the death-rate is lower than for males. From the above statement it will be clear that a considerable excess of women (as in a residential district with a large number of domestic servants), or a considerable excess of either sex at the ages of 15 to 45 (as in most large towns), in proportion to the number living at other ages, would produce a lower total or crude death-rate, which does not necessarily imply a more healthy condition than that of another district which is less favourably constituted so far as the proportion of the sexes and the numbers living at different ages are concerned. By a means of correction now to be described, this source of error can be eliminated.

The method of obtaining the *factor for correction* can be best understood by an example. The annual death-rate of England and Wales in 1881–90 was 19·15, and the death-rate at

Table I.

England and Wales.—*Deaths to* 1000 *living at each of Twelve Groups of Ages.*

	All Ages.	Aged 0–	5–	10–	15–	20–	25–	35–	45–	55–	65–	75–	85 and upwards.
Males . .	19·5	60·4	3·8	2·2	3·6	5·3	7·1	12·3	20·0	37·2	69·8	152·6	300·3
Females . .	17·3	50·7	3·9	2·3	3·3	4·3	6·1	10·0	15·4	29·8	61·5	142·6	272·0

each age-group is given in the following table:—

Ages.	Mean Annual Death-Rate in England and Wales, 1881–90, per 1000 living at each Group of Ages.		Population of Huddersfield in 1891.		Calculated Number of Deaths in Huddersfield.	
	Males.	Females.	Males.	Females.	Males.	Females.
Under 5	61·59	51·95	4,551	4,785	280	249
5	5·35	5·27	4,691	5,081	25	27
10	2·96	3·11	5,113	5,165	15	16
15	4·33	4·42	4,905	5,549	21	25
20	5·73	5·54	4,541	5,461	26	30
25	7·78	7·41	7,466	8,834	58	65
35	12·41	10·61	5,576	6,265	69	66
45	19·36	15·09	3,944	4,649	76	70
55	34·69	28·45	2,393	3,017	83	86
65	70·39	60·36	1,128	1,590	79	96
75 and upwards	162·62	147·98	250	466	41	69
Totals			44,558	50,862	773	799
			95,420		1572	

The population of Huddersfield at each of the corresponding age-periods, as given by the census of 1891, is also shown in this table, and in the last column the number of male and female deaths that would occur by applying the death-rates for England and Wales to the population of Huddersfield are shown. The total number of deaths thus calculated is 1572 in a population of 95,420, and the total death-rate = 16·47 per 1000. This is the *standard death-rate*, *i.e.* the death-rate at all ages, calculated on the hypothesis that the rates at each of twelve age-periods in Huddersfield were the same as in England and Wales during the ten years of the last intercensal period for which the information is available, viz. 19·15 in 1881-90. But the standard death-rate of Huddersfield would have been 19·15, instead of 16·47, were it not for the fact that the distribution of age and sex in the Huddersfield population is more favourable than in the country as a whole. Hence it must be increased in the ratio of 19·15 : 16·47, *i.e.* multiplied by the factor $\frac{19{\cdot}15}{16{\cdot}47} = 1{\cdot}1627$. When the crude or recorded death-rate for 1900 of 16·78 is multiplied by this factor we obtain the *corrected death-rate* of $16{\cdot}78 \times 1{\cdot}1627 = 19{\cdot}51$ per 1000, which is the correct figure to compare with the death-rate of 18·31 for England and Wales in that year. If the death-rate of England and Wales be stated as 1000, then $\frac{1000 \times 19{\cdot}51}{18{\cdot}31} = 1066$, is the *comparative mortality figure* for Huddersfield. Similarly, in the year 1900 the comparative mortality figure of London was 1093, of Croydon 831, of Norwich 919, while that of Liverpool was 1539, of Salford 1541. In all the towns, except Plymouth and Norwich, the corrected death-rate is higher than the crude or recorded death-rate. This implies that in all except these two towns the factor of correction is greater than unity.

This is a convenient point for briefly discussing the RELATIONSHIP BETWEEN THE BIRTH-RATE AND DEATH-RATE. The opinion is commonly held that a high birth-rate is a direct cause of a high death-rate, owing to the great mortality among infants. Table I. shows that the death-rate at ages under five is three times as high as at all ages together, and it is therefore natural to suppose that a high birth-rate, by producing an excessive proportion of persons of tender years, will cause a high general death-rate. This might be so if the birth-rate was to remain high for only five years. But if the high birth-rate continued longer the proportion of the total population at ages of low mortality would be increased, and the general death-rate be lowered. We have already seen that in nearly all the great towns, in which the birth-rate is higher than in rural districts, the age distribution of the population is more favourable to a low death-rate than in rural districts, and their relatively high crude death-rate is made still higher than that of rural districts when the necessary factor of correction is applied.

The INFANTILE MORTALITY should be stated in terms of the infantile population. This is more accurately assumed to be equal to the number of births in the given year than estimated from the number stated to be under one year of age at the last census. The number of deaths

under one year of age per 1000 births was 163 for England and Wales in 1899, being lowest in the agricultural counties and highest in mining and manufacturing counties. In the thirty-three great towns it averaged 172 in the year 1900, ranging from 132 in Croydon, Huddersfield, and Halifax, to 236 per 1000 births in Preston. Of 1000 male children born in England and Wales in 1881-90 the number surviving at the age of three months was 921, at the age of six months 889, twelve months 839, while the number of female children surviving one year of 1000 born was 869. In towns a smaller number survive. In the causation of this high infantile mortality ignorance and inexperience on the part of parents play a considerable part, especially as influencing the food and mode of feeding.

The death-rates at other age-groups beyond infancy are given in Table I.

Season influences the death-rate. The third quarter of the year has the lowest death-rate, unless the amount of epidemic diarrhœa has been excessive. In the first quarter of the year the highest death-rate usually occurs. Mild winters and cool summers both lower the mortality. The seasonal incidence of infectious diseases need only be mentioned in passing.

Density of Population has important bearings on the death-rate. Thus the urban districts in 1899 had a death-rate of 19·2, and the rural of 16·3 per 1000 of population. Farr found that the death-rate increased with the density of populations, not in direct proportion, but in proportion to the 6th roots of the contrasted populations. This rule does not now hold generally good. It is only after the density has reached a certain degree of intensity that it begins to exert an appreciable effect. Even then it is what is implied in aggregation, rather than the aggregation itself, that is pernicious. In particular, poverty is usually greater in densely populated districts than elsewhere, with its accompaniments of deficient food and clothing and bad housing. Hence the excess of phthisis in tenemented houses, especially in houses with three rooms or less. I have shown that the true density that should be considered is the number of persons to each room, not the number of persons on a given area ("The Vital Statistics of the Peabody Buildings," *Roy. Statist. Soc.*, Feb. 1891).

Occupation and Mortality. — To obtain correct statistics showing the influence of occupation on vitality, one must know the number and age of those engaged in each industry and the corresponding number of deaths. A statement of the mean age at death of those engaged in different occupations would be most fallacious (p. 494). The best plan is to restrict the statistics to men aged twenty-five to sixty-five, and calculate for these *death-rates in a standard population*, after the fashion already described (p. 491). By this means a "comparative mortality figure" can be obtained. For all males it is 1000, for farmers 563, teachers 603, lawyers 821, doctors 966, butchers 1096, plumbers 1120, brewers 1427, innkeepers 1659, potters 1706, file-makers 1810. Speaking generally, the occupations are most unhealthy in which there is the greatest exposure to dust, to the breathing of foul air, and to excessive indulgence in alcoholic drinks. (For further details, see the author's *Elements of Vital Statistics*, p. 169 *et seq.*)

Deaths from Various Causes.—These may be stated in proportion to total deaths from all causes, or in terms of the population. The first plan must be adopted only when it is desired to ascertain the proportional share of a given cause of death in the total mortality. In 1899 in England and Wales the diseases named in the first column of the following table were the most prolific causes of death :—

England and Wales, 1899.—*Deaths from various Causes to* 10,000 *Deaths from all Causes.*

Bronchitis	880	Influenza	213
Phthisis	729	Whooping-cough	174
Pneumonia	685	Measles	172
Old age	541	Diphtheria	160
Diarrhœa, Dysentery	511	Enteric fever	108
Cancer	452	Scarlet fever	64
Apoplexy	327	Smallpox	3

The diseases in the second column are given in order to indicate their proportional share of the total number of deaths.

The proper plan of stating the death-rate from a given disease is in terms of the population, or better still, subdivided into death-rates from the diseases for different age-groups, as in Table I., if the number of deaths is not too small to admit of this. The importance of stating the death-rate for different age-groups is greatest for such diseases as diarrhœa, whooping-cough, and measles, in which most of the deaths occur at ages under five. In the following table are given the death-rates from the causes of death which are most important, either from their magnitude, or because of their preventible character :—

England and Wales, 1899.—*Death-rate per* 1000 *Persons living.*

Smallpox	0·005	Old age	0·99
Measles	0·32	Apoplexy	0·60
Scarlet fever	0·12	Convulsions	0·57
Influenza	0·39	Valvular disease of heart	0·38
Whooping cough	0·32	Bronchitis	1·61
Diphtheria	0·29	Pneumonia	1·26
Enteric fever	0·20	Gastro enteritis	0·61
Typhus fever	0·001	Bright's disease	0·29
Cholera	0·04	Accidents	0·59
Diarrhœa, Dysentery	0·94	Ill-defined and not specified causes	0·78
Cancer	0·83		
Phthisis	1·34		
Other tubercular diseases	0·58		
Premature birth	0·58	All Causes	18·33

DETERMINATION OF LONGEVITY. — We have hitherto considered only death-rates, *i.e.* the numbers dying each year out of each 1000 of population. The mean duration of life involves another aspect of the same problem. Although nothing is more uncertain than the duration of individual life, the duration of life for the entire community is subject to so little variation that annuities and life assurance can be made the subject of exact calculations. Of the tests employed to measure the duration of human life the most commonly employed is the MEAN AGE AT DEATH.[1]

Mean age at death $= \frac{\text{sum of ages at death}}{\text{number of deaths}}$. This is a fair method of stating the average longevity of a particular group of persons, if the group is sufficiently large to avoid the possible error caused by paucity of data (p. 496). But it would be entirely unsafe to assume that by this means a safe standard of comparison between two groups can be formed. Thus in 1890 it was stated that the mean age at death of workmen was twenty-nine to thirty years, of the well-to-do classes, fifty-five to sixty years. This statement throws no light on the relative vitality of the two classes under comparison. The well-to-do classes consist largely of those whose working days are past, and it is as untrustworthy to compare their mean age at death with that of workmen as it would be to base any conclusion on the fact that the mean age at death of bishops is much higher than that of curates. The mean age at death is lowest in countries with a high birth-rate. Hence it would be very fallacious to compare the mean age at death in England and France.

The PROBABLE DURATION OF LIFE (*vie probable*) is a term sometimes employed to denote the age at which any number of children born into the world will be reduced to one-half. In practice it can only be ascertained from a life-table (p. 495).

The true mean duration of life, or expectation of life, can only be ascertained from a LIFE-TABLE, and this must therefore be briefly described. This is the true *biometer*, of equal importance in all inquiries connected with human life with the barometer or thermometer and similar instruments employed in physical research. The life-table represents "a generation of individuals passing through time." The data required for its construction are the number and ages of the living, and the number and ages of the dying, *i.e.* the data required for ascertaining the death-rate for each year of life. Theoretically the best plan for forming a life-table would be to observe a million children, all born on the same day, through life, entering in a column (headed l_x) the number who remain alive at the end of each successive year, until all have died, and in a second column (headed d_x) the number dying before the completion of each year of life. This method is impracticable, and were it otherwise the experience would be obsolete before it could be utilised. The method employed in constructing the national life-tables for England is, without tracing the history of individuals through life, to assume that the population being given by the census returns, and the death-rates for each age for a given decennium being known, that the same death-rates will continue during the remainder of the lives of the population included in the census returns.

The total mean number living and the total number dying for a given age-period are known. The mean chance (p_x) of living one year during this age-period is found by the fraction:

$$\frac{\text{Population} - \frac{1}{2}\text{ deaths}}{\text{Population} + \frac{1}{2}\text{ deaths}} = p_x.$$

It is usual to start with a million or 100,000 children at birth, and to make a separate table for the proportionate number of males and females at birth. Thus, in Brighton in 1881-90, these were in the proportion of 51,195 and 48,805. Starting with 51,195 male infants at birth, and multiplying this number by ·84608, the probability of surviving for one year, we obtain $51,195 \times ·84608 = 43,315$. For the second year of life the probability of surviving was ·93398; hence the number of survivors is $43,315 \times ·93398 = 40,452$; and so on.

The general arrangement is shown in the following example of a life-table, which only gives the data at or near the two extremes of life, the intermediate figures having been omitted from considerations of space:—

BRIGHTON LIFE-TABLE.—MALES

(*Based on the Mortality of the* 10 *Years*, 1881-90.)

Age. x.	Dying in each Year of Life. d_x.	Born and surviving at each Age. l_x.	Sum of the Number living, or Years of Life lived at each Age, $x+1$, and upwards to the last Age in the Table. Σl_{x+1}.	Mean After-Life Time (Expectation of Life) at each Age. e^0_x.
0	7880	51,195	2,206,174	43·59
1	2863	43,315	2,162,859	50·43
2	996	40,452	2,122,407	52·96
3	733	39,456	2,082,951	53·29
4	440	38,723	2,044,228	53·29
...	...	...	...	...
97	12	29	43	1·60
98	7	17	26	1·53
99	4	10	16	1·48

The 43,315 males surviving to the end of the first year of life out of 51,195 born, will have

[1] There is no general agreement as to the exact sense in which the words *average* and *mean* should be used. They are used here interchangeably.

each lived a complete year in the first year, or among them 43,315 years. Similarly, the 40,452 males will live among them 40,452 further complete years, and so on, until all the males started with become extinct at the age of 105. Evidently, therefore, the total number of complete years lived by the 51,195 males started with at birth will be 43,315 + 40,452 + 39,456 + 38,723 + . . . + 10 + 6 + 4 + 3 + 2 + 1 = 2,206,174 years; this sum being obtained by adding together the numbers living at each age beyond (*i.e.* below on the table) the age in question right down to its last item. This number of years is lived by 51,195 males. Hence the number of complete years lived by, *i.e.* the expectation of life of each male—

$$= \frac{2,206,174}{51,195} = 43{\cdot}09 \text{ years.}$$

This is the *curtate expectation of life*. It deals only with the complete years of life, not taking into account that portion of lifetime lived by each person in the year of his death, which may be assumed to be, on a average, half a year. Hence the *complete expectation of life*, according to the above table, is 43·59 years.

In the following table the expectation of life (complete) for various towns and for England is given:—

Life-Table.—Expectation of Life at Birth.

	Males.	Females.
English Life-Table, 1838-54 (Farr) .	39·91	41·85
,, ,, 1871-80 (Ogle) .	41·35	44·62
,, ,, 1881-90 (Tatham)	43·66	47·18
London, 1881-90 (Murphy) . .	40·66	44·91
Brighton, ,, (Newsholme). .	43·59	49·25
Manchester, City, 1881-90 (Tatham)	34·71	38·44
Glasgow, 1881-90 (Chambers) . .	35·18	37·70

Formulæ of varying degrees of accuracy have been devised for giving, in the absence of a life-table, an approximation to the expectation of life. Willich's Formula is as follows:—If x = expectation of life, and a = present age, then—

$$x = \frac{2}{3}(80 - a).$$

Thus at the age of fifty years the expectation of life, according to this formula, is twenty years. By the English life-table for 1881-90 it was 18·82 for males, and 20·56 for females. Farr's Formula is based on the birth- and death-rates:—If b = birth-rate, and d = death-rate per unit of population, then—

$$\text{Expectation of life} = \left(\frac{2}{3} \times \frac{1}{d}\right) + \left(\frac{1}{3} \times \frac{1}{b}\right).$$

Thus—

b for England and Wales, 1889-98 $= \frac{30{\cdot}3}{1000} = {\cdot}0303$,

and

d ,, ,, ,, $= \frac{18{\cdot}4}{1000} = {\cdot}0184$.

$$\left(\frac{2}{3} \times \frac{1}{{\cdot}0303}\right) + \left(\frac{1}{3} \times \frac{1}{{\cdot}0184}\right) = 47{\cdot}2 \text{ years,}$$

as compared with the expectation of life for 1881-90, shown in the table on p. 494.

In a life-table the number out of which one dies annually,
The mean age at death,
And the expectation of life,
} are identical in value

when the whole duration of life, from birth to death, is included in the calculation. This is only true for a stationary or life-table population, in which the number dying is assumed to be regularly replaced by a corresponding number of persons of the same age.

Life-Capital.—The life-tables now in use are those based on the experience of 1881-90. The gain in any subsequent year, as in 1900, may be ascertained as follows:—The mean population and the death-rate for each age-group, as 0-5, 5-10, etc., are calculated. Then the mean death-rate of the same community for 1881-90 is applied to this population. By this means the "calculated number" of deaths in 1900 is obtained. The difference between these numbers and the "actual number" obtained from the death-registers gives the gain or loss during the year. Next multiply these differences by the mean expectation of life for the corresponding groups of years. By adding the gains thus ascertained, and subtracting any losses, we obtain the net gain in "life-capital" (Tatham) during the year 1900.

Tests of the Health of a Community.—1. The *general death-rate* is the test most commonly applied and generally trusted. It has its limitations in this respect. It may usually be trusted in comparing a town or district for a single year with preceding years, as the age and sex distribution of a given population only change slowly. But when comparison with other towns or districts is made, the possibility that erroneous conclusions may be drawn becomes considerable. (*a*) Before the death-rates of two districts can be compared, either this comparison must be made by means of death-rates for age-groups (0-5, 5-10 . . . 65-75, etc., etc.), or the factor of correction, the method of obtaining which is described on p. 491, must be applied. (*b*) It must be ensured that in the two compared districts an equal amount of correction has been made for deaths occurring in public institutions and among visitors (p. 490). (*c*) Even when the above precautions are taken it is conceivable that a town with a death-rate of 15 per 1000 may really be as healthy as another with a death-rate of 12 per 1000, though a statistical justification of this statement is a difficult task. Social conditions, quite irrespective of the sanitary condition or the natural salubrity of a district, have an important influence on the death-rate. Poverty, and all that it denotes, necessarily involves a higher death-rate than occurs among the well-to-do. Furthermore, the domestic servants employed by the latter frequently die in districts other than those in which they are employed, without

any possibility of the requisite corrections being made.

2. The *zymotic death-rate* is frequently quoted as a test of sanitary condition. This is a death-rate based on the deaths from the "seven chief zymotic diseases" — smallpox, measles, whooping-cough, diphtheria, scarlet fever, fever (chiefly enteric), and diarrhœa. This death-rate should be entirely discarded, the death-rate from each infectious disease being separately stated. A high death-rate from enteric fever would be a much more serious reflection on the health of a town than a high death-rate from whooping-cough.

The death-rate from each of these diseases in London and in England in 1899 was as follows:—

Death-Rate in 1899 *per* 1000 *Living.*

	England and Wales.	London.
Smallpox	·005	*Nil*
Measles	·32	·47
Scarlet fever	·12	·08
Diphtheria	·29	·43
Typhus	·001	*Nil*
Enteric fever	·20	·18
Whooping-cough	·32	·38
Diarrhœa	·94	·92

A statement of the death-rate from each of these diseases for a series of years is a much more trustworthy test than a similar statement for a single year, in which accidental causes may have caused a temporary increase, or a statement of the average result for a series of years, which tends to conceal the epidemic variations of the disease in question. The danger of such averages has been well exposed by Chadwick in the remark that "a mean between the condition of Dives and Lazarus tends to make it appear that after all Lazarus has not so much to complain of."

3. The *infantile mortality* (p. 492) is a delicate test of mixed sanitary and social conditions, and stress may always be laid on it from these standpoints. The importance of comparing death-rates at other age-groups has already been emphasised.

4. The most delicate and exact method, if all the data are accurate and complete, is to construct a *life-table*, and ascertain the expectation of life in comparison with that of other communities.

The preceding statistical tests of the salubrity of a community, and any others that may be available, should all, when practicable, be utilised; and it should always be remembered that these tests, especially the general death-rate, are most trustworthy when contrasting the experience of a community with its past experience, and least trustworthy when contrasting its experience with that of others, owing to the difficulty in the latter case of ensuring the avoidance of error arising from *non ceteris paribus*.

Statistical Fallacies. — If "fallacies" be regarded as synonymous with "errors," clearly they may occur at every step. They may be classified as errors of data and errors of methods. The most important *errors of data* are erroneous estimates of population and erroneous returns of deaths, especially in the direction of exclusion of certain deaths (p. 490). Death-rates for short periods are relatively untrustworthy. The erroneous use of the mean age at death as a test of longevity has been mentioned (p. 494). These are, in part also, *errors of methods*, and numerous mixed examples are given below.

Errors from paucity of data frequently arise (the "fallacy of small numbers"), a too hasty generalisation being the most common fault in medical writings, especially in therapeutics. The degree of approximation to the truth of a varying number of observations is estimated by means of Poisson's Formula. If—

μ = total number of cases recorded in two groups.
m = number in one group.
n = number in the other group, so that $\mu = m + n$.

The extent of variation in the proportion of each group to the whole will vary within the proportions represented by—

$$\frac{m}{\mu} + 2\sqrt{\frac{2.m.n}{\mu^3}} \text{ and } \frac{n}{\mu} - 2\sqrt{\frac{2\,m.n}{\mu^3}}.$$

The larger the number of the total observations (μ), the less will be the value of $2\sqrt{\frac{2\,m.n}{\mu^3}}$, and the less will be the limits of error in the simple proportion $\frac{m}{\mu}$.

Thus, of 147 cases of enteric fever 17 died, a fatality of 11·4 per cent. The possible error is determined by the second half of the above formula:—

$$= 2\sqrt{\frac{2 \times 17 \times 130}{147^3}} = 2\sqrt{\frac{4420}{3{,}176{,}523}} = \cdot 0746,$$

i.e. the possibility of error = ·0746 to unity, or 7·46 per cent. In other words, in a second series of cases of enteric fever under the same conditions as the above, the fatality may vary from 3·94 to 18·86 per cent, a vague result, which indicates that the first series cannot be regarded as establishing more than a *prima facie* case in favour of any special method of treatment that may have been adopted.

Non Ceteris Paribus.—The necessity that data to be compared shall be collected on a uniform plan, and be of a strictly comparable nature, is very frequently ignored. The conclusion that the administration of a given antiseptic is a valuable means of treating enteric fever is not demonstrated, by the fact that the fatality in the series of cases thus treated is 7 per cent, while in another series treated without antiseptics it is 14 per cent, unless it is shown that the age and other previous conditions of

the patients in the two groups were not widely different, and unless the series are sufficiently long to avoid the fallacy due to paucity of data.

Errors from the Composition of Rates.—If the death-rate of A, having a population of 10,000, is 10 per 1000, and of B, having a population of 20,000, is 15 per 1000, the combined death-rate is not $\frac{10+15}{2} = 12{\cdot}5$. To obtain the correct combined death-rate the number of deaths in A (= 100), and in B (= 300), must first be ascertained, and then the death-rate in a population of 30,000 in which 400 deaths occurred = 13·3 per 1000.

Errors from stating Deaths in Proportion to Total Deaths.—There is nothing erroneous *per se* in stating the proportion of deaths at one age as a ratio of the total deaths at all ages, or the deaths from one cause as a ratio of the total deaths from all causes. It is a useful, and, in fact, the only method practicable, when it is required to give the proportion of one of these to the other. But beyond this such ratios cannot be trusted. For instance, the proportion of fatal accidents among male infants is 12·2, and among female infants 25·1 per cent of the total fatal accidents in the male and female sex respectively. But it would be erroneous if it were concluded from these figures that females are more subject to accidents than male infants. The only conclusion that they justify is that, at higher ages, females are much less subject to fatal accidents than males. In actual fact, for every 1000 infants born, only 2·9 females, as against 3·1 males, die under one year of age as the result of accident.

Again, suppose the case of two towns, A and B. A, with a population of 10,000, has 150 annual deaths, of which 20 are caused by cancer; the general death-rate, therefore, being 15, and the death-rate from cancer 2·0 per 1000, while the deaths from cancer form $\frac{2}{15}$ of the total deaths. B, with the same population as A, has 300 deaths, its death-rate being 30 per 1000, and 40 deaths from cancer, its cancer death-rate being 4·0 per 1000, while the proportion of the deaths from cancer to the total deaths is $\frac{2}{15}$ as before. It is useful to know in regard to each of these individual communities that cancer causes $\frac{2}{15}$ of its total mortality, but no comparison between the two is practicable on this basis. The only proper comparison is between the death-rate from cancer per 1000 of population in A and B, which shows that it is twice as high in B as in A. A still more accurate method is to ascertain the number of deaths from cancer, and the number living at different age-groups, thus avoiding any errors due to variations in age and sex distribution of population.

Errors as to Averages.—The most common of these results from paucity of data (p. 496). Note that the results obtained from an average cannot be applied to a particular case. The mean duration or expectation of life obtained from a life-table expresses, with almost mathematical certainty, the number of years of life of the members of a community *taken one with another*, but is often not accurate when applied to a single individual.

In *army statistics* errors have arisen by failure to comprehend what is meant by the *average strength* of a force. The statistics must comprise the lives of a given number of persons, as well as the deaths occurring among them, *for an entire year*, or allowance must be made in this respect when required.

Hospital statistics for similar reasons are frequently fallacious. Thus death-rates have been frequently given per 100 occupied beds, which are most misleading, as the frequency of succession of patients, as well as the nature of the patients' complaints, will vary greatly in different hospitals. The only proper method of stating hospital death rates is on the basis of the aggregate annual number of cases treated to a termination. The cases should be further subdivided according to age, and sex, and disease.

Average death-rates from epidemic diseases, when used to compare one community with another, may give rise to erroneous conclusions. This is inseparable from the nature of such diseases. During the period under comparison one town may happen to have had, say, three epidemics, and the other four; and possibly, if two or three additional years had been added to the series, the place of the two towns would have been reversed as regards their average death-rate from the disease in question. The proper plan is to give the death-rates from the epidemic disease for every year recorded, to draw a curve of these death-rates for the two towns on the same scale, and to compare the height, the variations of height, and the trend of the curve in each instance.

Vitalia. *See* Diet (*Animal Food, Beef Juices*).

Vitelline.—Relating to the vitellus or yolk sac; *e.g.* the *vitelline circulation* (the vessels of the umbilical vesicle or the omphalomesenteric arteries and veins), the *vitelline duct* (the stalk of the umbilical vesicle or yolk sac), the *vitelline sac* (the umbilical vesicle), etc.

Vitellus.—The yolk of an egg; its chief constituent is the proteid vitellin. *See* Fœtus and Ovum, Development of (*Graafian Follicle*).

Viterbo. *See* Balneology (*Italy, Sulphur*).

Vitiligo.—A cutaneous disease characterised by irregularly shaped patches from which the colour has disappeared surrounded by deeply pigmented areas. *See* Skin, Pigmentary Affections of the (*Leucodermia*); Syphilis (*Parasyphilis, Pigmentary, Syphilide*).

Vitiligoidea.—Xanthoma. *See* XANTHOMA (*Synonyms*).

Vitium.—A disease or defect; e.g. *vitium divinum* (epilepsy), *vitium conformationis* (a malformation).

Vitreous Humour, Diseases of.

CONGENITAL ABNORMALITIES . . . 498
DEGENERATIONS 498
INFLAMMATORY INFILTRATION . . . 499
Treatment 499
INJURIES 500
HÆMORRHAGE 500
Treatment 501
FOREIGN BODIES 501
Treatment 501
PARASITES 501

See also CHOROID, DISEASES OF (*Plastic Choroiditis, Pathology*); DIABETES MELLITUS (*Symptoms, Affections of the Eye*); EYEBALL, INJURIES OF (*Contusions, Hæmorrhage into the Vitreous*); SCLEROTIC, DISEASES OF (*Injuries*).

CONGENITAL ABNORMALITIES. — The vitreous humour is developed from mesoblastic tissue, which passes into the eye through an opening in the secondary optic vesicle known as the fœtal cleft.

It is seen microscopically in the fœtus, to be traversed throughout by a dense network of exceedingly fine branching fibrils which have granules attached to them, mostly at the points where they divide. These granules stain with nuclear stains, but are several times smaller than the nucleus of any cell. Before birth this network of fibrils becomes much spaced out; a great number of them disappear together with the granules. It is the persistence of some of the granules on detached fibrils which give rise to the opacities which are seen enoptically, and known as *muscæ volitantes*. These are of such common occurrence that they may be regarded as a physiological condition.

By looking at a white surface, such as a cloud or a white sheet of paper, through a stenopæic aperture they can be best brought into view, and appear as one or more branching delicate lines of opacity with clear round dots on them; they slowly float in front of the eye.

Some people discover their presence accidentally, and then, by concentrating their attention on them, and daily watching their movements, become much alarmed. Short-sighted people and those who suffer from dyspeptic troubles are the class of patients who most frequently complain of them. They have to be diagnosed from floating opacities in the vitreous of pathological formation, which, unlike the muscæ volitantes, can generally be detected by the surgeon on careful examination with the ophthalmoscope. Patients who have discovered their presence should be told that they are of no importance, and encouraged to try and forget them.

In the fœtal eye the vitreous humour is a vascular structure. An artery passes through the centre of it, starting from the central artery of the optic nerve and proceeding to the back of the lens, where it breaks up into branches which encircle the lens in a fibro-vascular sheath. This central artery of the vitreous lies in a canal bounded by a hyaline membrane, and in the vicinity of the optic nerve is surrounded by a cellular lymphatic sheath.

Before birth this artery and its cellular sheath disappear. Various abnormalities may result from their persistence. The artery may remain throughout its whole length, or for a portion only of its extent, either patent and carrying blood, or reduced to a fibrous cord.

When a fibrous tag alone remains persistent, attached either to the optic disc or the back of the lens, it will sometimes be seen, with the ophthalmoscope, to lash about on movements of the eye in an undulatory way, resembling closely the movements of a worm. Indeed, these persistent tags of the central hyaloid artery have sometimes been mistaken for filaria in the eye.

A portion of the cellular lymphatic sheath which surrounds the artery near the optic disc will sometimes alone persist, the artery having completely disappeared. A white membrane is then seen lying on the surface of the disc in front of the retinal vessels.

When the artery remains persistent, patent, and carrying blood, portions of the mesoblastic tissue which should form vitreous humour may develop into fibrous tissue instead, *atypical development of the vitreous*. Irregular greyish masses of fibrous tissue are then seen in the vitreous chamber with blood-vessels coursing through them, and a history of defect of sight in the eye from birth will be obtained.

DEGENERATIONS. — The vitreous humour in the fully developed eye consists of a clear fluid, very similar in composition to the aqueous humour, contained in the mesh of a delicate network of transparent tissue which is bounded externally by a hyaline membrane, the hyaloid of the vitreous. There are a few fixed cells in the hyaloid membrane and also some wandering cells in the humour itself.

The hyaloid membrane has firm attachments to the optic disc and to the retina at the ora serrata.

A lymph channel bounded by a hyaloid membrane passes through the centre of the vitreous, *the canal of Cloquet*, in which the central artery, already referred to, at one time lay.

After birth the vitreous humour, being devoid of blood-vessels, is nourished by fluid secreted by the ciliary body. Its normal consistency may become altered by inflammatory or hæmor-

rhagic effusions into it, by changes in the ciliary body checking its nutrient supply, and by elongation of the eyeball in high myopia. If the network permeating it becomes broken up it is liquefied, and instead of having the usual viscid character, it is thin and watery like the aqueous humour. It may also shrink and contract away from the structures surrounding it, fluid collecting between them and its hyaloid membrane. This condition is termed detachment of the vitreous; posterior detachment when it is separated from the retina, and antero-lateral detachment when it is separated from the suspensory ligament of the lens.

Liquefaction of the vitreous or *synchisis* can most certainly be detected clinically when associated, as it commonly is, with opacities, the result of past exudate or hæmorrhage into it. These opacities, when the humour is fluid, float rapidly about on movements of the eye. Opacities in the vitreous can be differentiated from opacities in the lens by continuing to move when the eye has come to rest, and by moving in the opposite direction to the eye; when the eye turns up they move down.

The poor support which fluid vitreous offers to the posterior surface of the lens will sometimes allow the lens and iris to tremble on movement of the eye. Tremulousness of the lens and iris is, however, by no means a certain sign that the vitreous is fluid, it may result from defects in the suspensory ligament, and it is not always present when the vitreous is in this condition.

The tension in eyes with fluid vitreous may be normal, very often it is diminished, and when this is the case the eye is very liable, subsequently, to have detachment of the retina or to shrink.

During operations on the eye the surgeon sometimes becomes painfully aware of the liquefying change which the vitreous has undergone by seeing it ooze away from the wound he has made as a very pale yellow-tinted fluid.

In rare cases where the vitreous is fluid it becomes filled with cholesterine crystals; the appearances then seen ophthalmoscopically are of a most beautiful and striking character. The crystals appear as a number of bright golden particles which float about and sparkle with every movement of the eye. The condition, which is spoken of as *sparkling synchisis*, is mostly met with in elderly people, and though associated in some cases with old choroido-retinitis, it may be present without any defect of vision.

Inflammatory Infiltration.—The vitreous humour being a non-vascular structure and containing only a very few cellular elements, can but rarely, if ever, be the seat of a true primary inflammation. The conditions which are commonly spoken of as hyalitis are the result of effusion of inflammatory products into the vitreous from the ciliary body or retina, in cyclitis, retinitis, or choroido-retinitis.

These effusions differ in character in different cases, and may be described as either serous, plastic, or purulent. Clinically they give rise to opacity in the vitreous, cause disturbance of vision, and are associated with symptoms of inflammation of the structure from which they are derived, and which are described elsewhere.

The appearance of the vitreous seen ophthalmoscopically varies with the character of the effusion. When serous, webs and irregular-shaped strands of opacity may be seen, or there may be a general diffuse dust-like haze obscuring all the details of the fundus, and through which the light reflex from the optic disc appears like a full moon in a fog. Such a condition is commonly met with in syphilitic retinitis, whilst either condition may be seen in serous cyclitis or in association with patches of choroido-retinitis.

On subsidence of the active inflammation which gives rise to the serous exudate the opacities tend to clear up and the vision improves. Some liquefaction of the vitreous and floating opacities frequently remain.

When the effusion into the vitreous is of a more cellular and plastic character, the opacity from the first is more dense and of a yellowish colour. Later it tends to develop into fibrous tissue, by the contraction of which the retina frequently becomes detached, and the root of the iris may be drawn back into the circumlental space so as to deepen the anterior chamber at its periphery.

This is the form of effusion which is met with in connection with the cyclitis and retinitis which occur during epidemic cerebro-spinal meningitis, and also in connection with the meningitis in children, the result of ear disease or syphilis. The yellow reflex which it gives rise to from behind the lens simulates that produced by glioma of the retina, for which indeed the condition has several times been mistaken; it is hence often spoken of as pseudo-glioma, under which name it is more fully dealt with elsewhere.

As the result of septic infection of the eye, either through wounds or pyæmic emboli, septic pus may be effused into the vitreous; when this happens suppurative panophthalmitis, with intense injection, much swelling of surrounding structures, and rapid destruction of the eyeball ensues.

Treatment.—The treatment of inflammatory exudation into the vitreous consists in the administration of such drugs as iodide of potassium and mercury with the object of promoting absorption. In the recent serous cases they certainly seem to do good; their use often has to be continued for some months. In cases of some standing the administration of saline mineral waters may prove beneficial.

In the plastic cases no treatment is of much avail, the eye affected frequently after some time shrinks.

In the suppurative cases enucleation or evisceration of the eye becomes urgently necessary for the relief of pain.

INJURIES.—In injuries of the eye the vitreous humour may become displaced. In concussion injuries, if the suspensory ligament becomes ruptured, the vitreous humour may protrude forwards round the side of the lens, between it and the ciliary body, even into the anterior chamber. In such cases, if an incision be made to remove the lens, some of the vitreous is exceedingly liable to present at, or escape from the wound. In some cases, as the result of wounds or from concussion, the lens may be dislocated right back into the vitreous chamber.

In wounds of the eye, from which the lens has escaped or where it is absent, the vitreous humour may pass forward and acquire an adhesion to the cornea. Such anterior synechiæ of the vitreous humour are met with sometimes after needle operations performed for membranes left after the removal of cataracts. A tag of vitreous humour follows the needle on its withdrawal, and becomes entangled at the point of puncture in the cornea.

A tag of vitreous humour prolapses sometimes through a scleral wound, either one inflicted accidentally, or made by the surgeon, in the operation of scleral puncture. A piece of vitreous humour lying prolapsed through a wound, either corneal or scleral, is liable to form the track along which infective organisms may gain entrance to the eye and give rise to inflammation of the uveal tract or retina. In all operative procedures care should be taken to prevent or remove any prolapses of this character.

When the vitreous humour becomes displaced in the way above mentioned there is always, necessarily, some detachment of its limiting hyaloid membrane from one or more of the structures with which it is normally in contact, aqueous or serous fluid occupying the space left.

If a portion of the vitreous humour escapes from a wound, though the eye may fill out and regain its normal tension, no new formation of vitreous takes place, the space left by that which is lost becomes occupied by serous fluid, or a fluid having the character of the aqueous humour. It is surprising in some cases how large an amount of vitreous may escape and yet the eye regain its normal size and dimensions. Detachment of retina is, however, very liable to occur in eyes where a large amount of normal vitreous has been replaced by fluid.

HÆMORRHAGE. — There are three possible sources from which hæmorrhage into the vitreous may proceed, viz. the blood-vessels of the ciliary body, of the retina, or of the choroid, provided the retina has been ruptured or perforated by a direct wound.

It may result either from injury or disease. In some elderly people with degenerate arteries it occurs after straining efforts, such as those occasioned by constipation. Certain young men are occasionally met with who are peculiarly liable to attacks of recurrent hæmorrhage into the vitreous, and in whom no definite constitutional cause has so far been detected. They are usually also liable to attacks of epistaxis, and some of them have been the descendants of gouty parents. Hæmorrhage into the vitreous is also sometimes met with in patients suffering from diabetes.

The clinical appearances presented by hæmorrhage into the vitreous vary with its amount. It may be so extensive that, on examination with reflected light from the opthalmoscope mirror, the whole pupillary area appears dark, none of the normal red reflex being obtained. In some cases the dull reddish hue of the blood-clots can be seen by focal illumination, and in some the hæmorrhage is localised so that only a portion of the normal reflex of the fundus is obscured.

Blood effused into the vitreous may become completely absorbed, leaving only a few floating strands of opacity behind, or the clots may form the matrix for bands of fibrous tissue to develop in. These when formed appear ophthalmoscopically as dense, greyish-white, branching, striated masses in front of the retinal vessels, and having blood-vessels coursing through them continuous with those of the retina.

What is often spoken of as retinitis proliferans commonly arises in this way, *i.e.* by the organisation of blood-clots and the new formation of fibrous bands in the vitreous chamber. As the fibrous tissue develops it contracts, and being adherent to the retina, drags that structure away from the choroid for a portion of its extent.

It is only very extensive and sudden hæmorrhages from the retinal vessels which have sufficent force to extend from the tissue of the retina and break through the hyaloid membrane into the vitreous.

Sometimes blood from a retinal hæmorrhage will collect beneath the hyaloid membrane of the vitreous, raising it away from the surface of the retina over a circular area, like the skin over a blister. Such a condition is most usually met with in the macular region. By the time the surgeon has the opportunity of examining such hæmorrhages opthalmoscopically, they have, from the effect of gravity, altered from being perfectly circular to having a straight upper border. These subhyaloid hæmorrhages may completely absorb, leaving only a granular condition or a few white spots in the locality in which they were situated, or they may break or

diffuse through the hyaloid membrane and render the whole vitreous turbid and opaque.

Treatment.—In non-traumatic cases of hæmorrhage into the vitreous any tendency to constipation should first be remedied by the use of saline purgatives. All stooping and straining efforts should be avoided. Iodide of potassium in five-grain doses three times a day is given, partly on account of the gouty tendency of many of these patients, and partly with the hope of promoting absorption of the blood-clots. In the cases where the hæmorrhages are recurrent half-drachm doses of the liquid extract of ergot have been recommended. The periodical withdrawal of blood from the temple by the artificial leech, or by real leeches, might also be tried.

Foreign Bodies.—Any hard substance, provided it is propelled with sufficient force, may penetrate the coats of the eye and become lodged in the vitreous, but by far the commonest foreign bodies to find so situated are fragments of steel. There are specimens in the Moorfields Hospital Museum showing in the vitreous chamber such strange foreign bodies as three eyelashes carried in by a knife, grains of gunpowder, and a tin tack.

Foreign bodies which are loose in the vitreous tend to gravitate to its lowest part, and rest on the non-plicated part of the ciliary body. Occasionally a foreign body may pass right through the vitreous, and escape from the eye by perforating the coats at a point opposite that of entrance. In other cases, when the foreign body is propelled with less force, it may strike the back of the globe, injuring the retina and choroid, and then rebound, ultimately coming to lie in the anterior and lower part of the chamber.

If the point of entrance is so situated that in the passage of the foreign body into the vitreous the lens is uninjured, and if it is not lying too far forwards, it may be possible to see it with the ophthalmoscope. It is only in a few cases of injury, however, in which this can be done; in the majority it is very difficult to be sure, from the patient's history and symptoms alone, if simply a punctured wound of the eye has been received, or a foreign body has entered and remained in. The patient usually volunteers the statement that he does not think there is anything in his eye, because he is unable to feel it,—his idea of a foreign body in the eye being necessarily associated with great discomfort.

Accurate knowledge of the presence or absence of a foreign body in the vitreous chamber, if it be metal, stone, or glass, and of its exact position, can be obtained by the use of the Röntgen rays and Mackenzie Davidson's cross-thread localising apparatus—the measurements afterwards being worked out on a schematic diagram of the eye.

The entrance of a foreign body into the vitreous is attended by a certain amount of inflammation of the structures in its vicinity, varying in character and amount according to the nature of the foreign body and the germs which are carried in with it. This inflammation may be localised, and only sufficient to surround the foreign body with fibrous tissue; on the other hand, it may be suppurative in character, and rapidly extend to all the structures of the eye.

Treatment.—In a recent case, or one of only a few weeks' duration, in which a piece of iron has become located in the vitreous, its removal should be attempted by an electro-magnet. In some cases, by approaching the eye close to the conical-shaped terminal of a very large and powerful magnet, such as that introduced by Professor Haab, and generally known as the "Giant Magnet," a piece of iron can be drawn out of the eye through its aperture of entrance, or through the suspensory ligament of the lens and pupil into the anterior chamber. From this latter position it can afterwards be extracted, through an incision made with a keratome, by the hand magnet or by forceps.

In some cases, after the position of a piece of iron in the vitreous has been accurately localised, it can best be removed through a meridional incision in the sclerotic made in its immediate vicinity by the introduction into the vitreous of the terminal of a hand magnet.

A foreign body other than iron which has become lodged in the vitreous can only be removed by grasping it with forceps, a very difficult procedure, but one which is facilitated if the exact position in which it is situated is first determined by the X-rays.

Foreign bodies which can be seen with the ophthalmoscope to have become encapsuled, and which are exciting no irritation, may be left in the eye; such cases should, however, be kept under careful observation for some years.

Eyes in which a foreign body is lodged in the vitreous, and which are the seat of either suppurative inflammation or of an inflammation likely to excite sympathetic ophthalmitis, must be enucleated or excised.

Parasites.—The parasite which has most frequently been met with in the human vitreous is the Cysticercus cellulosæ. Filaria are also described as having been seen, and one undoubted case of hydatid cyst in the vitreous is on record (Hill Griffith).

In this country cysticerci in the eye are exceedingly rare. The writer has only seen two cases. Hill Griffith in Manchester states that he met with 6 cases out of 180,000 patients. In North Germany and Poland cases of this sort are much commoner, due probably to the large consumption in those parts of uncooked meat.

The Cysticercus cellulosæ is the cystic stage of the Tænia solium, the eggs of which find their way into the stomach either from a worm

in the intestinal tract of the patient, or are introduced from without either with food or water. The embryos develop in the stomach from the eggs, and by means of hooklets penetrate its walls and enter the circulation. They are then carried to various parts of the body; if they enter an eye they may leave the circulation, growing into a cysticercus either beneath the retina or in the vitreous. If it starts beneath the retina it raises that membrane up away from the choroid, and later on breaks through it into the vitreous.

The ophthalmoscopic appearance of a cysticercus in the vitreous is that of a bluish grey rounded mass, in which often heaving movements at irregular intervals may be detected. The head may sometimes be seen protruded on the surface of the cyst, and its circle of hooklets and suckers be recognised. When the head is retracted it appears as a white, shining spot in the wall. After a cysticercus has existed in the vitreous a short while a fibrous tissue covering forms around it, and it appears as a dense white mass, the diagnosis of which from other conditions may be difficult.

If left, an eye with a cysticercus in it is liable to have attacks of irido-cyclitis and ultimately to shrink. In some cases the parasite has been successfully removed through a meridional incision in the sclerotic.

Vittel. *See* BALNEOLOGY (*France, Calcareous Waters*); MINERAL WATERS (*Calcareous*).

Vitus's Dance. *See* CHOREA (*Synonyms, St. Vitus's Dance*).

Viviparous.—Bearing young that are alive, which are sufficiently developed to be able to live at once apart from the mother, and no longer in structural continuity with her.

Vivisection.—The practice of experiments upon living animals with the purpose of discovering physiological processes and pathological developments with a view to the perfecting of the means of treating diseases by drugs, operation, or other methods. The restrictions under which vivisection experiments can be carried out in this country are detailed in the Cruelty to Animals Act of 1876 (39 and 40 Vict. c. 77), which does not apply to invertebrate animals.

Vocal.—Relating to the voice; e.g. *vocal cords* of the larynx, *vocal fremitus, vocal resonance*, etc. *See* VOICE; AORTA, THORACIC, ANEURYSM OF (*Symptoms*); AUDITORY NERVE AND LABYRINTH (*Nerve Deafness, Affections of other Cranial Nerves*); LARYNX, NEUROSES OF; TYPHOID FEVER (*Complications, Laryngeal*).

Vogt's Point.—The point on the cranium where trephining is performed in cases of traumatic meningeal hæmorrhage.

Voice. *See also* HYSTERIA (*Disorders of Respiration, Aphonia*); NOSE, EXAMINATION OF (*Character of Voice*); PALATE (*Repair of Cleft, Voice Training*); PHYSIOLOGY, RESPIRATION (*Voice*); PUBERTY (*Signs*); STAMMERING.—Normal voice production depends on the following factors:—

1. A healthy condition of the respiratory muscles.

2. A normal capacity of the pharyngeal, oral, and nasal cavities, with good muscular development and action of the various muscles whose action influences the shape of these cavities.

3. Unimpaired functional activity of the nerves and nervous centres which control the muscular movements involved in 1 and 2. All defects in voice production are due to some disturbance in one or more of these factors. In the more important cases of defects in articulation in children all the factors are involved. For purposes of illustration it is advisable to select a type of defective articulation, and we may briefly consider the defect as seen in children when due to neglect of the normal process of respiration with resulting structural defects in the respiratory vocal apparatus.

1. *A Healthy Condition of the Respiratory Muscles.*—Probably the most important cause of voice defects in children is to be found in abnormal respiration. A marked failure to expand the chest properly in ordinary and even in deep respirations is characteristic of these cases. An equally serious defect is present in the expiratory phase of respiration, which is short and uncontrolled. And it is this want of control of the expired airs that is one of the primary defects in defective articulation. The first essential in treatment in these cases, therefore, is the due performance of breathing exercises. The child should be made to lie flat on his back and breathe in and out slowly and deeply for half an hour night and morning, special care being taken with the initial exercises to ensure a proper expansion and slow retraction of the chest. The continuation of these exercises for even a few weeks will effect in most cases a remarkable increase in the respiratory capacity of the child, which will react favourably in voice production.

2. *The Resonating Cavities.*—As a result of long-standing neglect of the normal powers of respiration, *e.g.* in typical mouth-breathers, the shape of the various resonating cavities becomes altered in a manner unfavourable to normal voice production. The most striking alterations are—

(*a*) A narrowing of the nasal cavity. This narrowing takes place on all sides. The palate is abnormally high, and the nasal cavity proper is narrowed by the undue approximation of its osseous walls, and also by a thickening of its lining membrane.

(*b*) Chronic enlargement of the tonsils, with hypersecretion from their surface, and also from the congested pharyngeal mucous membranes.

(*c*) There may be secondary alterations in the aural apparatus, deafness of varying degrees, with indrawn thickened membranes and obstructed Eustachian tubes. When the deafness is pronounced it is an aggravating cause of the voice defect. The treatment of these cases will vary with the degree of the local lesions. Operative measures—removal of tonsils or adenoids or other obstructions in the nose—may be necessary as a preliminary. The subsequent treatment consists in an attempt to restore the unacquired art of normal nasal respiration by the use of exercises such as those already described, and, thereafter, the daily use of vocal exercises conducted under the supervision of a medical man who has special knowledge of the defect and its treatment. In these exercises special attention should be devoted to the particular defects present, *e.g.* if the explosives are specially involved, the exercises should be directed to training the special muscular mechanism involved. In many cases the treatment has to be conducted along lines similar to those laid down for stammerers in vol. ix. Treatment has to be continued for many months.

3. *The Nervous Mechanism involved.*—Defects in voice production due to disturbance of the central or peripheral nervous mechanism are more common in adults than in children. Any conditions which prevent approximation of the cords interfere with normal voice production, *e.g.* paresis of the abductors, and any painful inflammation (laryngitis of different types). But the most striking cause of defective articulation is to be found in hysteria, in which functional aphonia is common.

The treatment of the defect in voice production in these cases should be directed to the general disease, the cure of which will be followed by a return of the voice to its normal.

Volar.—Relating to the hollow of the hand or foot (Lat. *vola*, the hollow of the hand).

Volatile Oils.—Essential oils, such as oil of turpentine, of cajuput, of cloves, of peppermint, of pine, of buchu, and of sandalwood. *See* Oils; Pharmacology (*Volatile Oils*); and under the *various oils* themselves (Cajuput Oil, etc.).

Volition. *See* Physiology, Neuro-Muscular Mechanism (*Cerebrum, Discharging Mechanism, Volition*).

Volkmann's Deformity. — Congenital dislocation of the ankle (tibio-tarsal luxation).

Volkmann's Seat. *See* Spine, Surgical Affections of (*Lateral Curvature, Treatment, Preventive*).

Volsella.—A pair of forceps each blade of which carries one, two, or three sharp prongs; it is used in gynæcology to fix and drag down the cervix uteri; vulsellum. *See* Gynæcology, Diagnosis in (*Volsella*).

Volt.—The unit of electro-motive force, or the force needed to make a current of one ampere to flow against a resistance of one ohm.

Voltolini's Disease.—Purulent labyrinthitis in childhood.

Volvulus. *See* Intestines, Surgical Diseases of (*Obstruction, Volvulus*); Peritoneum, Acute Peritonitis, General (*Etiology*).

Vomer.—A mesial bone of the cranium, shaped like a ploughshare (Lat. *vomer*, a ploughshare), forming the posterior part of the septum of the nose. *See* Palate (*Cleft, Appearances*).

Vomica.—A cavity (Lat. *vomicus*, ulcerated) in an organ, but especially in a lung. *See* Lung, Tuberculosis of (*Physical Signs of Vomica Formation*).

Vomiting. *See also* Abdomen, Clinical Investigation of (*Symptoms*); Abdomen, Injuries of (*Traumatic Peritonitis*); Abdominal Aneurysm (*Symptoms*); Adrenal Glands, Addison's Disease (*Symptoms*); Alcohol; Alcoholism; Anæsthesia, Chloroform (*Vomiting and Retching*); Anæsthesia, Ether (*After-Sickness*); Brain, Tumours of (*Symptoms*); Brain, Inflammations (*Clinical Features*); Brain, Surgery of (*Concussion, Symptoms*); Brain, Surgery of (*Cerebral Abscess*); Bronchi, Bronchial Glands (*Symptoms of Pressure*); Cholera, Epidemic (*Symptoms*); Cocaine (*Uses*); Digestion and Metabolism (*Chemical Examination of the Gastric Contents*); Emetics; Gall-Bladder and Bile-Ducts, Diseases of (*Gall-stones, Symptoms*); Gastro-Intestinal Disorders of Infancy (*Symptoms*); Hernia (*Strangulation, Symptoms of*); Hysteria (*Disorders of Digestive System*); Intestines, Surgical Affections of (*Obstruction*); Labour, Operations (*Induction, Indications*); Leucocythæmia (*Symptoms*); Liver, Perihepatitis (*Symptoms*); Liver, Tropical Abscess (*Symptoms*); Liver, Portal Thrombosis (*Symptoms*); Lung, Tuberculosis of (*Gastro-Intestinal Disturbance*); Malingering (*Digestive System*); Mediastinum (*Growths, Symptoms*); Meningitis, Tuberculous and Posterior Basic (*Symptoms*); Meningitis, Epidemic Cerebro-Spinal (*Symptoms*); Muscles, Diseases of the (*Trichiniasis, Symptoms*); Nephritis (*Chronic and Renal Cirrhosis, Symptoms*); Nerves, Multiple Peripheral Neuritis (*Causes, Arsenical Poisoning*); Pemphigus (*Symptoms*); Pericardium, Diseases of (*Pericarditis, Symptoms*); Peritoneum, Acute Peritonitis (*Symptoms*); Pneumonia, Clinical (*Symptoms in Childhood*); Pregnancy, Affections and

Complications (*Digestive Disturbances*); Pregnancy, Diagnosis (*Symptoms*); Pregnancy, Causes of Death during; Spine, Surgical Affections of (*Injuries, Fracture-Dislocation*); Stomach and Duodenum, Diseases of; Temperature (*Depression, Causes*); Thyroid Gland (*Exophthalmic Goitre, Symptoms*); Toxicology (*Phosphorus*); Trades, Dangerous (*Lead Poisoning, Symptoms*); Uræmia.—*Examination of Vomited Matters.*—In examining vomited matters, the first essential is to filter the vomited matter, and then separately investigate the *solid residua* and *the filtrate*. The main points to be determined are—

(*a*) The reaction of the fluid.

(*b*) The presence of pepsin, or trypsin, or rennic ferments.

(*c*) The presence of bile or blood.

Assistance may be obtained in the microscopic examination by the use of various aniline dyes which have an affinity for the different types of cells present in the vomit derived from the mucous membrane of the stomach, œsophagus, pharynx, or mouth. It is important to bear in mind that vomited matters may consist either of substances present in the stomach when vomiting begins, or of substances entering it during the process. The latter includes saliva, bile, pancreatic juice, blood, and sometimes fæces.

The act of vomiting is induced by a contraction of the stomach muscle with relaxation of the cardiac orifice. Along with the contraction of the wall of the stomach there occurs a simultaneous contraction of the diaphragm and muscles of the abdominal wall. The whole of this mechanism is under the control of a vomiting centre situated in the medulla close to the respiratory centre. This centre is readily excited to action by irritation of various afferent nerves. Of these the most important are—

1. The nerves of the stomach, vagus and sympathetic.

2. The nerves of the liver and gall-ducts, vagus and sympathetic; hence the occurrence of vomiting in cases of hepatic derangement, which may be either primary from a biliary calculus, inflammation, and the like, or secondary to inflammation of the pleural surface of the diaphragm.

3. The nerves of the kidney and bladder. Vomiting is frequent in cases of renal calculus; also in various forms of Bright's disease.

4. Uterine and ovarian nerves. Irritation of these nerves is one of the commonest causes of reflex vomiting.

5. The glosso-pharyngeal nerve to the soft palate, root of the tongue, and the pharynx.

There are a few points of clinical importance *re* vomiting that are liable to be overlooked. In the first place, in children the act of vomiting is frequently the earliest symptom of an acute infective disorder such as scarlet fever, pneumonic fever, acute tuberculosis, and similar disorders. In such cases the vomiting is merely symptomatic, and calls for no special consideration or treatment. Again, in children, but more especially in adults, vomiting is frequently an important symptom of renal disease, often unsuspected.

The treatment of vomiting is the treatment of the disease causing it.

Von Baer's Law.—The fact that in the embryology of the higher animals there are temporary stages representing lower adult forms; the fact that ontogeny gives a condensed phylogeny. *See* Embryology.

Von Baer's Triangle. *See* Scrotum and Testicle, Diseases of (*Supporting the Testicles*).

Von Fleischl's Hæmometer.—An instrument for determining the colour of blood; a fixed volume of blood in an aqueous solution of known strength is compared, as to colour, with a movable tinted glass wedge.

Von Græfe's Symptom. *See* Thyroid Gland, Medical (*Exophthalmic Goitre, Symptoms*).

Von Hacker's Operation. *See* Stomach, Surgical (*Posterior Gastro-Enterostomy*).

Von Jaksh's Anæmia.—A form of primary anæmia, peculiar to the child; there are structural alterations in the red cells, an increase in the leucocytes, and an oligocythæmia and oligochromemia; anæmia infantum pseudoleukæmica.

Von Noorden's Method. *See* Obesity (*Treatment, Dietetic*).

Vowels. *See* Stammering (*Analysis of Ordinary Speech*).

Vox.—The voice, as in *vox cholerica* (the weak, hoarse voice of epidemic cholera), *vox abscissa* (aphonia), and *vox clangosa* (the shrill-voice of laryngeal disease).

Vulsellum. *See* Volsella.

Vulva, Diseases of the.—We shall describe these in the following order:

I. Injuries of the Vulva . . . 505
Accidental Injuries 505
Injuries during Coitus . . . 505
Injuries during Labour . . . 505
II. Inflammation of the Vulva . . 505
Simple Vulvitis 505
Infective Vulvitis (including Gonorrhœa) 505
III. Vascular Disturbances . . . 506
Varicocele or Varix 506
Hæmatoma 506
Œdema 506
Gangrene 506

IV. Cutaneous Diseases of the Vulva 507
Erythema 507
Eczema 507
Herpes 507
Lichen 507
Tuberculosis 507
Syphilis 507
Condylomata 507
Elephantiasis 507
Vulvitis Pruriginosa . . . 507
Kraurosis Vulvæ 508
Pruritus Vulvæ 508
V. Cysts and Tumours of the Vulva 508
(*a*) *Cysts*—
Mucous and Sebaceous Cysts . 508
Bartholinian Cyst or Abscess . 508
Hydrocele of the Canal of Nuck . 509
Hernia 509
(*b*) *Tumours*—
Lipoma 509
Fibroma 509
Myxoma 509
Angeioma 509
Papilloma 509
Sarcoma 509
Carcinoma 509
VI Morbid Conditions of the Clitoris and Hymen 509

See also Generation, Female Organs of (*External Generative Organs*); Generation, Female Organs, Arrested Development of (*Malformations of Vulva*); Gonorrhœal Infection; Hermaphroditism; Labour, Stages and Duration (*Second Stage, Phenomena*); Labour Injuries to the Generative Organs (*Injuries of Perineum, Laceration of Vulva, Vulvar Hæmatoma*); Pruritus (*Clinical Varieties*); Pudenda, Ulcerating Granuloma of Puerperium, Pathology of (*Vulvitis*); Syphilis (*Primary and Secondary*); Tabes Dorsalis (*Symptomatology, Genital Organs*); Uterus, Malformations of (*Under-Development of Vulva*).

I. Injuries of the Vulva.—These occur in three ways: by accident, during sexual intercourse, and during parturition.

Accidental injuries are rare, but may result from a kick, a fall with the legs apart upon such a thing as the back of a chair, or the breaking of a chamber utensil when the patient is sitting upon it. Inasmuch as the vulva is very vascular, hæmorrhage may be very free. Such injuries require to be dealt with on ordinary surgical principles, according to the nature of the case.

Injury during coitus consists mainly in unusual tearing of the hymen. Bleeding from a tear of the hymen itself is always inconsiderable; but if the tear extend beyond the base of attachment of the hymen into the substance of the lateral wall of the vagina, bleeding may be free, and even alarming. Treatment will consist in securing any bleeding vessel and sewing up the tear.

Injury during parturition nearly always consists of a rupture of the perineum; occasionally there may be tearing of the lateral walls of the vagina, extending into the vulva, but then the cause is usually to be found in careless instrumental delivery.

Rupture of the perineum is due to disproportionate size or hardness of the fœtal head; precipitate labour; unskilful use of instruments; or morbid conditions of the perineum, such as undue rigidity or undue softness and friability, such as may result from long-continued pressure of the head. The tear may be partial, involving little more than the lower part of the vagina and the fourchette; or complete, extending through the anal sphincter and up the anterior rectal wall; or it may be of any degree between these extremes. In very rare cases a central rupture has occurred, the anterior part of the perineum remaining intact, while a perforation takes place from the vagina to some place between the fourchette and the anus.

The result of ruptured perineum is, in the first place, a tendency to prolapse of the vaginal walls; and when the tear involves the rectum there is more or less loss of control over the bowel contents.

Treatment.—When a perineum is torn during labour it should be sewn up at once. With due precautions as to cleanliness, union is usually rapid, especially when the tear is due to too rapid delivery. But when it follows a protracted second stage the bruising of the tissues may lead to a failure of union. It is always important that the tear should be repaired on its vaginal as well as on its perineal aspect. If union fails, a secondary perineorrhaphy is required, for a description of which a text-book of gynæcology should be consulted.

II. Inflammation of the Vulva.—Vulvitis may be simple, infective, or of the nature of a dermato-neuritis.

Simple vulvitis may be due to want of cleanliness, irritating discharges from the uterus, or excessive intercourse. In children it is said to result from thread-worms. It may also be a part of a trauma of the vulva. The symptoms are heat, pain, and throbbing in the vulva, aggravated by exercise. The vulva is congested, red, and swollen. If due to injury, bruising may be seen. The treatment consists of rest, thorough cleanliness, and fomentations. Intercourse must, of course, be avoided.

Infective vulvitis is nearly always gonorrhœal, and it then forms part of a more general infection affecting the genital tract. The symptoms are as above, with the addition of two important features, namely, a muco-purulent yellow discharge, and scalding pain on micturition. On physical examination the vulva is red, swollen, and bathed in a yellow discharge, the urethral orifice is nearly always reddened,

and on pressing along the urethra from within outwards a drop of pus can usually be forced out. The orifices of the Bartholinian ducts are often red and unduly patent. Microscopically the gonococcus can be detected in the secretion, usually in the interior of cast-off epithelial cells. The presence of the gonococcus is the only certain indication that the disease is gonorrhœal; but infection of the urethra and Bartholinian ducts is in itself strong presumptive evidence.

Treatment.—The vulvar affection is, of course, the least important part of an attack of gonorrhœa, and the main purpose of treatment is to reach the cervix and vagina. The best plan is a thorough antiseptic washing of the vagina and cervix with biniodide of mercury (1 in 1000), after which douches of a weaker solution of the same lotion (1 in 3000) should be given twice or three times daily. The exhibition of mercury internally often exerts a very favourable effect on the course of the disease. The urethral condition is best treated by diuretics and copious drinking of milk and barley water; if it remains obstinate a dressed probe soaked in a 2 per cent solution of nitrate of silver may be passed into the urethra. Diet should be light, and stimulants forbidden.

Erysipelas of the vulva usually follows labour, and occasionally injuries of the vulva. It presents the same features as when it affects other parts of the body; but owing to the laxity of the connective tissue of the labia there is always much swelling. The inflammation spreads to adjacent parts, such as the thighs, abdomen, and buttocks. The brunt of the affection commonly falls on the labia minora; for, owing to inadequacy of blood-supply, as the result of compression, they may ulcerate or become gangrenous. Treatment must be carried out on general surgical principles.

Diphtheritic vulvitis is characterised by the formation of a membrane, which is composed of fibrin, and stains typically with Weigert's stain: the Klebs-Lœffler bacillus is found in it. It is important to remember that a membranous vulvitis may be non-diphtheritic; in that case the membrane, in contradistinction to the features above indicated, is composed of necrotic material, in which outlines of cells may still be made out, and containing little or no fibrin. The organism present is usually the streptococcus.

Dermato-neuritis is considered under cutaneous affections.

III. Vascular Disturbances of the Vulva.—Under this heading we have to consider varicocele, hæmatoma, œdema, and gangrene.

Varicocele or varix of the vulva is a condition in which the numerous veins of the labia majora assume a dilated and tortuous character. Very often one side only is affected, and the swelling which results may attain the dimensions of a fist. It is not uncommon to find a moderate degree of varicocele during the later months of pregnancy; but any other source of pressure on the veins in the pelvis may produce it. As a rule the vaginal veins share in the dilatation, and varicose veins may be present also in the thighs and legs. When due to pregnancy, the condition commonly subsides after delivery; but it may persist, and in that case, if treatment by rest in the horizontal position fails to restore the normal condition, it may be necessary to excise the varicocele. The operation is usually attended by free bleeding, but when done with care it gives good results.

Hæmatoma of the vulva is an accident that is apt to occur during labour in a case which has presented a varicocele during pregnancy. It is due to bruising and subcutaneous rupture of one or more veins. The feature of the condition is a rapid swelling, beginning in the labium majus of one side, and enlarging till it may attain the size of a fœtal head. As a rule the hæmorrhage ceases spontaneously by its own pressure; and absorption may then be looked for. It is promoted by rest and gentle elastic pressure applied by means of a thick pad of absorbent wool, kept in place by a spica bandage. If the hæmorrhage threatens to become alarming, as indicated by rapid or continuous increase in size, a free incision should be made, the clots rapidly turned out, and the bleeding points secured.

Œdema of the vulva is due to obstruction of lymphatic vessels. It may occur during pregnancy; and we have seen it follow severe vulvitis, and, in one case, an operation for ruptured perineum. The labia minora and prepuce clitoridis are specially apt to be affected; in other cases the œdema spreads over the labia majora and mons veneris. In extreme cases the swelling may form a mass the size of a fœtal head.

Treatment consists of rest in bed, fomentations, and special attention to the evacuations. If the cause is a pelvic tumour obstructing circulation, the tumour must be removed. Pregnancy may require to be prematurely terminated. In some cases the subsidence of the swelling may be hastened by small punctures with a narrow-bladed scalpel.

Gangrene of the vulva is the result of interference with the blood-supply. As we have said, it is particularly apt to occur in cases of erysipelas; we have seen the labia minora slough off in such a case. It may also result from phagedænic ulceration, when the clitoris chiefly suffers; from injuries during parturition; and as a complication of some exanthemata, such as smallpox, scarlet fever, measles, and typhus.

Treatment.—The parts must be kept clean; boracic fomentations should be applied; pain

may be relieved with opium; and the patient's strength must be supported.

IV. Cutaneous Diseases of the Vulva.— The vulva is liable to be affected by a number of cutaneous diseases, of which the most important are erythema, eczema, lichen ruber, tuberculosis, syphilis, condylomata, leukoplakia, elephantiasis, vulvitis pruriginosa, and kraurosis.

Erythema is usually a transient condition, but it may assume a more chronic form as the result of irritating discharges and want of cleanliness. In very stout people it may be due to intertrigo, when it becomes worse in hot weather. Treatment consists of rest, cleanliness, and the use of dusting-powders, such as oxide of zinc, calomel or bismuth subnitrate, mixed with starch or talc powder.

Eczema presents the same characters as in other parts of the body; the mucous surface is not usually involved, but the cutaneous surface shows the typical papules, which become vesicular and break. The escaping serous fluid forms crusts, and the condition is usually aggravated by scratching, which is almost unavoidable owing to the intense irritation. It may be associated with constitutional conditions, such as rheumatism, gout, and diabetes; in other cases it is kept up, if not caused, by irritating discharges from the uterus, or by leakage of urine, as from weakness of the bladder-sphincter or a vesico-vaginal fistula.

Treatment. — Any constitutional or local causes that may be present should be treated. The importance of avoiding scratching should be insisted upon. In the dry forms a lotion of 2 per cent formalin will be found useful, followed by an ointment such as hydrargyrum ammoniatum 1 part in 32 parts of lanolin. In moist forms the crusts should be removed with a strong potash soap, and a dusting powder applied, composed of glutol, 1 part, and talc powder, 7 parts.

Herpes resembles eczema, save that the vesicles are smaller and grouped together. It is apt to come on during pregnancy and at the menstrual periods, and usually runs a short course, if not aggravated by scratching. Local treatment should be carried out on the same lines as for eczema.

Lichen ruber occasionally attacks the vulva; in the course of disappearance it is apt to simulate kraurosis. It usually yields readily to the exhibition of arsenic internally.

Tuberculosis is very rare, and is somewhat difficult to distinguish from syphilitic nodules or ulceration. It may appear in the form of lupus, resembling that seen on the face, in which case it presents one of two forms, being mainly ulcerative or mainly hypertrophic and "tubercular." Histologically such an ulcer is composed of characteristic tubercular granulations, while typical tubercles are scattered through the deeper tissues. Tubercle bacilli may be demonstrated therein. The best treatment is to excise the affected parts.

Syphilis is seen either as a primary sore or chancre, or as mucous plaques and tubercles. In the later stages a condition of leukoplakia may be seen; but it is important to remember that a typical leukoplakia may occur on the vulva quite independently of syphilis, and it may then be the precursor of epithelioma.

Condylomata of the vulva are sometimes syphilitic; but they are also, and indeed more frequently, seen in cases of gonorrhœa where there is no evidence of syphilitic infection. They appear as raised patches with a flattened surface on which red points appear, which may merge into ulcerated patches. The whole vulva may be covered with condylomata, and they may extend backwards and surround the anus. The surface of the vulva is bathed in a muco-purulent discharge. The patient complains of great pain and soreness, which is bad when walking, and which may be even worse when sitting down. This condition yields very rapidly to proper treatment, of which the first and most important principle is cleanliness. Rest should be enjoined as far as possible. The local treatment which we have found the most satisfactory is bathing the vulva several times a day with lotio nigra, and vaginal douching with 1 in 2000 of biniodide of mercury; this is followed by the application of mercurial ointment, and mercuric iodide is administered internally.

Elephantiasis. — This condition is due to chronic inflammation of the lymphatics, with dilatation of their canals, leading to hypertrophy of the cutaneous structures, and a thickened rugose condition of the surface. In the more advanced stages thrombosis occurs in the lymphatic vessels, and later still the glands are affected. The parts principally involved are the labia majora; less frequently the clitoris and labia minora; whilst the affection may spread to the perineum and adjacent parts of the thighs. It is but rarely seen in temperate climates; in the tropics it is more common, and the negro races are the principal sufferers. From rubbing against the thighs and clothes, superficial ulceration is apt to occur. The only effectual treatment is the removal of the mass or masses.

Vulvitis pruriginosa is a dermatitis of the vulva in which the nerve-endings are largely involved, with the result that intense irritation occurs. The skin of the affected parts is red and hot at first; later the surface may become paler and the epithelium appears thickened and sodden; usually marks of scratching are seen. It would appear that in some cases this condition is the precursor of kraurosis. The cause of it is not clear; it is probably due generally to trophic nerve-disturbances. It is doubtful whether it can be brought on by the

irritation of excessive secretions, though these may lead to pruritus.

The treatment is similar to that of kraurosis.

Kraurosis vulvæ is an atrophic disease affecting first and principally the labia minora, and spreading thence to the vestibule, clitoris, and labia majora. The surface becomes very tender, and dyspareunia results. Further, the patient complains of unbearable irritation, which may come on at all times of the day, and is worse at night when the patient gets warm in bed. Inspection of the vulva shows that the surface of the affected parts is smooth, shiny, even glazed-looking; by degrees all folds and creases disappear, and the vaginal orifice becomes contracted until it will barely admit the tip of the finger. Here and there, and especially in the neighbourhood of the urethral meatus and in the situation of the hymen, small reddish patches are seen; these are very sensitive to the touch. When the disease is advanced, the labia can no longer be recognised, and the clitoris and its prepuce almost disappear. The disease usually comes on after the age of forty, and is best described as a progressive atrophy of the vestibule and nymphæ.

Microscopically there is seen hypertrophy of the epidermis, and hyaline transformation of the outer portion of the corium, with entire absence of hair-follicles and sebaceous glands. In the sub-epithelial tissue there is sclerosis of elastic and muscular fibres, great development of fibrous tissue, and small cell infiltration, and small localised hæmorrhages occur. The nerve-endings become compressed, and this probably accounts for the great irritation.

Treatment.—In this condition, as in the preceding, temporary relief may be afforded by sedative and cooling applications, such as evaporating lotions, glycerole of belladonna, or opium or cocaine ointment. A useful lotion is one composed of one ounce each of glycerine and dilute acetic acid, with 10 oz. of a 1 in 20 solution of carbolic acid. In more obstinate cases the affected parts may be painted over, under an anæsthetic, with a 20 per cent. solution of carbolic acid in glycerine: the resulting sore is treated with some sedative ointment. In very severe and intractable cases the diseased area must be freely excised; the cut margin of the skin is then sutured to the cut edge of the vaginal mucous membrane.

Pruritus vulvæ is a symptom which may be due to a variety of causes. These fall naturally into three groups:—

(i.) Irritating discharges.
(ii.) Diseases of the vulva.
(iii.) Reflex irritation.

Under the first heading are comprised conditions causing profuse leucorrhœa, and irritating conditions of the urine, as in diabetes, cystitis, and vesico-vaginal fistula. In the second group we have the vulvitis and kraurosis already described, as well as infection of the pediculus pubis, and congestion of the vulva due to varicose veins or hyperæmia. The latter is apt to come on during pregnancy, and also during menstruation, in which case the pruritus is periodic. Pruritus is also associated with masturbation; but whether as cause or effect it is not always easy to decide. In the third group we find reflex disturbances arising in the rectum and bladder.

The treatment of pruritus will depend on the causal condition; and this should, therefore, be always carefully inquired into.

V. Cysts and Tumours of the Vulva.—(*a*) *Cysts.*—These are found as mucous and sebaceous cysts; cysts of Bartholin's gland or duct; and hydrocele of the canal of Nuck. Hernia is included here for convenience.

Mucous cysts are found on the inner surface of the labia minora. They are small and uncommon, and are treated by incision or excision.

Sebaceous cysts resemble such cysts in other parts of the body. The small black spot marking the orifice of the duct shows their true character. They are liable to suppurate, and should be treated by excision.

Cysts of the duct of Bartholin's gland are due to occlusion of the orifice of the duct; this is sometimes incomplete, and the swelling then varies in size according to the quantity of fluid present. This fluid is usually viscid, but is sometimes watery. In chronic cases the gland itself is involved in the cyst wall. The patient complains of fulness in the vulva, and pain or discomfort when walking, sitting, or during sexual intercourse. On inspection, a pyriform swelling with apex upwards is found in the lower or posterior portion of the labium majus; and the orifice of the duct is nearly always red and unduly prominent. In cases of gonorrhœal or septic infection the contents of the cyst suppurate, resulting in a *Bartholinian abscess.* This presents the same symptoms and signs as a cyst; but pain is usually more acute, and the skin over it is commonly reddened.

The position of the swelling distinguishes it from a hernia and from a hydrocele of the canal of Nuck, in both of which conditions the swelling starts in, and principally affects, the upper or anterior part of the labium in the neighbourhood of the inguinal canal. The only other condition that can be mistaken for a Bartholinian cyst is hæmatoma of the vulva; this is more diffused, doughy, attended with ecchymosis, and has not the pyriform shape characteristic of the cyst.

Treatment consists in excision of the cyst. Its walls are usually well-defined, and it can generally be enucleated entire from the surrounding structures without difficulty. A few deep sutures should be passed under the bed of the cyst, to check oozing; and the skin-margins should be brought accurately together. An

abscess must be similarly dealt with; but the separation of the wall of the abscess is usually more difficult, because it becomes closely incorporated with the parts around it, and it is best to insert a drain for twenty-four hours.

Hydrocele of the canal of Nuck is the result of the persistence of a peritoneal pouch by the side of the round ligament, with subsequent closure of its abdominal end. The cavity becomes distended with fluid and appears as an oblong swelling extending from the inguinal canal towards the labium majus. It is distinguished from a hernia by being irreducible, and by a more or less distinct interval between it and the inguinal ring. It is not, however, always possible to distinguish it from a hydrocele of an old hernial sac.

The treatment is excision.

Hernia appears in the vulva in the inguinal form. When large it may greatly distend the labium majus, entering it from above.

(*b*) *Tumours.*—The following are met with in the vulva: lipoma, fibroma, myxoma, angeioma, papilloma, sarcoma, and carcinoma.

Lipomata arise in the labia majora and sometimes in the mons veneris. They are usually sessile and seldom attain a large size. They can be left alone unless they cause discomfort, and then they should be enucleated.

Fibromata are rare, and seldom large; but occasionally a fibroma may form a large pedunculated tumour, causing dragging pain by its weight. These tumours are easily removed.

Myxomata form soft pedunculated masses, arising generally from the labium majus. The pedicle is nearly always narrow. Sometimes the skin covering them is deeply pigmented. They can be removed if they produce discomfort.

Angeiomata are sometimes seen as nævi, mostly in children: more rarely a plexiform angeioma occurs. A nævus seldom requires to be interfered with; a plexiform angeioma is best treated by electrolysis.

Papillomata or warts are common; the ordinary simple wart may be present, but more often papillomata are associated with irritating discharges in cases of gonorrhœa. They are usually pedunculated, but may be sessile; and a considerable area may be covered by them.

Treatment.—Gonorrhœa, if present, must be energetically treated, and the vulva must be kept scrupulously clean. Considerable improvement may usually be effected by the use of mercurial lotion and ointment; if the papillomata persist they should be snipped off, and their bases touched with nitric acid or solid nitrate of silver.

Sarcoma is only rarely met with in the vulva; and it usually takes the pigmented form (melanoma). As a rule the growth is disseminated over other parts of the body, and the outlook is very unfavourable. A localised solitary sarcoma should be freely removed.

Carcinoma of the vulva generally attacks the clitoris or one of the labia majora, and is of the squamous form (epithelioma). When the clitoris is involved, this structure becomes replaced by a mass which may attain the size of a small egg. The surface is usually ulcerated in parts. On the labia majora carcinoma commences as a wart-like structure, which enlarges rapidly and soon breaks down on the surface, forming an ulcerating mass discharging a fœtid secretion. Carcinoma has been known to arise in a vulva affected with kraurosis, and we have met with an example. The glands in the inguinal region become affected, although they may remain free for a relatively long time. Once they are involved the swelling in the groins rapidly increases and spreads to the interior of the pelvis. When the case is seen early enough free removal of the disease gives a favourable prospect of cure. The vulva should be dissected away at some distance from the growth; and the cut edges of healthy skin can generally be united to each other and to the cut edge of the vaginal mucosa. Any glands that can be felt in the groins should be removed at the same time.

Adeno-carcinoma is rarely seen in the vulva; when present it involves the Bartholinian gland. It grows rapidly and shows a marked tendency to recurrence after removal.

VI. Morbid Conditions of the Clitoris and Hymen.—These can be briefly dealt with. Some of the morbid conditions of the clitoris have been described, such as carcinoma and elephantiasis.

Inflammation of the clitoris may be a part of a general vulvitis; or it may be due to want of cleanliness, leading to accumulation of secretion under the prepuce. This may lead to excoriation and even to adhesion of the glans to the prepuce; and considerable irritation may be set up in consequence. Sometimes the pent-up secretion becomes inspissated and hard, forming a kind of concretion.

Treatment.—When the margins of the prepuce are adherent they should be separated with a probe or director; in old cases some dissection may be required to remove a concretion. Cleanliness will generally accomplish the rest.

The *hymen* is sometimes unusually rigid, leading to difficulty in intercourse; and in rare cases it may remain unruptured and present an obstacle to child-birth, conception having occurred without complete penetration. At other times the hymeneal orifice may be unusually small, or it may present a transverse or longitudinal band, so that there are two orifices. The so-called "imperforate hymen" is really a condition of vaginal atresia; the hymen may usually be distinguished on the under surface of the septum. This is dealt with under "Malformations of the Vagina." A rigid hymen will require incision and stretching to allow of coitus taking place.

*Caruncul*æ *hymenales* are the remnants of the hymen after intercourse has taken place. After child-birth the hymen becomes further destroyed, its remnants being then known as *caruncul*æ *myrtiformes*. Both kinds of caruncle may become acutely tender and painful, leading to dyspareunia. A number of congested spots resembling small recent bruises or petechial hæmorrhages are then seen. They require to be excised.

Small cysts lined with epithelium have sometimes been found in the tissues of the hymen.

Vulvar or Vulval.—Belonging to the vulva, *e.g.* vulvar abscess (abscess of the Bartholinian gland), vulvar canal, vulvar condylomata, etc. *See* VULVA, DISEASES OF.

Vulvitis.—Inflammation of the vulva or pudenda. *See* VULVA, DISEASES OF (*Inflammations*); PUERPERIUM, PATHOLOGY, PUERPERAL INFECTION (*Vulvitis*).

Vulvo-.—In compound words *vulvo-* means relating to the vulva; e.g. *vulvo-vaginitis* (inflammation of the vulva and vagina), *vulvo-uterine* (relating to the vulva and uterus), *vulvo-vaginal*, etc. *See* VULVA, DISEASES OF THE; VAGINA, DISORDERS OF THE; etc.

Wachendorf's Membrane.—The membrana pupillaris. *See.*

Wachsmuth's Mixture.—An anæsthetic mixture containing one part of oil of turpentine to four parts of chloroform.

Wade's Balsam or Drops.—Compound tincture of Benzoin; Friar's Balsam. *See* BENZOINUM.

Wagner's Corpuscles. *See* SKIN, ANATOMY AND PHYSIOLOGY (*Tactile Corpuscles or Meissner's Bodies*).

Wagner's Operation. *See* BRAIN, SURGERY OF (*Trephining, Osteoplastic Method*).

Wahlberga. *See* BALNEOLOGY (*Norway and Sweden*).

Waistcoat, Strait. *See* INSANITY, GENERAL TREATMENT OF (*Mechanical Restraint*).

Walcher's Position.—The hanging thigh position; the body of the patient is brought so that the buttocks rest on the edge of the operating table or bed and the legs are allowed to hang over the edge; it is of use in cases of contracted pelvis in labour, for it causes a slight increase in the size of the conjugate diameter of the pelvic brim. *See* LABOUR, PRECIPITATE AND PROLONGED (*Treatment of Labour with Contracted Pelvis*).

Waldeyer's Epithelium.—The germinal epithelium covering the free surface of the ovary during development.

Walking. *See* CHILDREN, DEVELOPMENT OF (*Motor Functions, Age of Walking*).

Wallerian Degeneration.—The degeneration which takes place in nerves which have been separated from their trophic centres. *See* PHYSIOLOGY, NEURO-MUSCULAR MECHANISM (*Spinal Cord, Conducting Paths, Wallerian Method*).

Walther's Ducts.—The ducts of the sublingual salivary gland.

Walther's Ganglion.—The coccygeal ganglion or ganglion impar.

Wandering.—Changing position; *e.g.* a *wandering kidney, liver, or spleen* (floating kidney, liver, or spleen), a *wandering tumour* (*e.g.* a dermoid ovarian tumour or a uterine fibroid which has got separated from the organ from which it grew), and a *wandering rash* (superficial glossitis of the tongue). *See* SPLEEN, SURGERY OF (*Movable Spleen*); TONGUE (*Glossitis*), etc.

Wanklyn's Process.—The ammonia method of estimating the nitrogenous organic matter present in water.

Warburg's Tincture.—A diaphoretic and antiperiodic mixture (*tinctura antiperiodica*) containing quinine, aloes, opium, rhubarb, camphor, and various aromatics; it is used in doses of 1 to 4 fl. dr. in malaria. *See* MALARIA (*Treatment*).

Wardrop's Disease.—Paronychia or onychia maligna. *See* NAILS, DISEASES OF THE (*Onychia*).

Ward's Paste.—Confection of black pepper.

Warmbrunn. *See* BALNEOLOGY (*Germany, Thermal Waters*).

Warming. *See* VENTILATION AND WARMING.

Wartpox.—An abortive type of smallpox, in which about the fifth day the vesicles dry up instead of filling with pus.

Warts or Verrucæ. *See also* DERMATITIS TRAUMATICA ET VENENATA (*Tar Workers*); EAR, EXTERNAL, DISEASES OF (*Epithelioma of External Meatus*); LUNG, TUBERCULOSIS OF (*Complications, Integumentary System, Warts*); VENEREAL DISEASE (*Allied Diseases, Venereal Warts*); VERRUGA PERUANA. —VERRUCA VULGARIS or Common Wart.—Common warts usually occur about the backs of the *hands* and *fingers*, but sometimes also on the palmar aspect. The groove along the side of the nail is a favourite situation. They are

more often seen in children, and in them are generally multiple. In adults they are more often single. Common warts are more or less hemispherical in shape, or with a flattened top, especially on the palms. Their surface is irregular, rough, and, when of long standing, more or less dirty in colour. On the *face*, although they begin in the same way, they may become more elevated and wider at the top than at the base. The filiform warts of the *eyelids* and *neck* are well known. On the *scalp* they are digitate or cock's-comb-like. In this situation they may be single and last for years. A more uncommon localisation is the *sole*, giving rise to great pain, and unfitting the sufferer for work. Warts of the *lips* have been described, but in one case exhibited as such the condition was, in my opinion, inflammatory exfoliation of the lips (cheilitis exfoliativa).

They are contagious from person to person, and auto-inoculable.

Common warts are epidermal growths (infectious acanthomata, Unna), with an accompanying elongation of the papillæ.

Epithelioma may occur as a complication, and cutaneous horns sometimes develop from warts. In acanthosis nigricans (keratosis nigricans), they are part of the clinical picture in some of the recorded cases.

Treatment.—"Charms" for the cure of warts are well known to folk-lorists. There is no doubt that warts have disappeared after "incantations." This "suggestion cure" has led to the idea of an etiological nerve origin as opposed to the microbial view. But warts sometimes go spontaneously.

For warts, various acids, glacial acetic, trichloracetic, nitric, and also a saturated solution of chromic acid in water (this acid may form a dangerous compound with spirit); or alkalies, such as caustic potash, may be used. Care must be taken to prevent these remedies running over on to the sound skin. Slicing the warts away with a scalpel and then touching the exposed parts with pure carbolic acid is another way. In obstinate cases of hard, long-standing warts of the hands curetting answers best. As to the painful plantar wart, Radcliffe-Crocker recommends electrolysis, by means of a stout, curved, flat surgical needle passed through the base. The digitate and filiform warts should be ligatured with fine silk. When radical measures are objected to, a salicylic acid plaster can be used to soften and remove the top layers, and then an aqueous chromic acid solution applied. Dirty and disfiguring applications should be avoided as much as possible for the face.

Among the internal remedies, arsenic and thuja occidentalis may be mentioned. Colrat of Lyons recommended sulphate of magnesia in doses sufficient to ensure two or three motions a day. This plan certainly answers in some cases.

VERRUCÆ PLANÆ JUVENILES. — This is a variety of wart which deserves separate mention, especially from the clinical point of view. They are usually seen on the backs of the hands and on the face, but they may attack other parts of the body. Individually they are roundish, from a pin-point to a lentil in size, slightly raised above the level of the skin, flat, and from a dull reddish hue to a shade of yellow or brown. They are numerous, in some instances slowly increasing in numbers, in others coming out rapidly in crops. They are sometimes preceded, for some time, by a single flat wart (mother wart).

In the case of a girl, aged 13, the backs of the hands and fingers were thickly covered with small flat warts. They had been present for nearly four years. There was a history of warts in adult members of the family, and latterly a lady friend of the child had also developed a few about the hands.

In contrast with this progressive case, a more acute instance observed by Dr. Chalmers Watson may be briefly referred to. The patient, a boy, aged 13, had numerous flat warts about the face, some distinctly arranged in lines, as is not unusual. They had been present six to eight months, and had increased greatly in number after an attack of influenza. The boy had had warts on his hands for a year or more. The warts disappeared under treatment appropriate to the constipation from which the patient suffered. The case is recorded and illustrated in the *Brit. Jour. of Dermatology*, May 1903.

The small flat wart I have also seen in great numbers on the sides of the face of a man, in whom they appeared to have been spread by shaving.

Verrucæ planæ juveniles could not well be mistaken for lichen planus, especially on the face, where the latter disease is of extremely rare occurrence.

Treatment.—Here, again, sulphate of magnesia should be tried. Arsenic is said to clear them off in a few weeks, and thyroid is also stated to have got rid of them. Locally, radical measures are out of place. Washing with soft soap has been recommended, as also various ointments (salicylic acid, resorcin, and so forth). Wiping the parts over with liq. carbonis deterg., sp. vini rect. pt. æq., is useful. In the case of the adult mentioned above, the warts disappeared after perseveringly applying a saturated solution of salicylic acid in alcohol.

VERRUCÆ SENILES, Senile Warts, also called Seborrhœic Warts.—These occur about the face, trunk, and arms in elderly and old people. They are usually of finger-nail size, raised, more or less round, greasy, and of a dirty brown or even black hue. On removal of the superficial portions, by scratching with the nail, the elongated papillæ show up beneath.

They may be present in great numbers sparsely distributed. Radcliffe-Cro... seen a copious crop on the chest, a... only with acute eczema, in an elderly woman has

In a case of my own, a number of th... ted size of lentils, developed about the ex... surfaces of the wrists and lower part of... forearms, in an elderly lady in the course... influenzal patchy pneumonia. The warts could be partially scraped away with the nail. They came out two or three at a time; some had involuted, leaving light sepia pigmentation. She also had two small warts about the right thumb which followed a burn. When I saw the patient there was also post-influenzal desquamation about the hands and feet.

Epithelioma may occur as a complication (*see* Pernet on "TUMOURS OF THE SKIN," vol. x. p. 168).

Treatment.—The affection is a trifling one. Soap and water and cleanliness generally are the only things needful. If their removal should be demanded, thorough curetting would be best. Any senile wart showing signs of ulceration and growth should be excised.

VENEREAL WARTS are observed about the genito-anal regions and adjacent parts of thighs in women, and in the neighbourhood of the corona glandis in men. They commence as minute lesions, which increase in size and become tufted and pedunculated. They may become very large and cauliflower-like, with inflammation and a fœtid discharge, causing great inconvenience and pain. These growths are associated with irritating discharges such as occur in gonorrhœa, pregnancy, chancres, etc.

Although experimental inoculation has failed, there is no doubt as to their being contracted as a result of impure intercourse. Diday was of opinion that individuals suffering from them had, or had had, warts about the hands.

In a case of warts about the glans penis, recently under my care, there was a history of their commencing ten days after coitus. The warts were millet-sized and flat, with here and there small tufted ones. On the hands I found two hemp-seed-sized flat warts, which the patient had not noticed.

Treatment. — Cleanliness first, and keeping them dry with boric acid powder may suffice. Dusting with talc containing a little salicylic acid, or with the following:—Pulv. frondum sabinæ, pulv. aluminis, āā 10, pulv. cupri sulph. 1 (Finger of Vienna); or they may be removed when large by means of the galvano-cautery. Finger warns against ligaturing them. In pregnant women they may disappear spontaneously after confinement. Nothing should be done during pregnancy, except in the way of cleanliness. Small warts of the penis can be lightly touched with pure carbolic acid, nitric acid, or acid nitrate of mercury, applied with a wooden match end.

WARTY STREAKS.—This condition comes into the category of *ichthyosis* (*hystrix*) *linearis*.

VERRUGA PERUANA (*verruga*, a wart) is a serious acute infective disease of Peru, with multiple lesions (also called Carrion's disease). In some recorded cases only a few verrugas have occurred, and that some time after leaving infected districts of the Andes (see p. 441).

Warty Lupus. *See* SKIN, TUBE[RCU]-

Wa... (*Lupus Verrucosus*).

Wa... —A lotion, *e.g.* black, red, wh... e, or yellow wa... *Sub-acetatis Dilu...* *See* LEAD (*Liquor Plumb* ...s); MERCURY.

Washerwo...an's Itch. ...ZEMA.

the hands in washerwo...men. *See* ...shing out of a

Washing Out.—The ... of the stomach hollow viscus, more especiall... INJURIES AND DIS- and bladder. *See* BLADDER, ...ment); STOMACH EASES OF (*Chronic Cystitis, Trea...latation, Treat-* AND DUODENUM, DISEASES (*D...* *ment*). *Stings*); STING-

Wasp. *See* MYIASIS (*Insect* ING INSECTS.

Wasserman, Reacti...n of. *See* E KNOWLEDGE SYPHILIS, RECENT ADVANCES IN T... *Reaction*). OF (*Serum Diagnosis, Wasserman...* Y; CHILDREN,

Wasting. *See* ATROPH... *Limbs*, etc.); CLINICAL EXAMINATION OF (*Head*... YPHILIS (*Chil-* HYDROCEPHALUS (*Symptoms*); S... *dren, Early Signs*). AMINATION OF

Watch-Test. *See* EAR, E... (*Watch Test of Hearing*).

Water.

Various
Quantity of Water required for v... . 513
purposes 513
Intermittent versus *Constant Supply* ...ater 514
Dual Supplies . . . . . de-
What constitutes a Hygienically Pure W... ter,
Various Sources from which Water is ...ell
rived: Rain Water, Surface Wa... of
Spring and Subsoil Water, Deep-u... 514
Water, River Water, and Causes ...r-
Pollution . . . . . re,
Diseases produced by Impure Water: Dia... 516
rhœa, Typhoid Fever, Cholera, Goit... us
Plumbism, Entozoal Diseases . . 518
Protection of Water Supplies from vario... le.
sources 519
Purification of Water on the large sca... ds
Domestic Purification . . . 519
Hard Water versus *Soft Water. Metho...*
of Softening 520
Chemical and Bacteriological Examinati...
of Water. Methods of Analyses
Interpretation of Analyses . .
Collection of Samples for Examination
The Law relating to Water Supplies

See also AIR, EXAMINATION OF (*Water Vapour*); ASEPTIC TREATMENT OF WOUNDS (*Water Infection, Hot Water*); BALNEOLOGY; CHOLERA, EPIDEMIC (*Etiology*); CHOLERA NOSTRAS (*Etiology*); DIET (*Beverages*); DYSENTERY (*Etiology, Water*); HYDROPATHY; MINERAL WATERS; SEWAGE AND DRAINAGE; THYROID GLAND, MEDICAL (*Goitre, Causes*); TOXICOLOGY (*Lead*); TRADES, DANGEROUS (*Lead-Poisoning*).

WATER, though not strictly speaking a food, is absolutely necessary for the maintenance of life, both of plants and of animals. In the human economy it is required as a solvent and as a diluent. For these purposes only, a very small quantity would suffice, probably two to three pints per person per day. The exigencies of modern civilisation, however, demand a far larger supply. Water is required for the cooking of food, for the cleansing of the person, of the house and its appurtenances, for animals and stable purposes, and for gardens. All these may be considered under the head of water for domestic use. The public authorities require water for cleansing streets, flushing sewers, and extinguishing fires, possibly also for supplying baths and washhouses. These may be considered as municipal purposes. Moreover, in most towns it is found to be to the public advantage to supply water for trade and manufacturing purposes.

The amount of water required daily for domestic use varies considerably. Excluding stable and garden, the amount usually allotted in making estimates is 10 gallons per head per day. Where great economy is used, and where earth-closets are provided, this amount might suffice, but it is less than the estimate made by the late Dr. Parkes, based upon records of the amounts used in various households. The estimate is as under:—

	Gallons daily per person.
Cooking	·7
Fluid as drink (water, tea, coffee, etc.)	·3
Ablution, including a daily sponge bath	5·0
Share of utensil and house washing .	3·0
Share of laundry washing . .	3·0
Total . .	12·0

Where water-closets are used from two to six gallons more will be required. In a water-closet town, therefore, it would not be safe to provide a supply of less than 20 gallons per head per day for domestic purposes. Water not sufficiently pure for domestic purposes is often available for municipal and manufacturing purposes; where such is not the case it is usual to allow 10 gallons per head per day for each of these uses, hence we may conclude that the amount of water actually required will vary from a minimum of 20 gallons to a maximum of 40 gallons. There are many towns not providing this minimum quantity, but in all such there are periods when the supply is inadequate. In other towns more than this maximum is provided, but in such cases the excess is almost certainly wasted, either on account of defective mains or house fittings, or the carelessness or extravagance of the users.

In many towns an attempt is made to economise the use of water by making the supply "intermittent." In such case the mains are only kept full and under pressure for a few hours daily, when the house cisterns fill and are drawn upon for the remainder of the day. Theoretically this method should be economical, but in actual practice it leads to little if any saving, moreover there are many objections to it. The emptying and filling of the mains and the great variations in pressure often lead to insuction of polluting matters, larger house cisterns than would otherwise be necessary must be provided, again increasing the risk of contamination, larger mains are necessary, as the whole day's supply has to be furnished in a few hours, and, in case of fire, water may not be available.

In rural districts where public supplies are laid on by means of mains the amount of water required is not so great as in towns. Usually when the public supply is first furnished the amount used is astonishingly small, probably due to the continuance of habits of economy acquired when the supply was limited. In time, however, the demand increases, but usually the provision of 50 gallons per day per cottage is found to be abundant.

For many of the purposes for which water is required it is not necessary for it to be "pure." Sea water or an impure river water may be used for sewer flushing, road watering, and other municipal purposes; an impure water may also serve for manufacturing purposes. Even for certain domestic purposes a water not sufficiently pure for drinking and cooking might be tolerated. With a *dual supply* the amount of pure water required would be greatly decreased, and possibly in the future dual supplies may be introduced into towns where the available amount of pure water is limited. In country districts such dual supplies are very commonly met with. The well yields water for certain domestic purposes, whilst a neighbouring stream, ditch, or pond furnishes water for washing and slopping. There is little objection to such an arrangement, but in towns a dual supply would involve a second system of mains and supply-pipes, and if laid on to the houses there would be a constant risk of the impure water being used for the pure. This risk might be reduced to an almost negligible quantity, but it is doubtful whether the health of any community would be benefited by the introduction of say a sewage-polluted river water for

street watering, clothes washing, etc. At present, however, pure water can in nearly all cases be obtained in sufficient abundance as to render it more economical than a dual supply. In other words, it is cheaper to use pure water for municipal purposes than to lay down a second system to provide an impure water. On the coast several towns have during recent years utilised sea water for road watering and sewer flushing.

The terms *pure* and *impure*, as applied to water, are merely relative. A "pure" water does not occur in nature; water always contains gaseous and solid matters in solution, and dead and living particulate matters in suspension, taken up from the air or from the soil, or from both. What these matters are will be discussed later. All natural waters, therefore, are more or less impure, and when the term "pure" is applied to water we simply mean that the water contains no substances which, either from their quantity or quality, are likely to endanger the health of the persons using it. It is in this sense that the word is used throughout this article. An impure water need not of necessity be actually injurious to health. Water contaminated with sewage matter is often used, even by considerable communities, for long periods without the slightest apparent ill effect, but there is always the risk of such a water becoming infected with the specific organism of enteric fever, and possibly of cholera and other diseases; hence its use is attended with a certain amount of danger, and it does not, therefore, come under the definition of a "pure" water. It is impure and unsafe. The distinction is important, as the lay mind finds it difficult to understand that a water which is being used and does not cause any apparent injurious effects may still be impure.

The nature of the substances found in water will be best understood if *the source from which our supplies are obtained* is briefly considered.

All water is originally derived from the ocean. Roughly speaking, three-fourths of the surface of the globe is covered with water. In tropical regions the water evaporates into the superincumbent air with great rapidity. Anstey calculates that on an average about 700 gallons of water is evaporated every minute from each square mile of ocean surface, and Symons declares that "all the coal which men could dig from the earth in many centuries would not give out enough heat to produce by the evaporation of water the earth's rain-supply for a single year." Winds blowing over a large expanse of ocean, therefore, become laden with moisture, and as soon as a cooling effect is produced by the proximity of land, especially if elevated, rain descends. For these reasons the rainfall is heavier on the western side of the British Islands than on the eastern, and greater in mountainous districts than on plains. The amount of rain which falls is measured by the rain-gauge, and expressed in inches of depth. A rain-fall of one inch in depth corresponds to about $4\frac{3}{4}$ gallons on a square yard, or 22,620 gallons on an acre. Rain as it falls is the purest water which occurs in nature. It contains in each gallon about 8 cubic inches of gases, chiefly nitrogen and oxygen, with a little carbonic acid, and traces of saline matter, common salt, sulphates, etc., together with particles of suspended matter, soot, dust, microbes, etc., all of which have been taken from the air. The water which falls during the first portion of a shower, especially after a drought, is always more impure than that which falls later. As usually collected from the roofs of houses rain water is generally very filthy. Ordure of birds, soot, vegetable débris, etc., accumulate on the tiles or slates and in the spouts during the dry seasons, and are washed into the rain-water receptacle with the first heavy shower. These offensive matters undergo solution and putrefaction, but in some cases an apparently good potable water results. Rain water so collected is, however, unpalatable and unsightly, and too impure for drinking purposes. A much better water can be obtained if a rain-water "separator" is used. By attaching this little apparatus to the down spout the first portion of each shower, that portion which brings with it all the filth, is cast into the drain, whilst the water collected after is passed into the receptacle placed to receive it. It is unfortunate that in very few instances can enough rain water be collected from a roof to supply the requirements of the house. In summer time many slight showers occur which add little to the store, the water evaporating almost as quickly as it falls. When heavy downpours occur a good deal is wasted, the whole not being collected by the gutters or troughs. The amount which may be collected is from one-half to two-thirds of the rainfall. The area covered by the roof of an average cottage does not amount to more than 35 square yards. With an average rainfall 15 to 18 inches could be collected. This would amount to about 3000 gallons in the year, or 9 gallons per day. To collect this quantity a storage tank of about 1500 gallons' capacity would be required. With a smaller tank water would run to waste in the rainy season, and the accumulation would not suffice to tide over a dry summer. Tanks of brickwork lined with cement, or of slate, are the best. Galvanised iron tanks are largely used, but the water drawn therefrom usually contains a trace of zinc, which, however, is apparently harmless. Lead tanks yield a taste of metal to rain water, and as lead is a cumulative poison, this metal is not to be recommended.

Rain water collected from the surface of rocks in mountainous regions forms one of the best sources of supply for large communities. The water may either be collected in natural lakes,

as in Loch Katrine, for the supply of Glasgow, or in an artificial reservoir or lake, as at Vyrnwy, Wales, for the supply of Liverpool. If the hills are covered in part with peat the water may become more or less discoloured—"peaty." This peaty matter, unless in excess, is quite harmless, and such water if stored for a length of time in open reservoirs gradually bleaches. A water strongly impregnated with peat is believed to give rise to attacks of diarrhœa, especially amongst those unaccustomed to its use. Quite recently it has been demonstrated that in peat there are acid-producing bacteria capable of giving an acid reaction to the water, and such waters act markedly [illegible] lead, zinc, and iron. In the north of England [illegible] sands of cases of "plumbism" have occurred from the use of such waters distributed through leaden pipes. Surface water collected from the surface of moorlands upon which farms are situated is liable to pollution, and should not be used unless stored for a length of time, and finally carefully filtered before being distributed.

When rain falls upon a pervious surface, as of sand, gravel, and chalk, a portion penetrates the soil and accumulates there. It is then known as subsoil or ground water. Beneath the pervious rock there is always, at a greater or lesser depth, a stratum of an impervious character which holds up the water. The surface of this stratum usually sloping, the water generally travels more or less quickly in a given direction towards an outlet into the stream draining that particular area. Where the impervious stratum comes to the surface or outcrops the water will issue forth as a spring. Such spring waters are usually bright and pure on account of the water having travelled slowly, and for a considerable distance through the pores of the pervious rock. This subsoil or ground water is yielded by all shallow wells, and forms a most important and much maligned source of supply. The evil reputation of shallow well water is not due to the character of the subsoil water, but to the unfortunate sites selected for wells, and the unscientific and careless method of construction. The rain falling upon pervious ground, ground more or less cultivated, naturally takes up not only mineral matters from the soil, but also organic matter from manure, decaying vegetation, etc. Fortunately there are found in all fertile soils micro-organisms which rapidly act upon the dissolved organic matter, converting it into harmless carbonates and nitrates, and the natural filtration of a compact soil quickly removes all, or nearly all, suspended matter, including the bacteria. The more compact the soil and the greater the depth of the ground water, the purer is the supply likely to be. In compact sandy gravel, loam, chalk, etc., a pure water may be usually obtained from a properly constructed and situated well if the water level is six feet or more from the surface. In selecting a site for a well the proximity to highly manured ground should be avoided. Cesspools and drains should be as distant as possible, and the well should be on the side of the higher ground-water level. If, for example, the ground water flows from east to west the well should be to the east of the possible sources of pollution, any polluting matter entering the soil will then flow away from the well, whereas if the well is on the west the filth will flow towards the well and possibly contaminate the water. Water entering a well at a less depth than six or eight feet cannot be depended upon as being efficiently filtered or purified, therefore this upper portion should be constructed of impervious material, brickwork set in cement, glazed earthenware tubes, or of iron cylinders, and the top should be so finished off that no surface water can possibly gain access. For this purpose it is desirable that the top of the well should be brought up six to twelve inches above the ground surface and covered with a proper flagstone or iron cover. When the soil is gravelly an Abyssinian tube well may be used. This consists merely of a perforated and pointed steel tube driven into the ground until the point is well below the ground-water level. A pump is then fixed to the projecting tube. If care be taken to puddle well round the upper portion of the tube and to fix it firmly, so that no surface water can travel down the loose soil surrounding it, these tube wells are both economical and satisfactory.

In shallow wells, otherwise properly constructed, polluting matter often enters along the track of the pump pipe. This is, generally, simply put through a hole in the brickwork. The constant shaking loosens the earth along the track of the pipe, and surface water following this line of least resistance trickles into the well. The pipe should either enter the well above the ground surface or have a collar upon it, the collar being embedded in cement between the two layers of brick enclosing the well.

Springs are not generally utilised for large public supplies, as the flow is in most cases too variable, falling off seriously in the late summer just when the demand for water is greatest. They are largely used, however, in rural districts, and if properly protected are, in most cases, very satisfactory sources of supply. The water usually has travelled a considerable distance, percolating horizontally through the subsoil, and has become thoroughly purified and filtered.

The ground around a well or spring should be protected, no manurial matter of any kind being placed thereon, and cattle should not be allowed to gain access to the water, or to trample on the ground around. This protective area need not be more than a few yards in diameter, in the case of a well from which only a small quantity of water is drawn, as when

only one or two houses are supplied, but it is useless expecting to obtain a really good water from a well placed in a garden highly manured around or over the surface of the well, yet wells of this character abound.

Water from rivers or streams is very variable in character, consisting usually of an admixture of surface, spring, and subsoil water. During heavy rains the surface water may carry with it impurities from the soil, at other times animals may defile it, drainage from houses or farmyards may enter it, sewage or sewage effluents or manufacturing refuse may be discharged into it. Vegetable growths occurring in the stream or on the banks decay and give up organic matter to the water. For these reasons the majority of streams are so polluted as not to be available as sources of supply. In hilly and mountainous districts where there is little vegetation and few habitations the streams may yield a wholesome water. Where the flow of water in a river is enormous compared with the volume of polluting matter entering, and the water is impounded in reservoirs for subsidence, and as a reserve to avoid having to take water from the river when rendered turbid by floods, and the clarified water is finally submitted to most careful filtration, a good potable water may possibly be the result. London is supplied in this way, and the evidence of experience is apparently satisfactory. It is quite probable, however, that an impure river water may give rise to a high mortality rate from typhoid fever without causing epidemics. Thus in Newark, Notts, typhoid fever was constantly prevalent when the town was supplied with water from the river Trent. In 1893 a fresh supply was obtained from deep wells in the new red sandstone. The fever almost immediately subsided, and has since only been about one-tenth the previous average. The excessive prevalence of enteric fever in the towns in the Tees valley supplied with water from that river was attributed by Dr. Barry, an inspector of the Local Government Board, after a thorough investigation, to the polluted condition of the river water. There is no doubt that a polluted river water during its flow tends to become purified. This so-called "self-purification" is due to the action of air, light, vegetable and animal growths, sedimentation, and possibly other causes, but it is probable that no river in the British Isles is sufficiently long to effect complete purification.

Probably the purest of all waters is derived from deep wells, wells supplying water from a porous stratum lying beneath an impervious formation. Such water has generally travelled a long distance from the outcrop to the point from which it is drawn, and any organic matter originally contained in it has become completely oxidised, and filtration has been practically perfect, far more perfect than any artificial process. Immense stores of the purest water are contained in the chalk, sandstone, and oolites, and smaller quantities in the lias, greensands, Hastings and Thanet sands, and millstone grit. When the outcrop is at a much higher elevation than the ground surface at the site of the well the water may overflow. Such wells are usually called "artesian" from the province of Artois in France where such wells were first found. Such a well recently sunk at Bourn, Lincolnshire, overflows at the rate of about five million gallons per day, and is probably the most prolific underground spring yet tapped in England. It is calculated that the chalk in the Lea valley will yield forty million gallons of water per day in the driest years, and the Kent chalk twenty-seven million gallons. Deep wells are usually bored, and the boring lined with screw-jointed steel tubes. Where water can be obtained from deep subterranean sources there are many advantages besides that of purity. Very little land is required, no compensation water has to be provided, filtration is unnecessary, and protection from pollution is easy. On the other hand, pumping has usually to be resorted to, but with modern machinery this is not a serious item. Deep wells are expensive to construct, hence they are rarely sunk for the supply of single houses or small communities, save in districts where no other source is available. In some cases the well tube is brought above the ground surface and a pump attached. The only possibility of pollution is from perforation of the tube, when subsoil water, which may be impure, may be drawn in. In other cases the tube terminates at the bottom of a well, and this well is liable to all the risks of the ordinary shallow well.

Contaminated waters are often used for lengthened periods without producing any appreciable effect upon the health of the consumers, but it seems quite probable that, in many instances, the system has become accustomed to its use. It is a common experience to find that persons who have previously used a pure water suffer from diarrhœa when they have to drink such a polluted water. In some rural districts where pond water is used the old inhabitants often prefer it to a purer supply, whilst other families moving into the district are so affected by the water that they remove again as early as possible. Sooner or later, however, specific pollution occurs and disease follows.

Diarrhœa is the most common symptom of water-poisoning, and may be caused by quite a variety of substances. Finely divided mineral matter may cause the irritation, or an excessive amount of magnesium sulphate or chloride, and possibly other salts. Water impregnated with even a small quantity of sulphuretted hydrogen, or with the gaseous emanations from drains, has often been charged with causing diarrhœa. Water impregnated with decaying vegetable matter or with peat may affect persons un-

accustomed to its use. Sewage-polluted water has frequently caused outbreaks, often with choleraic symptoms. In Lincoln, in 1891, an epidemic of diarrhœa occurred, limited to persons drinking water from the river Witham. The water was turbid and had an offensive smell when heated, and contained much organic matter. Many other outbreaks of a similar character are recorded. Epidemics of dysentery have been traced to the use of water polluted with decomposing animal matter.

Goitre is believed to be caused by some constituent found in the waters of certain districts, but the evidence with reference to the nature of the goitre-producing substance is very conflicting. The most convincing proof of goitre being produced by the drinking water is the evidence given before a French Commission, to the effect that in the village of Bozel, in 1848, there was a population of 1472, of whom 900 were goitrous, whilst at St. Bon, a neighbouring village with a different water-supply, there was not a single case. When the water from St. Bon was carried to Bozel goitre decreased so rapidly that in 1864 there were only 39 persons affected. Whatever the special constituent, it appears to be most common in the water from the magnesian limestone formation.

Plumbism is frequently caused by the continued use of waters containing lead. This metal is very rarely found as a constituent of natural waters, but many very soft waters act energetically on lead pipes, cisterns, etc. Certain hard waters containing little or no calcium carbonate have also an action upon lead. Water by contact with peat takes up a minute amount of acid, believed to be produced by a micro-organism growing on the peat, and such acid waters have a marked plumbo-solvent action. Plumbism has been very prevalent in the West Riding of Yorkshire. "The amount of lead dissolved depends upon the character of the water, the time during which it is in contact with the metal, the temperature, pressure, and possibly upon other factors of which we as yet know but little. The effects produced by the small amount of lead dissolved are rarely so serious as to cause death, or even the severe colic or paralysis characteristic of lead-poisoning (or even the blue line on the gums), and for this reason the injurious results of the long-continued use of waters so polluted are only gradually receiving recognition. Amongst the effects produced are a state of listlessness leading to melancholia, depression, and actual insanity, pallor and debility, constipation and indigestion, paralysis, colic, gout, kidney disease, blindness, etc. Still-births increase, and the children of lead-poisoned parents are rickety and ill-developed."[1] There is no doubt that persons vary in susceptibility to the action of this cumulative poison. One-tenth of a grain per gallon has been known to produce ill effects. An "active" water which has been allowed to stand in lead service-pipes during the night may contain 1½ grains or even more per gallon. By filtering an "active" water through beds of chalk or limestone the acid may be neutralised and its plumbo-solvent action more or less completely destroyed.

Zinc is occasionally found as a natural constituent of water. The soft waters which act upon lead will also dissolve zinc from the surface of galvanised iron. A water may take up as much as 3 or 4 grains of zinc carbonate per gallon by its action on a long galvanised service main. There is reason to believe that the use of such a water may cause obstinate constipation, especially in children, but beyond this no ill effect appears to follow even from its habitual use.

Enteric or typhoid fever is undoubtedly due in a large proportion of cases to the use of polluted drinking water. So large a number of outbreaks have been attributed to this cause that it is difficult to make a selection. These outbreaks may be divided into two classes: those in which specific pollution of the water was proved, and those in which this proof was not forthcoming. The latter class is by far the more common. As illustrating the first class the following instances may be cited, as they also show some of the various ways in which a water-supply may become polluted. In 1874 over 2000 cases of enteric fever occurred in Over Darwen (Lancs). It was found that a person suffering from this fever resided in a house, the drain from which was defective at a point where it crossed a leaking water-main. It was demonstrated by experiments that this sewage was sucked into the main freely and regularly. Within a short period of the specific pollution occurring about one-tenth of the population had contracted the disease. In 1882 an epidemic occurred in Bangor, 540 persons out of a population of 10,000 being attacked. It was proved that the stream supplying water to the town had been polluted by the discharges from an enteric fever patient. About this time the filter beds were disturbed by the bursting of a main, and a fortnight afterwards the outbreak commenced. The Caterham and Redhill epidemic in 1879 was traced to the defilement of a conduit by the stool of a workman who had contracted a mild form of the disease. "The outbreak took place simultaneously in Caterham and Redhill, exactly fourteen days after the water-supply had been befouled in this manner." In 1884 an outbreak occurred at Beverley (Yorks). The drainage from the East Riding County Lunatic Asylum, in which there had been cases of enteric fever, was discharged over a field adjoining the Water Company's works. Certain defects were found in the well, and

[1] *Water and Water Supplies*, p. 139. London: Retman, Limited.

there was no doubt the imperfectly purified sewage effluent had reached the well. Numberless cases of epidemic outbreaks have been traced to the use of water polluted by sewage not known to have been specifically infected. Several such outbreaks were due to the insuction of filth from closet pans flushed directly from the mains, and many to causes already illustrated. There is no doubt that the majority of the recorded small outbreaks are due to the use of polluted well water, and a careful study of these cases shows that in nearly every instance the pollution was obvious and gross. Where a well is properly constructed the risk of pollution is infinitesimal, and there are no authentic records of water from such a well causing disease. The Maidstone outbreak (1897), one of the most serious which has occurred in this country, was entirely attributable to the pollution of a spring, one of the several sources of supply to the town. This spring arose at the edge of some highly manured hop-fields, and must inevitably from time to time have become polluted. Specific infection could not be traced. In 1889 a serious outbreak of typhoid fever occurred at New Herrington (Durham), 278 out of a population of 3600 being attacked. The water supply was from a deep well, and the polluting matter, to which the outbreak was attributed, travelled from a farmyard three-quarters of a mile away through fissures in the rock and reached the well at a depth of forty-five feet. The reports of the Massachusetts Board of Health contain many records of epidemics due to the use of polluted river water, and many such epidemics have occurred in this country, notably in the Tees and Trent valleys. London, supplied chiefly from the Thames and the Lea, experiences no special prevalence of typhoid fever, although the rivers receive enormous volumes of imperfectly purified sewage effluents; this is attributable in part to the great dilution, but chiefly to the great care taken in filtering the water before distribution.

To account for the outbreaks of typhoid fever from the use of sewage-polluted water it has been assumed that the bacillus coli communis, found in all fæcal matter, is merely a degenerate non-virulent form of the typhoid bacillus, which may, under certain circumstances, again become virulent. There is no reliable evidence in proof of this assumption.

That cholera is very frequently a water-borne disease is undoubted. That such was the case was suspected in the 1849 epidemic, but in 1854 an outbreak in London furnished conclusive proof. This outbreak occurred round Golden Square, Westminster, and was entirely limited to households using water from what was known as the Broad Street pump. About seventy-eight hours before the general outbreak it was found that a child had suffered from a fatal attack of "diarrhœa," and the stools had been thrown down a drain which ran near the well. This well was so defective that it was easily demonstrated that the sewage entered it. The much more recent outbreak at Hamburg was investigated by Koch, and attributed entirely to the use of the imperfectly purified water from the Elbe. The neighbouring town of Altona remained free from the disease until an accidental breakdown occurred at the water filtration works, which allowed impure water to be supplied for a short time to the town. Immediately afterwards cholera broke out in Altona.

Entozoal disease may be contracted from drinking water containing the ova of such parasites. In this country there is evidence that the ova of Ascarides lumbricoides and Oxyuris may gain access to the system through the medium of water—in my experience generally from the use of polluted pond water. Other entozoa infecting man, and which may be introduced by impure water, are:—Bilharzia hæmatobia, Filaria sanguinis hominis, Dracunculus medinensis, Rhabdonema intestinale, Trichocephalus dispar, and possibly species of tænia.

The effect upon animals of drinking impure water appears to have received very little attention, but outbreaks of anthrax, hog cholera, and glanders have been attributed to this cause. Farmers whose cattle have to drink water from polluted streams often attribute outbreaks of disease thereto, but such outbreaks are rarely carefully investigated. Probably such investigations would show not only that impure waters are inimical to the general health of cattle, but also a danger to those who consume their milk and flesh. A pure water should certainly be insisted upon for all dairy purposes, as notwithstanding the most careful supervision such water may contaminate the milk.

Public water supplies should be of such a character that the water requires no further purification by the consumers. Water derived from carefully protected springs or from deep wells requires no treatment whatever, but surface waters usually require careful filtration. During certain seasons minute forms of vegetable life are apt to occur, and these, either by their growth or decay, may render the water unsightly or even unpalatable. River waters, or water from any source liable to sewage contamination, should be efficiently purified before distribution. By proper collection in large reservoirs sedimentation occurs, and the sunlight destroys many bacteria; finally, filtration through beds of sand will remove over 99 per cent of the remaining organisms. This filtration, to be efficient, must be conducted in a scientific manner. Koch, who has paid great attention to this subject, asserts—(1) That the real effective agent in removing micro-organisms from the water being filtered is the layer of

slimy organic matter which forms upon the surface of the sand. (2) That if this surface be removed by scraping, or its continuity affected in any way, as by the freezing of the surface, the number of bacteria which pass through the filtering material increases considerably; in fact, both cholera and typhoid germs may pass in sufficient numbers to cause an epidemic amongst those who use the imperfectly filtered water. (3) That water should not pass through the filters at a rate exceeding two million gallons per acre daily. (4) That after a filter bed has been scraped, water should be allowed to stand upon it for at least twenty-four hours, to allow of the slime depositing before filtration is commenced, and that the water which first passes through should not be allowed to reach the pure water reservoir. He also asserts that filtered water containing more than 100 germs, capable of development, in a cubic centimetre should not be considered as sufficiently pure for distribution.

Many inventors have patented processes for purifying water on a large scale without recourse to sand filtration, as this not only requires the exercise of great care, but necessitates the possession of considerable areas of land for the construction of filters. These machines may be useful under certain circumstances, but of the many which I have examined not one produced results which could be compared with efficient sand filtration.

Domestic supplies may be purified in various ways. Where the impurities to be removed are of mineral origin, fine sand, clay, etc., or vegetable growths occurring in an otherwise unimpeachable source of supply, there are many filters which are efficient. If, on the other hand, there is the slightest suspicion of sewage contamination, the only safe methods of purification are (*a*) boiling, (*b*) filtration through materials which will remove all bacteria. Boiling for a few minutes destroys all disease-producing organisms, and is therefore thoroughly reliable. The only filters which can be relied upon to remove all bacteria are Pasteur's and the Berkfeld candles. These are hollow cylinders of porcelain (Pasteur) or compressed kieselguhr (Berkfeld), and the water in passing through the walls of the filter leaves the organisms and other sedimentary matter, usually slimy in character, on the surface of the filter. The Pasteur is the more reliable, and being harder is more durable than the Berkfeld, but it filters much more slowly, and is practically useless unless the water can be put under pressure. By employing a large number of cylinders, a so-called battery, a slight pressure will suffice. With the Berkfeld the same result can be obtained with little or no pressure, but the pores being more open the action of the filter cannot be relied upon for as great a length of time. Moreover, when the cylinders are removed for cleansing, brushing and boiling in water, or roasting over a red fire, they are more liable to fracture. A number of forms of these filters are made for attaching to taps, pumps, cisterns, etc. All the ordinary forms of domestic filter are practically useless for removing bacteria, they are simply fine strainers, and may produce a bright and sparkling water containing even more bacteria than the original unfiltered water. Equally good results can be obtained from a filter costing but a few pence, and made by filling a flower-pot with layers of fine gravel and sand. Dr. Bond, Medical Officer of Health for the Gloucestershire Combined Districts, has recently introduced a "floating film" filter, which consists of a cage of fine wire surrounded by a film of porous material, which can be obtained at about one shilling each, and fresh mantles or films at 1d. to 2d. each. The rate of filtration can be controlled. They certainly remove all visible suspended matter better even than many high-priced filters, but they cannot be depended upon to remove bacteria however slow the filtration.

Very hard waters may often be softened economically, and during the process a certain amount of bacterial purification is effected. This hardness depends upon the presence of salts of calcium and magnesium, usually carbonates and sulphates. The carbonates being held in solution by carbonic acid are precipitated by boiling, which drives off the carbonic acid. The hardness so removed is called "temporary." The hardness due to the sulphates not being removable by boiling, is called "permanent." The softening process consists in the addition of lime water, or of soda ash in solution, to the water. The free alkali combines with the carbonic acid, and the calcium carbonate is thrown down ($CaCO_3,CO_2 + CaO = 2CaCO_3$). When soda ash is used the sulphates are also decomposed ($MgSO_4 + Na_2CO_3 = MgCO_3 + Na_2SO_4$), so that both the temporary and permanent hardness are reduced. The carbonates in separating carry down with them many of the suspended bacteria, hence not only is the water softened, but bacterially purified. Water which contains so much of the calcium and magnesium salts as to produce an insoluble curd with soap are called hard, whilst those which form little or no curd and lather freely are called soft. There is no doubt that a soft water is far more economical and better adapted for domestic purposes than a hard water. It economises soap, is better adapted for tea-making and general culinary purposes, does not fur boilers, and minimises labour in washing, but there is no evidence that it is more beneficial to health. The Royal Commission on Water-Supply went fully into the question of the effect of hard and soft water upon health, and concluded that "where the chief sanitary conditions prevail with tolerable uniformity, the

rate of mortality is practically uninfluenced by the softness or hardness of the water supplied to the different towns." The opinion that "soft water" increases the tendency to rickets and "hard water" to the production of renal calculi is quite unfounded. The late Sir B. Ward Richardson was strongly of opinion that hard waters produced dyspeptic symptoms in persons unaccustomed to their use, and it is probable that soft water has a tendency to produce diarrhœa in persons accustomed to the use of hard water. Both effects are probably very temporary, and may account for the derangements from which persons visiting health resorts frequently suffer.

There are many makers of water-softening apparatus, some of which are adapted for use in mansions, public institutions, etc. Probably the most generally useful is the one made by the Maignen "Filtre Rapide" Co., which softens and filters the water automatically.

From what has been said, it will be evident that a water which does not contain such an excess of saline matter as to cause it to be too hard, and is free from lead or from an excessive amount of iron or zinc, and which gives no indications of sewage matter, and of more than a trace of vegetable matter, is hygienically pure and fit for domestic purposes. Whether this is so or not can be fairly well determined by bacteriological and chemical examination, but no such examination can be depended upon to indicate danger of pollution. A source which may usually yield a pure water may, if not properly protected, suffer from occasional contamination, and is therefore unsafe. Quite recently I investigated an outbreak of typhoid fever which the Medical Officer of Health attributed to the use of water from a certain spring. A sample of the water was sent to an analyst, who certified that it was pure, and went out of his way to assert that it could not possibly have caused typhoid fever. Upon investigation I found that the spring water had recently been noticed to become turbid after rain (always suggestive of pollution), and the cause was traced to a sewer broken at the crown. During dry weather no sewage escaped, during heavy rains the sewer filled, and sewage matter escaped and polluted the spring. Further, the sewer was traced to an isolation hospital in which there had been a case of typhoid fever, and a sample of the water collected after a heavy rainfall was found upon analysis to be highly polluted. An examination of the *source of supply* is therefore even more important than an analysis, and the nature of the source should always be considered when giving an opinion. Where a source is obviously open to pollution an analysis is unnecessary; where there is no possible source of pollution near the analysis is of little importance, and might almost be confined to the examination for hardness and noxious metals. For sanitary purposes the chemical examination is generally confined to the estimation of the hardness, chlorides, nitrites, nitrates, lead, iron and zinc, and ammoniacal and organic matter. The *bacteriological examination* may include the examination of the number of bacteria per cubic centimetre capable of growing on nutrient jelly, and tests for the presence of the bacillus coli communis and bacillus enteritides sporogenes, organisms always found associated in sewage and manure. The physical properties, colour, odour, and turbidity should also be noted. Such examinations require to be performed in a fairly equipped laboratory, in fact they are best undertaken in laboratories established especially for the purpose. I, however, have devised a small cabinet containing all the requisites for a simple chemical analysis. It is made by Messrs. Burroughs Wellcome & Co. That the cabinet and process is fairly satisfactory may be inferred from the fact that it has been adopted by Government, and a special case devised for saddle or shoulder. In this method[1] the reagents are in the form of "soloids," which keep well and contain a definite quantity in each. All the above chemical determinations can be made, and with a knowledge of the source from which the water is supplied, in the great majority of cases a correct opinion upon the character of the water can be given. In the ordinary laboratory the examination is made in the following manner:—

Colour and Turbidity.—Fill a 100 c.c. cylinder, place on a white slab and look down the column of liquid. Note the appearance.

Odour.—Put about 100 c.c. into a boiling flask, warm to about 120° F. Insert the rubber stopper, shake vigorously, remove the stopper, and apply immediately to the nostrils.

Residue left on Evaporation.—Evaporate about 20 c.c. to dryness in a platinum or porcelain dish. Note character of residue, then ignite very gently, and note any change which occurs.

Nitrites.—Place about 50 c.c. of the water in a Nessler glass, add 1 c.c. of zinc iodide and starch solution, and 1 c.c. of dilute hydrochloric acid. Allow to stand for five minutes. A blue colour indicates nitrites.

Nitrates.—If nitrites are absent add a minute quantity of zinc dust and stir. A blue colour will develop more or less rapidly according to the amount of nitrates present. If the water contains very little nitrate, 50 or 100 c.c. must be taken for the next experiment; if much is present, 5 to 20 c.c. will suffice.

In a 250 c.c. tubulated flask, place 20 or 30 grams of granulated zinc, coat with copper in the usual way, wash well, then add 100 c.c. of distilled water and 20 c.c. of caustic soda solution. Distil, collecting 10 c.c. at a time until

[1] *A Simple Method of Water Analysis*, 3rd edition, 2s. 6d. J. & A. Churchill, London.

the distillate is free from ammonia. Now pour into the flask the measured quantity of water by means of a pipette; distil over 50 c.c. Add to the liquid in the flask 50 c.c. of pure distilled water, and distil a second 50 c.c. Nesslerise the distillates. The amount of ammonia obtained multiplied by ·823 gives the nitric nitrogen in the quantity of water used for the experiment, and must be calculated to parts per 100,000.

Chlorine.—Measure 100 c.c. of water into a white porcelain capsule, add 10 drops of solution of chromate of potassium, and run in standard silver nitrate solution from the burette until a red tint appears. Each c.c. of silver solution used represents one part of chlorine in 100,000 of water.

Total Hardness.—Measure 100 c.c. of water into a 250 c.c. stoppered bottle, and add standard soap solution from a burette, with constant agitation, until an abundant lather is produced. (Similar to that produced by 1 c.c. of the soap solution in 100 c.c. of distilled water.) The number of c.c. of soap solution used minus 1 gives the hardness per 100,000 parts.

Permanent Hardness.—Place 150 c.c. of the water in a boiling flask and boil gently for 15 or 20 minutes, adding a little distilled water from time to time to replace that lost by evaporation. Allow to cool, make up to 150 c.c., and filter off 100 c.c. The hardness in this is estimated as above, and represents the permanent hardness.

Temporary Hardness.—The total hardness less the permanent hardness gives the temporary hardness.

Lead and Iron.—Place 50 c.c. of the water in a Nessler cylinder, and add about 5 drops of the solution of calcium polysulphide. A brown tint indicates lead or iron. Add dilute HCl drop by drop until the liquid becomes turbid. If the brown colour disappears the impurity is due to iron; if the brown tint remains, lead is present. The amount may be very simply estimated by contrasting the tint produced with that in artificially prepared solutions containing known amounts of lead.

Zinc.—Place 50 c.c. of water in a Nessler cylinder, very faintly acidulate with HCl, and add 2 or 3 drops of solution of potassium ferrocyanide. Put in a *dark* place for half an hour. If a turbidity is produced zinc is present. (If iron be present the liquid will become blue; if copper, a red tint develops.)

Free Ammonia.—Place 250 c.c. in a boiling flask and distil over 50 c.c. Unless the water contains a great excess of ammonia the whole is found in this distillate. The amount of ammonia in this distillate is determined by the process of "Nesslerisation." This consists of adding to the distillate 2 c.c. of Nessler's solution (a solution of mercuric iodide in caustic soda or potash), and imitating the depth of colour produced by adding Nessler's solution to pure distilled water to which definite quantities of solution of ammonium chloride have been added. The ammonium chloride solution is made of such a strength that 1 c.c. corresponds to ·01 mg. of ammonia. The water remaining in the retort is used in the next experiment.

Albuminoid Ammonia.—Distil over another 50 c.c. and pour into the distilled water bottle. Add by means of a funnel 25 c.c. of alkaline solution of permanganate of potassium and distil off two 50 c.c. Nesslerise as before.

The amount of ammonia obtained in the last two experiments multiplied by ·4 gives the ammonia in 100,000 parts of water. If multiplied by 4 the result will be in part per million.

Oxygen Absorbed.—Use a solution of permanganate of potassium, each c.c. of which represents ·1 mg. of available oxygen. In a stoppered flask (thoroughly well cleansed with dilute acid to which a little permanganate has been added and afterwards rinsed) place 250 c.c. of the water to be examined together with 10 c.c. of dilute sulphuric acid (25 per cent), and 10 c.c. of the dilute permanganate. Keep at 80° F. for four hours. If the colour fades much add a second 10 c.c. of permanganate.

Prepare a dilute solution of sodium thiosulphate by diluting 1 c.c. of a strong solution to 100 c.c. with distilled water.

To the water which has stood for four hours add 2 c.c. of solution of starch and zinc iodide, and run in from a burette the dilute thiosulphate solution until the blue colour just disappears. Note the amount a required. Now add 10 c.c. of the dilute permanganate solution, and again titrate with the thiosulphate. The amount b now used is the equivalent of 1 milligram of oxygen. Then

$$\frac{b-a}{b} = \text{oxygen consumed by 250 c.c. of water.}$$

This multiplied by ·4 gives the oxygen absorbed in parts per 100·000; if multiplied by 4 the result is in parts per million.

Bacteriological Examination of Water.—1. *Number of Colonies per Cubic Centimetre.*—Make two gelatine plates (Petrie dishes) using ·5 and 1 c.c. respectively of the water to be examined. Incubate at 20° C. for four days. Examine the plates daily, and if liquefaction is setting in count the number of liquefying and non-liquefying colonies. Non-liquefying colonies resembling those of B. typhosus or B. coli may be counted and picked out for further experiments.

Jelly of uniform composition and reaction should always be used. All the colonies visible under a small hand lense should be counted.

2. *Examination for Bacillus Coli.*—Add 1 c.c. of the water to 10 c.c. of broth containing ·05 per cent of phenol. Incubate at 36° to 37° C.

for 24 hours. Should a uniform turbidity be produced examine in the hanging drop, and if the organism is a motile bacillus: (*a*) stain the flagella by van Ermengen's method; (*b*) make a shake gelatine culture for formation of gas bubbles; (*c*) inoculate a broth tube for indol reaction; (*d*) inoculate litmus milk for production of acid and coagulation of casein.

3. *Examination for the Spores of Bacillus Enteritidis Sporogenes.*—Pass 250 or 500 c.c. of the water through a very small Pasteur filter, and brush the residue into 10 c.c. of sterile milk. Place the tube in a water bath, and keep at 80° C. for 15 minutes. Pour about 2 grams of melted vaseline over the surface of the milk and cool. The vaseline effectually excludes the air, and if the milk be now placed in an incubator for 24 to 48 hours, any spores of the bacillus present will germinate, and the presence of the organism be revealed by the coagulation of the casein in torn irregular masses, having the faintest possible pink tint and the evolution of gas which forces up the vaseline plug. Upon removal of this plug the gas will be found to be inflammable and the liquid has a strong odour of butyric acid.

Interpretation of Analytical Results.—A filtered water which contains more than 100 organisms per cubic centimetre capable of growing on nutrient jelly is not efficiently purified, but it must be remembered that this enumeration is of little value unless the water is examined immediately after it leaves the filter beds. The harmless bacteria found in water multiply rapidly, hence a water may contain 1000 or more organisms per c.c. and yet be perfectly wholesome. If, however, both the B. coli communis and the B. enteritidis sporogenes are found to be present there can be no doubt that the water is polluted and unfit for domestic purposes.

A water with a greenish or yellowish tint is generally contaminated, but it may be merely with a trace of vegetable matter. A brownish tint usually indicates "peat," and this, unless very excessive in amount, is quite harmless. A pure water should be practically colourless and free from any appearance of turbidity when viewed in a column six inches in depth. A peaty water may have a faint odour, but an odour from any other cause renders the water suspicious.

All waters contain chlorides, hence the chlorine determination has no great value. No inference can be drawn as to sewage contamination from the amount of chlorides present unless the analyst knows how much is contained in water of known purity from the same source. Sewage contains chloride of sodium (found in the urine), but considerable pollution must take place before it is recognisable by its effect upon the chlorides. Near the sea or tidal rivers the subsoil may be infiltrated with salt, and waters therefrom be rendered brackish. If the salt, however, is not in such quantity as to be appreciable to the taste it is harmless.

Sewage undergoing decomposition usually contains nitrites, whereas pure waters, save under very exceptional circumstances, are free therefrom; hence the presence of nitrites always indicates the necessity for careful inquiry as to their source. Nitrates, on the other hand, are the final products of the decomposition of sewage or manure by the micro-organisms in the soil, and indicate the nature of the ground from which the water is collected. Some analysts ignore the presence or absence of nitrates, others lay great stress on the amount present. Nitrates are perfectly harmless in themselves, but they indicate that the subsoil is more or less highly manured, and therefore that pollution may occur unless the well or spring is sufficiently far from the polluting matter, and the soil is sufficiently compact and "live" to be depended upon to effect complete oxidation and filtration. When the water contains an abundance of nitrates and decided traces of organic matter it should be unhesitatingly condemned. The nitrates are usually expressed in terms of nitric nitrogen, 1 part of which corresponds to 4½ parts of nitric acid, or about 6 parts of such nitrates as usually exist in drinking waters. No possible standard can be fixed, but even in agricultural districts, where the subsoil is porous and the soil highly manured, the presence of much over 1 grain of nitric nitrogen per gallon should render the water suspicious.

The hardness of water is usually expressed in degrees, each degree corresponding to the soap-destroying power of one grain of calcium carbonate in one gallon of water. When the soap-destroying power is under 10° the water may be considered soft, or moderately soft; from 10° to 15° it is moderately hard; 15° to 20°, hard; and over 20°, very hard or excessively hard. An excessively hard water is not suitable for most domestic purposes. A water from which much of the hardness can be removed by boiling is preferable to a permanently hard water.

The organic matter in a water cannot be accurately determined, and such a determination is rarely attempted. Other estimations are made which serve as indices of the amount of organic matter present, and at the same time give some indications of its character. Sewage and manurial matters yield much free ammonia on distillation, and after this has been distilled off a further quantity of ammonia can be obtained by the action of a strongly alkaline solution of potassium permanganate on the albuminous matters. This is called "organic" or "albuminoid" ammonia. Further, these impurities absorb considerable amounts of oxygen from acid solution of potassium permanganate solution, called "oxygen absorbed."

Hence the amounts of "free" and "albuminoid" ammonia and of "oxygen absorbed" are considered together in forming an opinion as to the purity of a water. Vegetable matter usually yields very little free ammonia, but may yield as much "organic" ammonia as animal matter; it, however, absorbs oxygen less freely.

Nearly all waters contain a trace of free ammonia, especially rain water, and occasionally a water which has stood for some time in contact with a metal will yield ammonia due to the reduction of nitrates by the metal—zinc, iron, or lead. In such cases traces of the metal can be detected in the water. In waters other than rain waters, or waters containing traces of the above-mentioned metals, the presence of more than ·05 parts of ammonia in one million parts of water should give rise to suspicion. If the "organic" ammonia exceeds ·05 parts per million, and yet is less than the free ammonia, pollution with sewage or manurial matter is indicated. If the "organic" ammonia is under ·10 per million, and the "free" ammonia under ·05, the organic matter is probably of vegetable origin.

The following table by Messrs. Tidy and Frankland was devised for interpreting the results obtained in the estimation of the oxygen absorbed:—

	Amount of oxygen absorbed by one million parts of water.	
	Upland surface water.	Water from other sources.
Water of great organic purity	Not more than 1·0	Not more than 0·5
,, medium purity	,, ,, 3·0	,, ,, 1·5
,, doubtful purity	,, ,, 4·0	,, ,, 2·0
Impure water	More than 4·0	,, ,, 2·0

In interpreting chemical results it must be remembered that the most delicate analysis will not reveal the presence of minute quantities of sewage, hence even waters of great organic purity may actually be contaminated, and a careful bacteriological examination may reveal the presence of organisms derived from sewage or manure.

In taking samples of water for examination great care should be exercised, as the introduction of a very little organic matter from the vegetation around, from dirty vessels, or from a cork, may mislead the analyst. A white glass-stoppered bottle holding about four pints is best. It should be thoroughly cleaned with oil of vitriol, and rinsed out repeatedly with good water. The bottle should then be completely filled with the water to be examined, a little poured away, and the stopper inserted. If the water is collected from a tap, the tap should be turned full on and the water allowed to run to waste for a few minutes before filling. If the sample has to be taken from a cistern, pond, or stream, then, if possible, the stopper should be inserted and the bottle placed well under the surface of the water, and in a few seconds the stopper removed and the bottle allowed to fill. The stopper is best secured by a covering of clean linen, or by an india-rubber cap. Whoever interprets the results of the analysis should be fully acquainted with the source of the water, as the interpretation is not a mere chemical problem.

Under the Public Health Acts it is the duty of all sanitary authorities to see that every house in their districts has an available supply of wholesome water, within a reasonable distance, sufficient for all domestic requirements. But this supply can only be enforced where it can be obtained at a reasonable cost, and this cost is defined as £8:13:4 per house (or by special permission of the Local Government Board, £13). Unfortunately in the great majority of cases a proper well cannot be constructed for this sum, hence many localities continue to be badly supplied with water. The terms "reasonable distance" and "available supply" are variously interpreted in different districts, hence there is great lack of uniformity in the administration of the Acts so far as they apply to water supplies. There are even differences of opinion as to what constitutes a "wholesome water." Where there are considerable congregations of houses it becomes the duty of the sanitary authority to provide a public supply. Unfortunately, again, this duty is too frequently shirked on account of the expense. Where any source of water-supply is so contaminated as to be injurious to health the sanitary authority may cause the source to be closed. This section of the Public Health Act is, however, of little use, since it is difficult to prove that a water is actually injurious to health. If the word "dangerous" were substituted for "injurious" the section would be of much greater value. The law generally relating to water supplies requires amending.

Water-gas. *See* TOXICOLOGY (*Gaseous Poisons, Carbon Monoxide, Water-gas*).

Water-hammer Pulse.—A pulse in which the stroke is exaggerated, but it lasts a very short time and collapses very quickly and very completely; it is characteristic of aortic incompetence; Corrigan's pulse. *See* HEART, MYOCARDIUM AND ENDOCARDIUM (*Physical Signs of Different Forms of Heart Disease, Aortic Incompetence*).

Waterhouse-Forbes Steriliser.—A method of sterilising water by means of boiling; it has the advantage of using little

fuel, of rapidly cooling the water, and of preventing loss of the gaseous constituents and so preventing insipidity.

Water Itch.—A skin disease of the tropics; "sore feet of coolies" or "pani ghao." *See* SKIN DISEASES OF THE TROPICS (*Pani Ghao*).

Waters. *See* BALNEOLOGY; MINERAL WATERS; LABOUR, STAGES AND DURATION (*Phenomena of First Stage, Rupture of the Membranes, Escape of Liquor Amnii*).

Water, Toast. *See* INVALID FEEDING (*Toast Water*).

Watery Vapour. *See* CLIMATE (*Individual Factors, Watery Vapour*).

Watt's Asphyxiator.—A means of testing the drainage system of a building; touch paper is used to produce the smoke which serves to indicate defects, and a draught is supplied by a fan.

Wave. *See* PULSE (*Sphygmographic Tracings*); PHYSIOLOGY, CIRCULATION (*Arterial Pulse*); etc.

Wax. *See* CERA (*Alba and Flava*); EAR, EXTERNAL, DISEASES OF (*Meatus, Excess of Cerumen*).

Waxy Degeneration.—Amyloid degeneration. *See* LARDACEOUS DEGENERATION.

Weaning.—Ablactation; the cessation of maternal nursing. *See* INFANT FEEDING (*Weaning*); PUERPERIUM, PHYSIOLOGY (*Lactation*); PUERPERIUM, PATHOLOGY (*Conditions Interfering with Suckling*).

Weather. *See* CLIMATE; METEOROLOGY; SEA VOYAGES; THERAPEUTICS, HEALTH RESORTS; etc.

Webbed Fingers and Toes.—Syndactyly. *See* DEFORMITIES (*Fingers, Toes*).

Weber's Glands. *See* SALIVARY GLANDS, DISORDERS OF (*Anatomy, Accessory Glands*).

Weber's Organ.—The sinus pocularis or uterus masculinus of the prostatic portion of the male urethra.

Weber's Syndrome or Symptom. — Paralysis of the oculo-motor nerve on one side and of the limbs and facial and hypoglossal nerves of the opposite side; it is due to a lesion of the upper region of the pons.

Weber's Test. *See* EAR, EXAMINATION OF (*Hearing, Tuning-Fork Tests*); EAR, MIDDLE, CHRONIC NON-SUPPURATIVE DISEASE (*Tuning-Fork Tests*).

Webs of the Larynx. *See* LARYNX, ACUTE AND CHRONIC INFLAMMATIONS (*Congenital Glottic Stenosis or Webs of the Larynx*).

Webster's Process.—The electrolysis method of the disposal of sewage.

"Weed." — Milk fever or mastitis. *See* PUERPERIUM, PATHOLOGY (*Affections of the Mamma, Mastitis*).

Weeks' Bacillus. — A short slender bacillus found in muco-purulent ophthalmia. *See* CONJUNCTIVA, DISEASES OF (*Acute Ophthalmia*).

Weeping. — Exuding or discharging watery fluid; *e.g.* weeping sores, weeping eczema, etc. *See* ECZEMA (*Lesions*).

Wegner's Disease.—Syphilitic osteochondritis in infants; Wegner's Sign of fœtal syphilis consists in the presence of an irregular yellow line crossing a long bone at the junction of the epiphysis and the diaphysis and due to the changes caused by syphilitic osteochondritis. *See* PREGNANCY, INTRA-UTERINE DISEASES (*Fœtal Syphilis*).

Weichselbaum's Bacillus.—The diplococcus intracellularis. *See* MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*History*).

Weidel's Reaction. — A test for xanthin bodies; substances containing these bodies, if treated by chlorine water containing nitric acid and then exposed to ammonia vapours, give a red or violet coloration.

Weigert's Stain.—A method of staining fibrin by the use of gentian violet, iodine solution, aniline oil and xylol. *See* POST-MORTEM METHODS (*Bacteriological Investigations, Stains*).

Weighing of Infants. *See* NURSERY HYGIENE (*Weighing Machines and Charts*).

Weights. *See* METRIC SYSTEM; PRESCRIBING; *see also* CHILDREN, DEVELOPMENT OF (*Growth in Weight*); INDIGESTION (*Loss of Weight in Dyspepsia*); LIFE INSURANCE (*Height and Weight*); LUNG, TUBERCULOSIS OF (*Symptoms, Weight*); NEW-BORN INFANT (*Physiological Peculiarities*); STOMACH AND DUODENUM, DISEASES OF (*General Symptomatology*).

Weilbach. *See* BALNEOLOGY (*Germany, Sulphur Waters*).

Weil's Disease. *See* LIVER, DISEASES OF (*Weil's Disease or Infective Jaundice*).

Weil's Syndrome. — Hyperæsthesia affecting the nerve trunks, muscles and bones of one side of the body; it is sometimes met with in tuberculosis of the lungs.

Weir Mitchell's Disease. *See* NEURASTHENIA.

Weir Mitchell's Method. — The treatment of neurasthenia and hysteria by isolation, rest, systematic feeding, passive exercises, and massage. *See* NEURASTHENIA (*Treatment*); HEART, MYOCARDIUM AND ENDOCARDIUM (*Treatment, Rest and Exercise*).

Weismann's Theory.—The theory of the continuity of the germ plasm in heredity; a deduction follows that acquired characters cannot be transmitted. *See* HEREDITY.

Weissenburg. *See* BALNEOLOGY (*Switzerland, Calcareous Waters*).

Weiss's Reflex.—A reflex observable in cases of myopia.

Weiss's Sign. — The occurrence, in tetany, hysteria, neurasthenia, and other diseases, of contraction of the facial muscles on light percussion.

Weitbrecht's Cartilage.—The occasional interarticular cartilage of the acromioclavicular joint.

Weitbrecht's Cord. — The orbicular ligament of the elbow joint.

Welcker's Angle. — An angle (or angles) used in craniometry, *e.g.* the nasal angle and the naso-basal angle.

Wells' Facies or Sign.—The peculiar physiognomy of a patient suffering from ovarian disease; named after Sir Spencer Wells.

Welsbach Burner. — The incandescent burner for coal gas in lighting.

Wemyss Bay. *See* THERAPEUTICS, HEALTH RESORTS (*Scotland*).

Werlhof's Disease. — Purpura hæmorrhagica. *See* PURPURA (*Werlhof's*).

Wernicke's Centre.—The auditory word centre in the first temporo-sphenoidal convolution of the cerebrum. *See* BRAIN, PHYSIOLOGY (*Functions of Cerebral Cortex, Word-Hearing Centre*).

Wernicke's Disease. — Polioencephalitis acuta hæmorrhagica.

Wernicke's Fissure.—The exoccipital fissure of the cerebrum.

Wernicke's Reaction. — Hemiopic inaction of the pupil. *See* EYE, CLINICAL EXAMINATION OF (*Anterior Chamber, Hemianopia, Wernicke's Sign*); PUPIL (*Nervous Mechanism*); VISION, FIELD OF (*Hemianopia*).

West Indies. *See* THERAPEUTICS, HEALTH RESORTS.

Weston-Super-Mare. *See* THERAPEUTICS, HEALTH RESORTS (*English*).

Westphal's Nucleus. — The small grey nucleus supposed to supply the sphincter pupillæ muscle; it lies below the aqueduct of Sylvius in the brain.

Westphal's Paradoxical Contraction.—A peculiar form of contraction of a muscle met with in paralysis agitans, alcoholism, tabes dorsalis, etc.; it consists in the fact that the muscle contracts when its ends are passively approximated, thus the tibialis anticus contracts when the foot is passively flexed.

Westphal's Sign.—The absence of the patellar reflex in tabes dorsalis, neuritis, and some other diseases of the nervous system. *See* TABES DORSALIS (*Symptomatology, Reflexes*).

West Virginia. *See* BALNEOLOGY (*America and Canada, Sulphur Waters*).

Wet-Nursing. *See* INFANT FEEDING (*Wet-Nursing*); NEW-BORN INFANT (*Feeding, Methods*).

Wet Pack. *See* HYDROPATHY (*Packs*); PUERPERIUM, PATHOLOGY OF (*Septicæmia, Treatment*).

Wetzel's Test.—A test for the presence of carbon monoxide in blood; water and tannic acid solution are added to the suspected blood, which becomes carmine red if carbon monoxide is present.

Weyl's Test.—If creatinin be present in urine, the addition of a few drops of a solution of sodium nitro-prusside causes a ruby-red colour changing into green and blue on the addition of a solution of sodium hydrate; also three tests for the detection of nitric acid in urine are named Weyl's Tests A, B, and C.

Weymouth. *See* THERAPEUPICS, HEALTH RESORTS (*English*).

Wharton's Duct.—The excretory duct of the submaxillary salivary gland. *See* SALIVARY GLANDS, DISORDERS OF (*Anatomy*); MOUTH, DISEASES OF (*Floor of the Mouth, Ranula*).

Wharton's Jelly.—The mucoid tissue of the umbilical cord of the fœtus. *See* FŒTUS AND OVUM, DEVELOPMENT (*Umbilical Cord*).

Wheals. *See* URTICARIA (*Symptoms*); PRURITUS (*Consequences of the Pruritic State*).

Wheelhouse's Operation. — A form of external urethrotomy for stricture. *See* URETHRA, DISEASES OF (*Stricture, Treatment, External Urethrotomy*).

Wheezing. *See* ASTHMA (*Symptoms*); BRONCHI, BRONCHITIS (*Physical Signs*).

Whey. *See* ATROPHY, INFANTILE (*Treatment*); INFANT FEEDING (*Milk Preparations,*

Whey); INVALID FEEDING (*Whey, Lemon, Wine, and Cream*); THERAPEUTICS, HEALTH RESORTS (*Germany and Austria, Meran Whey Cure*).

Whipworm.—The trichocephalus dispar. *See* PARASITES (*Nematodes, Trichotrachelidæ*).

Whiskey. *See* ALCOHOL (*Spirits*).

Whispering Pectoriloquy. *See* LUNG, TUBERCULOSIS OF (*Physical Signs, Vomica Formation*).

White Arsenic. *See* TOXICOLOGY (*Arsenic*).

White Bread. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Cereals*).

White Commissure. *See* SPINAL CORD, MEDICAL (*Anatomy*).

White Corpuscles.—Leucocytes. *See* BLOOD (*Corpuscles*).

White Disease. — Leucodermia. *See* SKIN, PIGMENTARY AFFECTIONS OF THE (*Leucodermia or Vitilego*).

White Flux. *See* SPRUE (*Synonyms*).

Whitehead's Gag. *See* PALATE (*Cleft, Operation, Instruments*).

Whitehead's Operation. *See* RECTUM, DISEASES OF (*Hæmorrhoids, Internal, Treatment*); TONGUE (*Carcinoma, Operative Treatment*).

White Lead. *See* LEAD (*Plumbi Carbonas*).

White Leg.—Phlegmasia alba dolens. *See* PUERPERIUM, PATHOLOGY (*Phlegmasia Alba Dolens*); TYPHUS FEVER (*Complications and Sequelæ, Venous Thrombosis*).

Whitelow. *See* FINGERS (*Inflammatory Affections*); MORVAN'S DISEASE; NAILS, AFFECTIONS OF THE (*Whitlows*); SYRINGOMYELIA (*Symptoms, Painless Whitlows*).

White Precipitate. — Hydrargyrum ammoniatum. *See* MERCURY.

White's Disease.—Keratosis follicularis. *See* ICHTHYOSIS (*Simplex, Symptoms*).

White Swelling. — Tumor albus or tuberculous arthritis.

"Whites."—Leucorrhœa (*q.v.*).

Whooping-Cough.

HISTORICAL NOTE 526
DEFINITION 527
MORBID ANATOMY 527
SYMPTOMS 528
COMPLICATIONS AND SEQUELÆ . . . 529
TREATMENT 530
Curative 530
Hygienic 531
Dietetic 531
Local Medication 531

See also ALCOHOL (*Indications*); ATHETOSIS (*Causation*); AUDITORY NERVE AND LABYRINTH (*Hæmorrhage into the Labyrinth*); BRAIN, INFLAMMATIONS (*Acute Encephalitis, Etiology*); BRONCHI, BRONCHITIS (*Acute, Etiology*); CONVULSIONS, INFANTILE (*Prognosis*); COUGH (*Clinical Varieties*); DISINFECTION (*Incubation Periods*); EPIDEMIOLOGY; INFECTION (*Quarantine Times*); LEUCOCYTOSIS (*Lymphocytosis*); LUNG, TUBERCULOSIS OF (*Etiology*); MENINGITIS, TUBERCULOUS (*Etiology*); MENTAL DEFICIENCY (*Postfebrile*); NOSE, POST-NASAL ADENOID GROWTHS (*Etiology*).

HISTORICAL NOTE.—Whooping-cough has been a prevalent disease through many centuries. Von Rosenstein, who wrote in 1771, states that it began in France in 1414, having been imported from Africa or the East Indies.

In the year 1580 it is reported that over 9000 children died in Rome from this disease alone. The earliest distinctive accounts of it were by Baillou in 1600 and Schenck in 1650. Prior to this period it does not appear to have been distinctly differentiated from such diseases as bronchitis or influenza. In the eighteenth century it is reported to have been generally epidemic over Europe, and we find the literature of the subject embraces the writings of many distinguished physicians at this time, more especially in England, Sweden, and Germany. Among the more important monographs were those of Hoffman, von Rosenstein, Stoll, Butler, Willis, and Biermer, the latter having written an account of all the epidemics during the century. It had all along been recognised as a contagious disease, and the general consensus of opinion tended to show that it was due to disorder of the digestive system, reflecting an irritation on the respiratory organs. During the nineteenth century the literature of the disease accumulated largely, especially in England, France, and Germany, and we find interesting treatises by such men as Trousseau, Barthez and Rilliet, Copland, Webster, Jahn, Kruckenberg, and many others. A mere epitome of the views of these and other well-known physicians during the last four centuries would occupy an extended space. Even at this time the older and more antiquated views of the nature and origin of the disease held their ground, with other additions to the many theories then prevalent. While the contagious nature of the complaint was fully recognised, no definite pathological facts had been adduced to form any foundation for the mass of theoretical reasoning. The theory which found most favour, especially among English physicians, was the neurotic one, whereas many of the

German writers held to the view that it was the result of primary affection of the bronchial lymph nodes producing pressure on the branches of the vagus, and thereby exciting reflex irritation. The French school held strongly to the theory that it was essentially a catarrhal affection of the respiratory tubes, complicated in severe cases by irritation of the central nervous system. Oppolzer considered the irritative cough was excited by foul secretions in the pulmonary alveoli. Letzerich was one of the first who made a step in advance of this, and advocated the view that the morbid catarrhal secretions contained inhaled spores which favoured decomposition and caused increased irritation. Finally, by a more or less general consensus of opinion the disease was classed among the zymotic group, and believed to be caused by a definite infection which localised itself in the first place in the air passages. It is interesting to note how through the theoretical groping of centuries the true cause of the disease was gradually approached, and is now being worked out on the lines of bacteriological investigation as applicable to the whole group of these diseases.

Definition.—It may be defined as a contagious and infectious disease, the leading symptom of which is a spasmodic or convulsive cough, associated with a catarrhal condition of the mucous membrane of the respiratory tract, principally of the nasal, faucial, laryngeal, and tracheal surfaces, as well as the larger bronchi, but which may also extend to the smaller bronchial ramifications. The cough is essentially paroxysmal in character, and recurs at intervals more or less frequent, the patient being free from cough at other times. The cough when fully developed is highly characteristic, and consists of a rapid succession of expiratory efforts accompanied by spasm and narrowing, or it may be closure of the glottis, which is followed by relaxation of the spasm, the inspired air rushing in with a whooping sound. The cough is ended by expectoration of tenacious mucus, often accompanied by vomiting. The whooping sound is loud and distinct in proportion to the severity of the spasmodic effort, not so marked or sometimes absent when the cough is less severe and forcible. There is a liability to various complications to be afterwards referred to. No more concise or true definition of the disease has been given than by Cullen, who summarises in a few words its leading characteristics, "Morbus contagiosus, tussis convulsiva, strangulans, cum inspiratione sonora, iterata, sæpe vomitus."

Whooping-cough is essentially an epidemic disease, rarely occurring sporadically. No conditions, telluric, atmospheric, or seasonal, are known in any way to influence its spread, nor do we know whether the contagium has any habitat outside the body. It occurs over all quarters of the globe quite independent of climate, which seems to have no influence either in its spread or in regulating its inception.

Season seems to have little or no influence in initiating or diffusing the disease, although in winter the epidemics are almost invariably attended by a larger mortality. This may probably be accounted for by the fact that hygienic conditions are less favourable at this season, the patients being unable to get out to the open air as frequently as in spring and summer. *Sex* as a predisposing cause seems to have a certain influence, girls being more susceptible than boys. *Age* has a powerful effect in causation. It is essentially a disease of childhood, although adults are not exempt. Strange to say, sucking infants seem to be less liable to it than older children. Statistics show that the period of greatest susceptibility is from two to five years. Cases have been met with where the fœtus has contracted the disease in utero from the mother, the infants showing all the characteristic symptoms a few hours after birth. In the case of adults as of children, women are more liable to infection than men, pregnant women more particularly so. The relation of this disease to other epidemics is interesting. It is well known and universally recognised that whooping-cough epidemics frequently precede or succeed outbreaks of the exanthemata, specially of measles and chicken-pox. The disease may also coexist with any of these diseases, particularly measles. In such cases the complication is often of great gravity, and predisposes to a fatal result.

Mortality.—Whooping-cough is one of the most fatal diseases of early life, particularly during the period of infancy and until the third year. After this the mortality gradually diminishes with advancing age. The mortality tables in England over a series of years show an average of 2 per cent from this disease, and the Registrar-General brings out a similar average for the last ten years in the eight principal towns of Scotland. The mortality in females always exceeds that in males. Previously healthy children of naturally sound constitution show a much smaller mortality than those of delicate constitution, or such as have been debilitated by previous disease. The fatality in the simple uncomplicated disease is small, complications generally being the cause of death.

Morbid Anatomy and Pathology.—Uncomplicated whooping-cough is rarely a fatal disease. In those cases which have been attended with a fatal result little has been found except a catarrhal condition, more or less marked, of the upper air passages, particularly of the larynx, on the laryngeal surface of the epiglottis, and on the intra-arytenoid mucous membrane.

The bronchial glands are usually in a hyperplastic condition, and there may be passive congestion of some of the internal organs, more especially of the brain. The lungs show patches of emphysema here and there.

The disease being of a zymotic nature caused by a specific materies morbi, no efforts have been spared by pathologists in recent years to detect the organism or organisms which cause the disease. It cannot be said that the question is definitely settled, although various organisms have been found in the catarrhal secretions of the respiratory surfaces. Letzerich, Poulet, Burger, and Deuchlar have all found different micro-organisms, but failed to identify any with the disease. Following these observers Afanassieff made numerous experiments on rabbits, which he inoculated with pure cultures of a bacillus which he found in the secretion, with the result that symptoms resembling pertussis followed. He found the same organism in children who had died of pertussis pneumonia, and believes this organism is the cause of the disease. Semtcheno repeated and confirmed Afanassieff's experiments. Koplik, one of the most recent investigators, considers he has discovered the specific organism, an anærobic and motile bacillus with rounded ends. The organism closely resembles that discovered by Afanassieff. Inoculation experiments in animals gave negative results in Koplik's investigations. Capelewski, however, who found the same organism, was attacked by severe catarrhal symptoms while conducting his experiments. More recently Behla has described an organism which he says is not a bacterium, but a protozoon, an amœba. Deichler and Kurloff have also expressed a like opinion. The organism increases by division and also by spores. The chief site of the parasite is in the larynx and trachea. It is interesting, further, to note that several of these observers discovered a toxin in the urine of children suffering from pertussis, and this was found to be identical with that obtained from pure cultures of Afanassieff's bacillus. As already stated, most of the fatal cases die from some complication which has involved one or more organs, and a reference to these may now be made.

Brain.—In addition to passive congestion of the cerebral vessels in rare cases meningeal hæmorrhages have been found, and sometimes effusion into the ventricles. The cerebral substance itself is never found to present any morbid appearances.

Abdominal Organs.—Apart from more or less congestion of the blood-vessels no changes have been found. Owing to the fact that the urine is frequently found to be saccharine the kidneys have been carefully examined by many pathologists, but without any evidence of gross lesion.

Heart.—During the paroxysmal fits of coughing the circulation is very much disturbed and the aeration of the blood temporarily interfered with, yet no heart lesion has been found either of the endocardium or muscular substance.

Conjunctivæ and Respiratory Mucous Surfaces.—The conjunctivæ are almost invariably found to be congested, and not infrequently hæmorrhage occurs from the vessels. The mucous surfaces of the nose, larynx, and bronchi are congested and somewhat swollen with excessive mucous secretion on the surface. *Emphysema*, as already noted, is rarely absent from the lungs. *Pulmonary collapse* is likewise of common occurrence, the excessive force of the expiratory effort emptying the alveoli of the contained air.

Catarrhal pneumonia is a frequent and serious complication, and the morbid appearances are indistinguishable from those found in this disease produced by other causes.

Tracheo-bronchial Adenopathy.—The constancy with which these glands are enlarged have led physicians of repute to promulgate the theory that this was the primary and essential cause of the disease, but the fact that the glands are enlarged from many other causes in cases where there is no spasmodic cough effectually disproves the allegation. One other morbid appearance deserves notice, and is met with in children after the dentitional period. I refer to ulceration of the frænum linguæ produced mechanically by the forcing of the tongue forwards on the lower incisor teeth.

Symptoms.—From the consideration given to the nature of the disease we conclude that it must be classed among the zymotic group, and that it is caused by a specific materies morbi, which, although not yet definitely determined, is of the nature of a micro-organism which attacks the respiratory mucous surface on which it grows, and produces a toxin which gives rise to more or less constitutional disturbance. This toxin does not cause much febrile movement, as do the toxins of the other zymotic diseases, for we rarely find the temperature above 100° or 101°, and that only during the first few weeks of the disease. The most marked effects of the toxin would seem to be determined in the central nervous system, and more especially that portion of it closely related to the respiratory apparatus in the lower motor neuron in the region of the nuclei of the vagus nerve. The upper motor neuron in the cerebral cortex would appear to be often gravely affected by the toxin of pertussis from the frequency with which convulsions occur in this disease. As in other zymotic diseases, the symptoms naturally arrange themselves with more or less regularity in certain stages.

The *incubation* period is not exactly known. It is probably variable, perhaps more so than in other diseases of the zymotic group, ranging from two or three days to as long as fourteen.

During this time the patient is apparently in average health.

The ordinary symptoms of the disease commence with the *catarrhal stage*, in which the child appears to suffer from a "cold," with slight cough, often of a short, hacking character, catarrhal discharge from the nostrils and sneezing, and congestion of the conjunctivæ with "watering of the eyes." If the temperature be taken at this stage it is found to be slightly elevated, perhaps not over 100°, for a few hours during the day. There is more or less anorexia, with slightly furred tongue, and a tendency to, or perhaps the actual occurrence of, nausea and vomiting. The child is generally irritable and fretful, and the sleep disturbed. The duration of this stage varies. In severe cases it is generally short, perhaps only two or three days; in moderately severe cases it may continue from one to two weeks, or even longer; and in some cases, and these are frequent in mild epidemics, the disease never passes on to the second or convulsive stage, and convalescence may be established in from a fortnight to three weeks. It is important to remember this in relation not only to diagnosis, but prophylaxis, as these cases spread the disease as readily as the more severe or fully developed cases, and should be isolated accordingly.

The *spasmodic* or *convulsive stage* is evidenced by the full development of the characteristic cough. During the catarrhal stage the cough is of a short and irritable character, occurring at irregular intervals, but cannot be said to possess any specially distinctive symptoms on which a positive diagnosis could be based. In this stage the cough is highly characteristic, and its true nature is readily apparent even to a casual observer. It occurs at intervals of tolerable regularity, and is always more frequent at night. The number of paroxysms vary infinitely according to the severity of the disease, from twelve or twenty to thirty or forty, or even more, in the twenty-four hours. The child is aware of its onset for a short time previously, and displays a certain restlessness and irritability. When the cough begins the child, if old enough, prepares to support itself by laying hold of anything that may be at hand, such as a chair or other article of furniture. The cough commences by a rapid succession of expiratory efforts, succeeded by a long inspiration, during which the whooping sound is produced by the sudden rush of inspired air through the partially relaxed glottis. This is followed by the expectoration of viscid tenacious mucus, or by vomiting, during which the stomach is emptied of its contents. During the cough the child's face becomes much congested, often livid, giving the appearance of impending suffocation. There may be bleeding from the mouth, nose, ears, or any of the mucous surfaces. After the fit the child is more or less exhausted, but often resumes its playfulness in a very short time. The action of the heart during the fit is much accelerated, and the pulse rapid. Auscultation of the lungs shows generally coarse, moist sounds in the larger bronchi, which usually disappear after the fit. The child's face during this stage of the disease is usually characteristic, presenting often a more or less congested appearance, especially of the conjunctivæ, with puffiness of the eyelids. In young infants the convulsive paroxysm of this stage of the disease presents a somewhat different picture; the convulsive efforts may be described as more of a tonic nature, in contradistinction to the clonic and interrupted spasm of the older child, the glottis seems to close rapidly, and the whole muscular respiratory apparatus, intrinsic and extrinsic, seems to be in a state of tonic contraction, during which the infant is veritably in a state of suffocation. On relaxation of the spasm there is seldom any whooping sound produced.

Complications and Sequelæ. — These are numerous and important, as it is the complicated disease that causes fatality.

Respiratory System.—Emphysema and *pulmonary collapse* go hand in hand, and are the direct result of the spasmodic effort during the second stage. In a few rare cases we meet with interstitial pulmonary emphysema, which can only be recognised during life when the air gets into the peribronchial tissues and passes to the anterior mediastinum, showing itself in the jugular fossa and at the root of the neck as subcutaneous emphysema.

Bronchitis of the larger tubes is present in most cases, and if the medium and smaller branches of the air tubes are unaffected it is of little moment. When the capillary bronchi and the alveoli are attacked we are face to face with one of the most fatal complications, pertussis pneumonia, caused by the pertussis bacillus and other organisms.

Circulatory System.—In severe cases the strain on the heart is great, and yet it is seldom the organ suffers in a serious degree. Not unfrequently dilatation of the right ventricle results, and the child suffers for a time from pulmonary regurgitation and other symptoms of inadequacy of the right heart. Dr. Cima, in investigating the condition of the blood in pertussis, finds leucocytosis present in all well-marked cases.

Hæmorrhages are frequent in almost any part of the body. Thus we have epistaxis, subconjunctival hæmorrhage, slight streaky hæmorrhages from the fauces tinging the expectorated mucus, hæmorrhages from the ears, a rare occurrence, cerebral hæmorrhages, almost invariably meningeal, producing different varieties of cerebral symptoms, convulsions, hemiplegia, monoplegia, or other forms of localised paralysis, or affection of the special senses. As a rule

convulsions or paralytic symptoms after pertussis pass off in a few weeks, the probability being that the hæmorrhage has been capillary and small in amount. Fatal cases have been recorded, proving the occurrence of hæmorrhages.

Digestive System.—Ulcer of frænum linguæ, hernia, prolapsus ani, gastric and intestinal catarrh. In cases where the child does not vomit freely, but swallows quantities of the mucus loaded with micro-organisms, an irritative catarrh is set up in the gastro-intestinal mucous tract, which often proves a serious complication.

Nervous System.—The effects produced in the brain by minute meningeal hæmorrhages have already been referred to, and also the less serious general effects of the toxin on the brain. These, of course, vary indefinitely according to constitutional tendencies of the child, delicate and hereditarily neurotic children suffering most severely.

Horvem, in an elaborate monograph on the subject of the paralyses of pertussis, treats more particularly of the rarer forms, and finds that in addition to the cerebral conditions which are most commonly found, cases of bulbar origin are met with. He notes also the occurrence of paraplegia, and cases resembling Friedreich's ataxia and disseminated sclerosis and peripheral neuritis (polyneuritis).

Apart from brain and lung lesions, pertussis in its immediate effects rarely leaves behind it any gross lesions of other organs. Perhaps the most important, because the most common, of the remote effects of this disease are manifested in general ill health and debility which often for many months affect the child, and this predisposes to one of the most serious and remote sequelæ of this disease, tuberculosis. In the records of my hospital cases of tuberculosis this is plainly brought out. No other disease, I believe, except measles, has such a powerful influence in preparing a soil for the ready reception of the tubercle bacillus.

Urinary System. — Transient albuminuria, probably due to congestive conditions, is not infrequent, and Gibb has pointed out the occasional occurrence of glycosuria.

TREATMENT.—In these days of enlightened sanitary science much is being done to check the spread of and stamp out zymotic disease. In regard to pertussis, a disease which, roughly speaking, causes about a fourth of the mortality in children in the larger towns of Great Britain, practically nothing has been done. It has not yet been included in the list of notifiable diseases.

Only in schools and public institutions has any attempt been made to isolate or stamp out this disease. Children are allowed to go about in public places, omnibuses, and tramcars, and railway carriages, coughing and vomiting on the public streets and thereby spreading the disease. Isolation is the only means at our disposal to limit the spread and stamp out the disease. Compulsory notification is a necessary first step in arousing the public to a realisation of the dangerous character of the disease in early life. So long as the old ideas prevail, which they do to a large extent, that children are just as well to "get over" such diseases as whooping-cough and measles, so long will this mortality continue. It is only by educating the public that we can hope to carry out a successful prophylaxis. As matters at present exist, what can be done? Among the poorer classes the difficulties are well-nigh insuperable. There is no possibility of isolation in their own homes, and sufficient hospital accommodation is not available. Sanitary authorities should use all possible means to prevent children of one family associating with those of others in contiguous houses, so that the disease may be restricted in its spread to the family originally affected. It ought to be rendered penal to take children suffering from the disease to public places and into public conveyances. The sanitary inspectors should see that the children in the affected house are under medical treatment, and that proper attention is directed to ventilation of the room and other hygienic measures. With regard to children of the better classes prophylaxis is more hopeful. Isolation in the house, if large enough, should at once be attended to, and the other children either removed altogether or kept strictly separate. In my own experience isolation can be successfully carried out in this way. The infection of pertussis requires more or less close contact, and its spread may be as successfully checked in this way as in scarlatina cases. Before removing unaffected children from the house, it is desirable that they should be quarantined in the house for a fortnight, after which time removal may be effected with less risk than in the case of immediate removal. Disinfection of the house and all articles of clothing worn by the child should be attended to, all carpets being taken up, and the floors mopped over with carbolic acid wash. In public institutions, with sick-room and hospital accommodation, isolation may be successfully carried out, the affected children being taken out daily if well enough, at a time when the other children are indoors.

Curative Treatment. — In the treatment of pertussis it is necessary to bear in mind the nature of the disease, a zymotic affection of self-limited duration, with which we have to deal. Our efforts must be directed on general principles to guide the patient as safely through the various stages of the complaint as possible, and to ward off complications. No hard and fast rule can be laid down as applicable in all cases. Mild cases running a favourable course in from two to four weeks require little or no special treatment. In many of these, as has been already indicated, the second stage is

hardly reached, or, at all events, the spasmodic efforts are so feeble as scarcely to merit special notice. In cases of average severity and in serious cases a great deal can be done by rational and prompt treatment to mitigate the severity and limit the duration of the disease, the acumen of the experienced physician having ample scope in regulating the treatment according to the special needs of the patient under observation, and avoiding irrational and unscientific routine.

Hygienic.—Pure fresh air is the most important means at our disposal. In suitable weather the patient should be out as much as possible if the case is uncomplicated, and the temperature non-pyrexial. If there is some febrile movement, as there is in most cases for a brief period, the child should be kept in bed. The nursery should be large and airy, the window kept always open. The bed should be placed so as to avoid a cross draught. The clothing should be warm and comfortable, particular care being taken in cold weather to keep the body and limbs from chill. It seems to me that this is one of the diseases of childhood which, in the future, will be treated with advantage on the open-air principle as in tuberculosis. In children there are many difficulties not obtaining in adults in carrying out this method, but I believe it has a great future before it in pediatric practice. In tuberculosis it is now being carried out in children of all ages with even more satisfactory results than in adults. We may reasonably expect that in zymotic and other acute microbic diseases great results may be looked for from open-air treatment.

Dietetic.—The diet during the height of the disease should be lighter than in health and easily digestible, and suited to the age of the child, milk and farinacea with soups forming the staple articles. During the course of the disease in many cases there is a liability to a more or less irritative gastro-intestinal catarrh, due partly to general systemic infection, but also directly to the irritant effects of the secretions from the air passages being swallowed. This is often more marked in cases where the child does not vomit freely several times in the day. In cases in which the vomiting is persistent after every severe paroxysm the child becomes greatly weakened and emaciated from inanition. In these cases it is the rule to feed the child with light and easily digested nutriment shortly after the paroxysm. The quantity should not be too large, but regulated according to the length of interval between the fits.

Medicinal.—The drug treatment of the disease is now conducted on a more rational and scientific basis than formerly. There are few diseases in which so many drugs have been tried, a tolerably sure indication of their general inefficacy.

During the catarrhal stage, whether the patient is confined to bed or not, it is well to give a saline expectorant mixture with liq. ammon. acetat., vin. ipecac., sodæ sulpho-carbolate in water. Benozate of soda may also be used, and is an excellent remedy in this stage. When the convulsive stage is reached drugs of a sedative nature should be given, such as lobelia, belladonna, and in some cases, with caution, morphia. Bromoform is a remedy which has been lauded by many, but in my own experience it is disappointing in its effects. Antipyrin is probably the most efficacious sedative of all. Phenacetin acts in a similar manner, but not with such good effect as antipyrin. Chloral and butyl chloral may be used with advantage in many cases where other sedatives fail. During the stage of decline we should trust to tonic drugs, and the old-fashioned astringent alum, which has well stood the test of time, is often useful. Undoubtedly the only satisfactory tonic in this stage is quinine or cinchona. The liquid extract of the latter may be given with syrup of Virginian prune. Excellent preparations of quinine are the tannate or hydrobromate. During convalescence no remedy does so much good as cod-liver oil, or, if there is anæmia, iron. The cod-liver oil should be continued for many months.

Local medication may be applied in solution, vapour, or the insufflation of powders. Careful and persistent local applications to the upper air passages are probably the most effectual means of relief to the irritative effects of poison on the mucous surface, if indeed by such means the disease may not be cut short. Nasal treatment is often of great service, and has in my opinion been too much neglected. Boracic acid is generally used for this purpose, the nostrils being washed out with equal parts of warm water and a saturated solution of the acid two or three times daily, either with an ordinary anterior nasal spray or syphon douche. Spray with 1 to 4000 sol. of hydrarg. perchlor. may be occasionally used in place of the boracic solution, but discontinued if any irritant effects are produced from swallowing small quantities of the solution. Watery solutions of sulphate or hydrobromate of quinine are often of much service when applied in this way to nose and throat. A very useful spray for nostrils and throat is 1 per cent carbolic acid and 3 per cent menthol in paroleine. When the child permits of its frequent and free application it affords great relief.

Insufflations are undoubtedly of much service in many cases where it is possible to apply them, but in my experience this application is difficult in most children. For this purpose quinine is the most efficacious, either alone or combined with one-fourth of resorcin. Iodoform, 1 to 4 of talc, has often a most sedative effect when insufflated into the nostrils. Boracic acid with powdered tragacanth is also a useful

powder. *Inhalants* are undoubtedly of great service, and should be commenced during the catarrhal stage, and continued regularly at intervals during the spasmodic period. Carbolic acid, No. 4 or 5 of Calvert's, creseline, naphthalin, and lysol are the best. They should be used in a vaporiser with a night-light, and placed on a table at the head of the child's cot at night, and repeated with more or less frequency according to the severity of the case. I generally recommend two or three inhalations of two hours' duration in twenty-four hours in severe cases. I have not found any poisonous effects as a result. In a well-ventilated room there is generally sufficient dilution to prevent this. Under persistent antiseptic inhalations the cough diminishes in frequency and severity. The only other inhalant which is of service in extreme cases when the paroxysm is so bad as to threaten suffocation is the inhalation of chloroform, a few drops on a handkerchief with or without a drop or two of amyl nitrite.

Intubation.—This has been lately tried and with good effect in very severe cases when the paroxysms are so frequent and the glottic spasm so severe as to exhaust the child in an alarming manner. O'Dwyer's rubber tube must be used instead of the ordinary metal instrument.

The treatment of complications must be conducted on general principles applicable to the various conditions, and does not come within the scope of our present remarks.

Whytt's Disease.—Hydrocephalus or tubercular meningitis (after Dr. R. Whytt of Edinburgh, 1714-1766).

Wichmann's Asthma.—Laryngismus stridulus (*q.v.*).

Wickersheimer's Fluid.—Two fluids are used, one for injecting specimens and the other for immersing them; both contain arsenious acid with potassium salts, glycerine, etc.

Widal's Reaction. *See* Blood (*Bacteriological, Widal's Reaction in Typhoid Fever*); Typhoid Fever (*Pathology, Widal's Reaction*).

Wiesbaden. *See* Balneology (*Germany, Muriated Waters*); Mineral Waters (*Muriated Saline*).

Wight, Isle of. *See* Therapeutics, Health Resorts (*English*).

Wildbad. *See* Balneology (*Germany*); Mineral Waters (*Thermal*).

Wilde's Incision. *See* Ear, Acute Inflammation of Middle (*Treatment, First Step in Operation*).

Wildermuth's Ear.—A congenital malformation of the outer ear, consisting in marked prominence of the antihelix.

Wildungen. *See* Balneology (*Germany, Calcareous Waters*).

Wilkinson's Disease.—Paralysis Agitans (*q.v.*).

Wilks' Disease.—Chronic parenchymatous nephritis; the large white kidney is called Wilks' kidney. *See* Nephritis (*Pathological Anatomy*).

Will. *See* Alcoholic Insanity (*Clinical Features, Impairment of Will*); Aphasia (*Will-Making*); Civil Incapacity (*Will-Making*); Hysteria (*Disorders of the Mental State*).

Willan's Lepra.—Psoriasis (*q.v.*).

Willan's Lupus.—Lupus Vulgaris. *See* Skin, Tuberculosis of.

Williams' Tracheal Tone.—A peculiar dull tympanitic resonance heard sometimes in the second intercostal space in cases of great pleural effusion and due to the vibration of air in a large bronchus which is surrounded by compressed lung tissue.

Williamson's Test.—A blood-test for diabetes depending on the fact that a warm alkaline solution of methylene blue loses its colour when mixed with a small quantity of glucose.

Willich's Formula. *See* Vital Statistics (*Expectation of Life*).

Willis, Circle of.—The system of inosculating arteries at the base of the brain. *See* Brain, Physiology of (*Blood-Supply, Circle of Willis*).

Willis' Disease.—Diabetes Mellitus (*q.v.*).

Willis' Glands.—The corpora albicantia in the interpeduncular space at the base of the brain.

Willis' Nerve.—The spinal accessory nerve (*q.v.*).

Wilson's Disease.—Dermatitis exfoliativa.

Winckel's Disease.—Hæmoglobinuria neonatorum or bronzed hæmaturic disease of the new-born infant. *See* New-Born Infant (*Winckel's Disease*); Hæmoglobinuria (*Infantile Type*).

Winds. *See* Climate (*Individual Factors, Influence of Winds*); Meteorology (*Anemometry*).

Wine-glass. *See* Prescribing (*Weights and Measures*).

Wines. *See* ALCOHOL; VINA; and under COLCHICUM, FERRUM, QUININE, etc.

"Winged" Scapula. *See* MUSCLES, DISEASES OF THE (*Idiopathic Muscular Atrophy, Facio-Scapulo-Humeral Type*).

Winking. *See* NICTITATION.

Winslow, Foramen of.—The opening between the greater and lesser cavities of the peritoneum. *See* PERITONEUM (*Anatomy*).

Winslow's Ligament.—The posterior ligament of the knee-joint. *See* KNEE-JOINT, DISEASES OF (*Anatomical Considerations*).

Winslow's Pouch.—The gastro-heptic omentum.

Wintergreen. *See* GAULTHERIA.

Wintrich's Sign.—The fact that the percussion note over a cavity in the lung differs in pitch with the opening or shutting of the mouth.

Wiring, Operation of. *See* FRACTURES (*Treatment, Wiring*); MOUTH, INJURIES AND DISEASES OF THE JAW (*Mandible, Fracture, Wiring*).

Wirsung, Duct or Canal of. *See* PANCREAS (*Physiology, Development*).

Witch's Milk. *See* MAMMARY GLAND, DISEASES OF (*Inflammatory Affections, Mastitis of Infancy*).

Woillez' Disease.—Acute congestion of the lungs as an independent disease.

Wolffian Body.—The mesonephros, the duct of which is called the Wolffian duct. *See* BROAD LIGAMENT, DISEASES OF (*Tumours of*); GENERATION, FEMALE ORGANS OF (*Organ of Rosenmüller*).

Wolffian Cyst.—A cystic tumour in the broad ligament or its immediate neighbourhood, believed to be developed from the relics of the Wolffian body or parovarium. *See* OVARIES, DISEASES OF (*Cysts*).

Wolfler's Operation. *See* STOMACH, SURGICAL (*Operations, Gastro-Gastrostomy*).

Wooden Tongue. *See* ACTINOMYCOSIS (*Synonyms, Scirrhus Tongue*).

Woodhall Spa. *See* BALNEOLOGY (*Great Britain, Muriated Waters*); MINERAL WATERS (*Muriated Saline*).

Wood Ticks. *See* SCABIES (*Other Acari, Ixodes*).

Wood Wool.—Pine-wood, finely divided and impregnated with corrosive sublimate; an antiseptic dressing.

Woolsorters' Disease. *See* ANTHRAX (*Synonyms*).

Woorara. *See* CURARA.

Word-Blindness. *See* APHASIA (*Alexia or Visual Aphasia*); BRAIN, PHYSIOLOGY OF (*Word-Seeing Centre*).

Word-Deafness. *See* APHASIA (*Clinical Varieties, Auditory Aphasia*); AUDITORY NERVE AND LABYRINTH (*Nerve-Deafness, Word-Deafness*); BRAIN, PHYSIOLOGY OF (*Word-hearing Centre*).

Work. *See* PHYSIOLOGY, TISSUES (*Muscle, Work of; Effects of Muscular Work*).

Wormian Bones.—Accessory bones found in the cranial sutures; ossa triquetra.

Worms. *See* PARASITES (*Trematodes, Cestodes, Nematodes*); *see also* ABDOMEN, CLINICAL EXAMINATION OF (*Symptoms*); CHOREA (*Causes*); ENEMATA (*Therapeutic Uses, Parasites*); FÆCES (*Foreign Bodies*); RECTUM, DISEASES OF (*Procidentia Causes*); URINATION, DISORDERS OF (*Enuresis*).

Worthing. *See* THERAPEUTICS, HEALTH RESORTS (*English*).

Wounds.

DEFINITION 533
VARIETIES 534
MODES OF REPAIR 534
TREATMENT 536
Immediate
General, Local 536
Subsequent
General 541
Local 542

See also ABDOMEN, INJURIES OF (*Wounds*); ANKLE-JOINT, REGION OF, INJURIES (*Wounds*); ASEPTIC TREATMENT OF WOUNDS; BLADDER, INJURIES AND DISEASES OF (*Penetrating Wounds*); BRACHIAL PLEXUS, SURGICAL AFFECTIONS OF (*Wounds*); CHEST, INJURIES OF (*Soft Parts*, etc.); DIPHTHERIA (*Clinical History, Wound Diphtheria*); HEART, SURGERY OF; HIP-JOINT, INJURIES OF (*Wounds*); KNEE-JOINT, INJURIES OF (*Wounds*); MALINGERING (*Wounds*); MEDICINE, FORENSIC (*Wounds and Injuries*); MENSTRUATION AND ITS DISORDERS (*Vicarious Menstruation*); NECK, REGION OF (*Injuries, Wounds*); SPINE, SURGICAL AFFECTIONS OF (*Contusions*, etc.).

DEFINITION.—A wound is a breach in the skin or a mucous membrane suddenly made by mechanical force, and thus opening a way into deeper tissues. This strictly surgical definition must be distinguished from the medico-legal use of the word, which, by judicial decisions, though not by statute, is held to include such

injuries as dislocation and fracture occurring beneath the unbroken skin as a result of violence. The popular application of the term to any open sore is here excluded. Fissures and other solutions of continuity in bone and cartilage, whilst having much in common with wounds, are, in precise surgical language, fractures.

Varieties.—It is usual and of practical convenience to classify wounds, according to the appearance of their edges, as follows:—

1. *Incised.*—The injury having been inflicted by a sharp object, as a knife or glass, shows clean-cut edges.

2. *Lacerated.*—In these the margins are irregular and ragged, the parts having been subjected to a stretching or dragging force. Striking examples occur in machinery accidents, as, for instance, where the hair gets entangled in a moving band, and the scalp is torn open and more or less detached by the excessive tension.

3. *Contused.*—Here also there is unevenness of the edges; but there is in addition a certain amount of adjacent and subjacent bruising,—that is, infiltration of the tissues with blood effused from ruptured vessels,—the extent of bruising varying with the force of the blow and the area and vascularity of the part struck. Wounds of this kind are caused indifferently by the sudden impact of a blunt object, or by a fall against a fixed surface or projection,—the efficient factor in either case being violent pressure or squeezing.

Irrespective of the character of its borders a wound is termed—

(*a*) *penetrating*, when it opens into a joint or cavity of the body;

(*b*) *perforating*, when the lesion extends through a part, so as to produce a wound of exit as well as one of entrance;

(*c*) *punctured*, when the depth exceeds the length; and

(*d*) *infected*, when pathogenic bacteria or their products have entered and set up morbid processes: (*poisoned* wounds resulting from pricks or scratches in making a post-mortem examination are examples of infection, and, in this connection, the special term is unnecessary; though it may be retained for wounds into which chemical, as distinguished from microbic, poisons are instilled).

Bites inflicted by the teeth and beaks of animals combine the characters of bruising and tearing; they are very liable to be infected by material conveyed from the animal's mouth at the time of wounding, though the liability is diminished if the teeth pass through layers of clothing before reaching the skin. The bites of venomous snakes owe their special danger to the poisonous gland-secretion discharged into the wound through grooved or canalised fangs, and giving rise to general toxæmia.

Repair of Wounds.—It may be premised that wounds of every kind are repaired by a method common to them all, the final result being that the breach is filled up with organised new material. In some specialised structures as nerve, striated muscle, and tendon, there may be, to a slight extent, an actual regeneration of similar tissue; but as a rule the new formation is a simple connective material known as scar-tissue. Injuries of bone are not here considered, but it may be noted in passing that, whilst their repair is effected by a solid substance, the earlier stages of the process are identical with what is observed in the soft parts.

When the surface-opening of a wound is closed without undue delay, and no disturbance or complication such as septic infection ensues, healing takes place by (1) *primary union*, or by (2) *the intermediation of blood-clot*, or (3) *under a scab*. In the case of an open wound, or the open part of one partially closed, healing proceeds by (4) *granulation*, or, in a later stage, by (5) *the union of granulations*.

1. *Healing by primary union or first intention* is exemplified in the repair of an uncomplicated incised wound, in which the cut edges and surfaces are kept in close contact after all bleeding has ceased. The immediate effect of the injury upon the contiguous zone of tissue is the setting-up of the nutritional disturbances characteristic of commencing inflammation, which proceed to their completion in organisation and resolution in such a manner that the chink of the wound becomes filled with vascularised fibres organically continuous with the neighbouring connective tissue, whilst the latter reverts to its normal condition. It will be sufficient here to give the following summary of the histological processes in question.

The first obvious change is dilatation of the arterioles, capillaries, and veins nearest to the blood-vessels whose divided mouths have been sealed by clot; and coincidently the rate of their blood-flow is quickened. These phenomena are followed by a slowing down of the local current, often to the point of stagnation, together with leakage of serous fluid and emigration of leucocytes into the surrounding connective tissue spaces in consequence of increased permeability of the altered vascular walls. This sero-corpuscular exudate undergoes partial coagulation with the formation of fibrin, and constitutes in the affected region a glue-like mass, continuous across the sides of the wound which are thus, so to speak, soldered together. Objectively to sight, the parts are rather more swollen and reddened, and to touch, warmer and firmer than in health. The cellular elements of the exudate become reinforced in number by the arrival of amœboid wandering cells, and also by the detachment and multiplication of fixed tissue

cells. The important function of forming new connecting tissue appears to be mainly if not wholly performed by the last-mentioned group. They become fusiform or otherwise modified in shape, and put forth slender fibrils which blend with similar offsets from other cells in their vicinity; and as repair advances, the exudate is in this way penetrated by a network of delicate interlacing fibres stretching across the track of the wound and effecting organic union of its walls. Blood-vessels develop for the conveyance of pabulum to the young fibrous growth: they appear as buds jutting from the walls of capillaries, and elongate into protoplasmic cords with tapering ends, which unite with like processes from neighbouring capillaries so as to form arches; canalisation takes place, and circulation of blood is established through the anastomotic loops thus formed. Meanwhile the redundant portions of the exudate are removed: fluid is drained away by the absorbents, surviving leucocytes migrate—some perhaps laden with molecular particles of disorganised structures ingested by phagocytosis, and the fixed tissue cells further assist in the work of scavenging. With the roofing-in of the surface by epithelium proliferated from the rete Malpighii, bordering on the edges of the wound, healing is complete, although the permanent condition of the fibro-vascular nexus—technically, the *cicatrix* or *scar*—is not attained for some months, during which gradual contraction goes on, leading to attenuation of the blood-vessels and shortening and condensation of the fibres.

2. *Healing by the Intermediation of Blood-clot.*—To a greater or less extent this mode of repair occurs in a large proportion of wounds united by first intention, inasmuch as small masses of coagulum collect in hollows and fissures opening from the cut wall; but it is seen in its typical form when a layer of clot of varying thickness separates the sides of a wound and prevents their close contact. Dendritic projections reach from the main mass into the divided blood-vessels, and the whole at first serves the useful purpose of a plastic cement supporting the wounded structures. Inflammatory exudation ensues upon the injury, and the exudate spreads as a thin coating between the clot and the tissues, whilst leucocytes and other migratory cells wander into the former, which now constitutes a matrix for the developing network of fibres and capillary loops. *Pari passu* with advancing tissue-genesis the blood-clot becomes decolorised and removed by disintegration and absorption of its corpuscles and fibrin. In the later stages epithelial cells restore the continuity of the surface.

3. *Healing under a Scab.*—In some cases where the margins of a wound are not actually touching, and the superficial stratum of exudate or blood-clot, or a mingling of both, dries up, the resulting crust is called a scab, and it may suffice to seal the aperture. Assuming that asepsis of the interior has been secured, the scab is a good barricade against bacterial infection later on; and under these circumstances union of the raw surfaces proceeds by first intention—quickly if the surfaces are touching, more slowly if there is an intervening clot. Epithelium grows across from the edges beneath the scab; and when the latter eventually falls off, the healed seam of the cicatrix is disclosed. When a dry crust covers the surface of a shallow wound made by superficial loss of substance, healing takes place by granulation, as described below.

4. *Healing by Granulation or Second Intention.*—Septic infection—a danger very likely to be met with in wounds of mucous membrane, and in accidental wounds generally, or those not carefully safeguarded against contamination—sets up a more energetic local reaction than is presented by the foregoing types, and is accompanied with a much greater amount of tissue-degeneration. Under these circumstances recovery is somewhat delayed, healing being effected by the second intention, a comparatively slow process, exhibiting phenomena in which granulations play a conspicuous part.

In the first instance, the injury excites a traumatic inflammation, but the limits of this are widely transgressed by the irritation consequent upon the invasion of micro-organisms belonging to the class of pyogenic bacteria. Those usually found include the common varieties of staphylococcus (S. pyogenes aureus, S. pyogenes albus) and streptococcus (S. pyogenes); but it would seem that other fungi also possess the power of inducing, or at all events promoting, the formation of pus, *e.g.* the gonococcus, the pneumococcus, and the bacillus coli communis. Once securing a lodgment in the damaged structures, the bacteria are able to excrete peptonising agents, and to produce poisonous chemical compounds (toxins), which are extremely hurtful to the tissues exposed to their action. By a kind of digestion, fibres, cells, and intercellular substance are softened and more or less disintegrated and dissolved, the resulting degenerative products being washed out by the copious liquid exudate escaping from blood-vessels in the site of inflammation, many leucocytes being at the same time cast off. On the other hand, some of the toxic material may enter the general circulation and bring about constitutional disturbance. Locally, the discharge from the wound thickens, assuming the yellowish colour of pus, and suppuration is established. The constituents of pus comprise fluid derived from the blood and liquefied tissue, numerous leucocytes, some altered tissue-cells, necrotic particles of fibres and corpuscles, and pyogenic cocci; besides leucin, tyrosin, and peptone. As the stress of the attacking infec-

tion declines, the boundaries of the lesion are fenced off by a protecting barrier of leucocytes, and evidences of repair manifest themselves by the appearance of minute granular points, bright red in colour, studding the raw surfaces. These are the granulations characteristic of healing by the second intention. Each granulation consists of a newly-formed vascular loop arising from pre-existing capillaries, enveloped in layers of cells, of which the superficial ones are constantly being shed, whilst those in contact with the vessel are developing into fibres. As reconstruction advances, the granulations grow upwards and from the sides so as to progressively fill the gap, whilst beneath them fibro-vascular scar-tissue increases in quantity, contracting as it does so, and thus diminishing the wound in all its dimensions. With the approach of granulations towards the level of the skin, epithelium grows across from the edges, and surface continuity is restored.

5. *Healing by the Union of Granulations or Third Intention.*—When the opposite walls of a wound are lined with healthy granulations, and these are pressed into apposition, as by suturing, organic union may supersede the mere mechanical contact. The process by which interlacing fibres and capillary loops blend the two sides together is identical with what has been already described. Advantage can sometimes be taken of this mode of repair in order to hasten recovery when primary union has failed.

Summary.—From a review of the foregoing condensed account of healing, it is evident that the processes and their results are essentially of the same nature in all classes of wounds. Whatever mode of repair is investigated, the underlying feature, to which there is no exception, is inflammation, associated, as recoverable inflammation invariably is, with degeneration and generation of tissue. Traced to their foundation, therefore, the diverse phases of wound-repair are simply so many variations in the intensity of inflammatory reaction called forth by injury, whether that be mechanical violence acting alone or aggravated by harmful micro-organisms. In the case of primary union pure and simple, or one of its modifications of healing through a blood-clot or under a scab, disturbance is slight and reaction proportionately small, and in favourable examples the amount of new tissue constructed is almost infinitesimal; and whereas the contrary holds good for granulating wounds, yet after all the difference is merely one of degree rather than of kind, and the ultimate result is universally the same, namely, the genesis of a fibro-vascular nexus, of which all scars in the soft parts are composed.

IMMEDIATE TREATMENT. — A. *General.* — The general condition of the patient will claim the surgeon's early solicitude. If there be much *shock*, the prompt administration of stimulants is indicated. This should take the form of hot stimulating fluids given by the mouth or rectum; or the injection of such drugs as ether, strychnine, digitalis and atropine under the skin. The hypodermic method will yield the most speedy results. The patient must be placed flat on his back with the head lowered by tilting up the foot of the bed on chairs; and warmth is to be promoted by blankets and hot-water bottles, care being also taken in cold weather to maintain the temperature of the room. *Anæmia* due to profuse bleeding demands the infusion of saline fluid into the subcutaneous connective tissue (hypodermoclysis). Normal salt solution 0·6 per cent warmed to 105° F. answers well, and as much as a pint may be allowed to flow into the chest-wall through a large hollow needle connected with rubber tubing and a glass funnel, the rate being kept slow by not unduly raising the funnel. Alternatively the fluid may be injected into the rectum. *Pain* should be quelled by morphia, which has the further beneficial effect in many cases of mitigating shock. In susceptible persons there is often a degree of *faintness* out of proportion to the extent of injury. The recumbent posture is here imperative, and indeed is advisable during the immediate treatment of all wounds. If the patient be seated, and syncope occur owing to painful manipulations or emotional disturbance, he may fall to the floor, to the confusion of the surgeon whose attention has been engrossed by the wound.

B. *Local.*—The paramount considerations to be kept in view from first to last are the securing of *asepsis* and *rest*. Of these asepsis is of supreme concern, but rest must also be regarded as absolutely essential to perfect success. If both conditions be efficiently maintained throughout, healing will take place quickly and satisfactorily with the slightest local changes, and, what is of vastly greater moment, constitutional disturbance, with all its attendant dangers, will be reduced to a minimum.

The technique of present-day wound-surgery and its scientific explanation are intimately related to the study of bacteriology—particularly that branch which concerns itself with pathogenic organisms. The fact is now thoroughly established that many varieties of harmful bacteria abound in the air and dust of all places inhabited by man. They find favourable harbourage in the skin and mucous membranes, entering the mouths of ducts and penetrating between epithelial scales into the deeper layers of the epidermis, whence they can readily invade the vascular tissues through any breach of surface-continuity. All objects, other than germicidal agents, freely exposed to the atmosphere of populated districts become in like manner contaminated by the deposition of particles of dust or dirt, which to the surgical mind are merely synonyms for germs, and,

commonly, pyogenic germs; and the contact of such contaminated objects with the raw tissues of a wound means infection. Thus clothing, sponges, water, lotions, instruments, ligatures, sutures, or dressings, if permitted to touch a wound without previous sterilisation, may give rise to most dangerous septic processes. Above all, the unpurified fingers of the surgeon or his assistants are liable to convey material into a wound with consequences no less fatal than those following the invasion of a bullet or bayonet into a vital organ.

A fundamental distinction must be drawn between wounds deliberately made by the surgeon through healthy integuments, and those arising from accident or assault. In the former group it is the bounden duty of the surgeon to adopt the most scrupulous precautions possible to ensure asepsis before, during, and after the operation. Not that complete sterilisation is certain to be attained by the means so far introduced, although it is the end to be strenuously pursued. The entrance of some germs into subcutaneous tissues freely exposed to air in its ordinary state is inevitable; but happily it is consistent with general experience that the presence of a small number of organisms introduced in that way does not prevent healing by first intention, always provided that the stringent rules of modern practice are faithfully followed. As to the propriety of seeking to remove or destroy these intruded bacteria by the application of an antiseptic, and, if thought appropriate, what antiseptic should be employed, surgical opinion is divided. The writer holds, with due deference to the views of others, that it is prudent, whenever extensive tracts are opened up in an operation, to apply a suitable antiseptic fluid to the cut surfaces before closing the wound; and for this purpose dilute izal lotion (izal 1 part, boiled water 800 parts) has proved satisfactory.

On the other hand, the surgeon is confronted with an altogether different set of conditions in the case of accidental wounds. Here there has been no preparatory disinfection, and therefore, having regard to the absence of prophylactic measures before the injury was inflicted, the only safe course is to assume, as a working rule admitting of very few exceptions, the existence of septic infection when the patient comes under surgical care. It is usually difficult, not seldom impossible, to overtake the initial disadvantages. The wound, soiled at the moment of its occurrence, may have been covered over by dirty wrappings, or, what is worse, meddled with by unprepared hands, carrying infection into its recesses. When, as frequently happens, the damaged parts are contused or lacerated, and still more when palpable foreign bodies have been ground in, the difficulties are immensely increased. But all this should not deter us from painstaking efforts to undo the mischief; on the contrary, it should be an incentive to the greatest nicety of detail in every step of the treatment, so far as the exigencies of any given case permit, in order that the conditions may be brought as closely as possible into line with those of a formal operation-wound. To this end it is essential that nothing but what is germ-free or germicidal should be allowed in or near the wound. In what follows it will be understood that accidental wounds are referred to, and the considerations stated above are assumed.

The requirements of local treatment comprise *hæmostasis*, *disinfection of the wound and its immediate surroundings*, *coaptation*, *drainage*, *dressing*, and *provision for rest and favourable circulation*.

1. *Hæmostasis.*—Bleeding is so pressing a danger when copious, and so much delays repair when allowed to go on even slightly within a closed wound, that the flow requires to be arrested at once and completely. The arrest may be natural or artificial. In either case the openings into the vessels become blocked by blood-clot or in other ways mechanically closed, the blood-pressure being at the same time lowered; and the obstruction persists until permanent sealing takes place. Hæmorrhage from small vessels will stop spontaneously, or upon exposing the part to the air. For a wound of an extremity, elevation of the limb is a most valuable means of checking further loss of blood, the value of this method being the greater the more distal the wound. In the case of a hand the whole limb should be raised vertically by grasping uninjured digits, or supporting the elbow. For moderate arterial bleeding, digital compression of the main artery is indicated as a temporary measure until the vessels are secured. Profuse hæmorrhage from a limb can be promptly arrested by tying stout indiarubber tubing tightly round the limb at a higher level, first protecting the skin encircled with folded lint to prevent bruising. It is well to bear in mind that unless the ligature is firmly applied, the supplying arteries will not be fully controlled and oozing will continue from the wound. It is not uncommon to see free venous bleeding when an improvised tourniquet has been imperfectly placed above a wound by a well-intentioned bystander rendering first aid after the accident. Such bleeding will diminish or entirely cease on removal of the tourniquet.

As a general rule the wound must be widely opened and examined in a good light. The surfaces should be freely flushed with hot antiseptic lotion squeezed out of swabs of sterilised gauze, and all bleeding points can then be seized one after another with pressure-forceps—Spencer Wells' pattern or some modification. If any form of compression has been practised, this is cautiously relaxed, and visible vessels clamped

until only oozing remains to be commanded, which is best done by gauze swabs. Ligatures, preferably of catgut, had better now be tied with reef-knots round the points grasped by the forceps, and these taken off; or torsion may be tried, but it is a less certain hæmostatic than the ligature. In some situations, for instance, near a bone, it may be necessary to underrun a vessel with a needle armed with catgut, which is then tied (suture-ligature); or a tenaculum hook may be required to draw up a bleeding point and render it more accessible. All ligatures are to be cut off short near the knot. Troublesome capillary oozing will often yield to the affusion of lotion or boiled water (to which common salt may be added in the proportion of one drachm to a pint), at a temperature of 115° F. Carnot's solution (gelatine 5 parts, calcium chloride 1 part, water 100 parts) has been recommended for the same purpose; and more recently preparations of adrenalin. Packing with a gauze tampon should only be resorted to when ligation is not available.

Incised wounds bleed much more freely than the contused and lacerated, as they are without the irregularities so conducive to coagulation possessed by the latter. In particular, deep punctures, as knife-stabs, into fair-sized arteries, are often attended by most dangerous hæmorrhage. Every such case must be treated on its merits; in many it will be necessary to explore the track by making a free incision so as to reach the wounded artery, which is then to be tied both above and below the wound, and divided between the two ligatures if there is undue tension of that portion; in others it will be justifiable to rely on pressure without disturbing the clot which has, perhaps, already formed a more or less adequate plug, cases of this class being regarded as exceptions to the almost invariable rule requiring wounds to be opened up.

The indications for hæmostasis having been thus complied with, the wound is to be covered with a gauze swab while attention is given to the next step.

2. *Disinfection of the Wound and its Surroundings.*—This will repay the utmost care that can be bestowed. With the region well uncovered, first the area around the wound should be systematically and most thoroughly cleaned. Here the razor aided by soap lather is invaluable, not only on the hairy scalp, but wherever it can be applied, as it removes gross dirt together with the outer epidermal scales. The precaution should be observed of drawing the blade in planes parallel to the wound or away from it, so as to avoid carrying dirt up to the margins. After shaving (or, if that be impracticable, without it) the surface is to be well scrubbed with soap and hot water, the brush having been previously boiled in 1 per cent sodium carbonate solution and kept ready for use in antiseptic fluid. Free ablution with boiled water will wash away traces of soap and loosened epithelium. Then follows brisk rubbing with methylated spirit on wool, commencing at the edges of the wound and working outwards in concentric rings, to get rid of fatty substances. Antiseptic lotion is to be douched over the purified area, which is then fenced off by towels as a safeguard against reinfection.

A word or two may be here interpolated as to the choice of antiseptic. Having regard to the fact that skilled workers, each employing a different chemical selected out of a large variety, can all point to equally successful results, it seems unquestionable that scrupulous observance of detail is incomparably more important than the actual substance employed. The ideal antiseptic has yet to be discovered. Intensely germicidal chemicals are irritants to the tissue-elements—though the harmfulness of this, so far as antiseptics of the strength in ordinary use are concerned, has perhaps been magnified, and at all events it is a far lesser evil than sepsis. After several years' ample experience of izal in the casualty department of the Queen's Hospital, Birmingham, the writer can speak very favourably of this substance in the treatment of accidental wounds. He prefers it as the antiseptic for routine purposes on the following grounds:—(i.) it is a powerful germicide; (ii.) it is not poisonous if absorbed; (iii.) it is less irritating to the tissues than some of its chief rivals; (iv.) it does not tarnish instruments; (v.) it is not costly. For recent wounds a dilution of 1 part izal to 800 parts of boiled water may be used; in suppurating cases and for application to the skin, double or treble that strength is indicated.

Turning now to the wound itself, this must be dealt with according to the conditions met with. There may be a simple incision or any gradation of wound up to an extensive laceration whose ragged surfaces are begrimed with grease from machinery or road-dirt which has been ground into the tissues by a cart-wheel. The merest bathing will suffice for the former; whereas the latter demands thorough-going measures, which can only be adopted, speaking generally, after the patient has been anæsthetised. The first essential in every case is to remove all extraneous matter. Defined and fairly loose foreign bodies, *e.g.* hair, grit, glass, straw, mud, can be removed by means of forceps, sharp spoon, and swab, supplemented by liberal douching; grease and embedded particles will need the careful employment of hand-brush and soap; dirt still more ingrained can only be cleared away at the sacrifice of some tissue by the judicious use of scalpel and scissors. The danger lurking in partially concealed spaces is to be remembered, and the wound enlarged when necessary and the roof of any undermined tract laid open, in order to fully expose the

whole of the interior and its recesses. Only in this way can sufficient access be gained to ensure thorough disinfection. Tags of skin or fat hopelessly damaged by crushing or detachment should be cut away, since they will necrose if left.

All bleeding having been finally arrested, the whole of the wound is now to be washed over with antiseptic lotion as completely as possible, no part escaping attention. The good effect of this is largely due to the mechanical removal of germs and débris, but there must also be added the undoubted power of the antiseptic to destroy, or, short of that, inhibit germ life. Following ablution, the parts are to be dried with gauze swabs.

3. *Coaptation.*—The closure of accidental wounds is apt to be overdone. It is much better to err on the side of doing too little than too much in this direction, even at the cost of some delay in healing. The majority of cases are furnished by the contused and lacerated varieties, and in many examples of these there are fragments of tissue bound to perish, whilst all of them contain parts so devitalised by traumatism that they can offer little or no resistance to pyogenic bacteria. Besides the admitted uncertainty of effecting absolute sterilisation, the risks of further infection are multiplied by a patient's lack of care and knowledge, together with the uncleanly circumstances of the poorer class; added to which there may be constitutional or local conditions prejudicial to quick repair. Hence, except in a richly vascular region like the face, it is seldom advisable to entirely coapt the edges, lest septic products should get shut in.

Within the wound the ends of any divided tendon or important nerve should be sought for and accurately stitched together,—with catgut, if there is reasonable prospect of speedy healing, otherwise with silk, as likely to hold longer. The search for the retracted proximal end of a tendon is not always an easy matter, and will often involve an extension of the wound: relaxation of the parts will here prove helpful. Cut muscle also should be sewn, but unless the division is complete a point of suture here and there will suffice. Wherever practicable, successive planes of divided tissue should be reunited in their proper layers by buried catgut sutures—muscle to muscle, fascia to fascia, skin to skin.

The question of coaptation may be summed up in the general statement that, as a rule, incised wounds should be closed, all others left more or less open. In the worst lacerations the idea of primary closure cannot be entertained; all that can be done is to fill the gap with a light packing of gauze and await the arrival of conditions favourable to secondary closure. The surgeon will exercise his discretion in dealing with the many intermediate forms of wound between those which may safely be brought into exact apposition and those which must be treated by the open method. Undue tension must always be avoided; it is certain to be followed by cutting through of the stitches, or death of the stretched flap,—as, for example, in the common case of a triangular flap of skin torn down a limb away from its blood-supply.

Suturing is the most suitable way of drawing the edges into contact. The interrupted method is to be preferred to the continuous, because the stitches are more independent, and one or two can be removed without disturbing the rest if it should become necessary to reopen part of the wound. The smooth and non-absorbable silkworm-gut, horse-hair, and silver wire, are the best suture materials for this purpose. Many wounds can be adequately closed by the pressure of dressings alone. Adhesive strapping is sometimes useful: it should be cut narrow in the middle and with broad ends (fiddle-shaped), so as to take firm hold of the skin on either side of the wound, and the latter should be covered with a strip of gauze to prevent the bridge of strapping from actual contact with the line of union. Or the gauze may be applied transversely, one end fixed with collodion, the margins of the wound approximated, and the other end of the tense gauze similarly fastened to the skin. Metallic clips without sutures have also been recommended.

4. *Drainage.*—Exudation of sero-albuminous fluid occurs in every wound, the quantity varying with the extent and characters of the injury. The absorption along lymph channels into the blood of fluid and other products of traumatism (notably fibrin-ferment, or, according to Schnitzler and Ewald, nucleins and albumoses), gives rise to the phenomena of simple (aseptic) traumatic fever. Moreover, the exudate, in virtue of its composition and temperature, is a pabulum highly favourable to the growth of pyogenic bacteria, and with their inoculation into the wound inflammatory reaction is greatly intensified, fluid exudes more copiously, and toxins are produced. Septic intoxication (sapræmia) results from the absorption of those chemical compounds, whilst locally the clinical evidences of inflammation manifest themselves and the wound undergoes retrogressive changes. The still more dangerous systemic phenomena of septicæmia or pyæmia follow the onset of suppuration if associated with the transmission of bacteria, alone or mingled with putrifying particles, into the blood-stream. The more abundant the secretions, provided there is no exit by which they can escape externally, the greater will be the tension about the wound and consequently the more readily will constitutional infection ensue; added to which, if the patient survive the dangers incurred, there may be considerable damage to structures in the region of the wound, resulting in permanent

loss or impairment of function. Further, the tension and other evils will be augmented if any bleeding goes on owing to imperfect hæmostasis.

It will be gathered from the foregoing data that, whilst exudation is an inevitable concomitant of wounds, retention of the products is always harmful, and the supervention of sepsis may imperil or destroy life. Hence it is imperative to minimise the secretions, protect them from bacterial infection, and facilitate their rapid escape. In the simplest cases of clean incised wounds whose walls can be brought into exact apposition, exudation is so insignificant that it may in practice be disregarded. At the other extreme of the series are the cases in which the most inexperienced will see the obvious necessity for providing wide drainage channels. For the rest it will be wise to apply the prudent aphorism, "when in doubt, drain." The simplest mode of doing this, supposing a wound in which it has been decided to insert sutures, is to leave the dependent angle open so that it may act as a natural drainage outlet. Artificial drainage may be effected in various ways; the objects they all have in common are to establish a route from the interior to the surface, and insert along it a conductor intended to guide discharges out of the wound. The fullest advantage must be taken of gravitation by arranging that the outlet shall be at the lowest point, the position of which will be determined by considerations of posture. Thus it will sometimes be requisite to make a counter-opening at a distance from the wound-edges for the easy drainage of a lowlying pocket, —for instance, where a large flap of scalp is torn down from the vertex. India-rubber tubing, introduced to surgical practice for this purpose by Chassaignac, is a valuable appliance for wound-drainage. Holes are made at intervals in the wall of a vulcanised tube of suitable calibre and length, and this is lodged in the wound so as to meet the requirements of any given case. For easier insertion the inner end may be cut obliquely to a point; the outer end should barely project beyond the surface, like a short spout, to avoid kinking or occlusion by the dressing, and may be prevented from slipping in or out by stitching it to one border of the wound with a single suture,—though, in general, only the former accident needs safeguarding, and this can be done by the simple device of transfixing the mouth of the tube with a safety-pin. Decalcified bone tubes are sometimes used instead of rubber. Most efficient drainage can be ensured by means of strips of gauze (cheese-cloth), which may be plain, or impregnated with an antiseptic, *e.g.* izal or iodoform. This material acts by capillary attraction, fluids being drawn from the interior and delivered into the dressings. In wounds designedly kept open, the gauze should be brought by light packing into contact with the greatest possible extent of the raw surfaces, in order to secure the rapid withdrawal of every drop of discharge. The increasing employment of capillary drainage in surgical work is tending to restrict the use of tubing, but the latter is undoubtedly of great advantage where the discharge is thick or has to be drained through a long fistulous track. A combination of both methods is often very serviceable, a tube being passed to the bottom and surrounded by gauze; or conversely, a wick of gauze may be threaded through an ensheathing tube,—a plan which facilitates subsequent removal of the drain. For a shallow wound, or one with walls but little separated, a narrow strip of gutta-percha tissue or green protective answers well. Satisfactory drains can also be made by knotting together a few strands of catgut or horse-hair: these can be readily placed in position with a probe or sinus forceps.

5. *Dressing.*—The objects in view here are to defend the wound against fresh infection, to absorb any discharges, and to maintain such a degree of equable pressure as will check oozing, obliterate "dead spaces," and generally support and protect the injured parts. Before applying the dressings it is well to finally cleanse and dry the skin around the wound, and then to squeeze out the last drops of moisture (blood, serum, or lotion), and to maintain the compression as the successive layers of material are applied. For the innermost dressing there is nothing better than gauze,—plain or charged with an antiseptic, *e.g.* izal, iodoform, double cyanide of mercury and zinc (Lister). This should well cover the wound, and a circular mound of it should surround the drain-exit, at which point more gauze is to be placed than elsewhere. Next follows a liberal supply of absorbent cotton-wool, a good form of which is the Gamgee-tissue introduced by the late Sampson Gamgee. This must extend well beyond the wound in all directions, as otherwise slight displacement would leave the part unguarded. It is often advisable, especially in the case of children, to fix the inner layer of wool to the skin by brushing collodion over its margins; or where extra support is desired, straps of adhesive plaster are most useful, and are strongly to be recommended in wounds of the abdominal wall, the strapping being cut broad and long. Unless the wool is sufficiently abundant, soakage through may occur and establish a path for infection, but the danger can be lessened by using an antiseptic wool, a course which should certainly be followed when copious exudation is expected. Waterproof material, as jaconet and gutta-percha tissue, placed outside the absorbent wool to intercept discharge coming through and turn it laterally, is not so much employed as formerly. The writer has found non-absorbent (fatty) wool useful for this purpose, and its employment is strongly advocated by Bloch of Copen-

hagen; it should of course be sterilised by heat. But the far safer plan is to promptly change the dressings that are soaked through. Lastly, to secure the other materials in position, and to exert through them the amount of pressure suitable for the particular case, bandages are applied. Domette is a satisfactory material for ordinary purposes; but where lightness is a desideratum, on the scalp for instance, gauze is to be preferred; and if unusual pressure is wanted in any situation elastic webbing is excellent.

6. *Provision for Rest and favourable Circulation.*—Unless wounded structures are maintained in due approximation, quick and firm union is impossible; and since mechanical disturbance, arising from muscular action or imparted from without, alters the relations of wound-surfaces, it inevitably tends to retard healing, and it may even undo what has been already gained in that respect. Moreover, it augments local irritation, and thus intensifies inflammatory reaction, with the consequence that the escape of fluid and cells is more rapid and abundant. If at the same time the covering dressings are displaced, the wound becomes an easy prey to infection. On various grounds, therefore, unrest is extremely prejudicial to repair, and must be prevented. The chief means to this end are posture, splints, slings and bandages; and these should be intelligibly made use of to meet the requirements of any case, according to the region injured and the special features of the wound, so as to secure the maximum of repose combined with enough relaxation to avoid tension. Confinement to bed is an ideal measure, but it is seldom available or submitted to except for moderate wounds of the lower extremity or severe ones in any situation. Besides saving the patient from undesirable movements, it moderates physiological activity in general, and conduces to regularity of blood-supply by keeping the cardiac rhythm uniform.

Closely connected with the consideration of rest are questions relating to the circulation. Retardation of the current in veins and lymphatics will seriously interfere with repair by inducing venous congestion and œdema. The utmost care must therefore be taken to avoid undue constriction or compression by bandages and splints. The omission of this precaution will also entail the risk of obstruction to the arterial supply, the effect of which would be starvation of tissues and probably gangrene of larger or smaller portions,—in particular, torn and contused fragments already seriously devitalised would certainly perish. Where the wound is situated in an extremity, elevation of the limb on an inclined plane sloping downwards to the trunk will promote rapid healing, combining as it does quiescence of the parts and most favourable conditions of circulation.

After-Treatment.—(A) *General.*—The reparative process is extremely susceptible to influences prevailing within and around a patient. In the busy routine of hospital out-patient practice these influences do not always receive the attention their great importance deserves, and in attending to details of dressing the surgeon may lose sight of that wider aspect of wound-treatment which may be expressed by the proposition that the growth of reparative tissue is subject to conditions governing the growth of normal tissue in childhood. Healing is powerfully influenced for good by such hygienic conditions as fresh air, sunshine, cleanliness, nourishing food, warmth, and cheerful surroundings, their combined effects being to invigorate the functions of nutrition and fortify the resisting power of the tissues against noxæ of all kinds. The readiness with which wounds will heal under favourable climatic conditions was strikingly illustrated in the recent South African compaign, the bright dry atmosphere to which the wounded were freely exposed benefiting the mental state as well as the physical. Too much stress cannot be laid on the value of a plentiful supply of fresh air. It is not only a prime necessity for enriching the quality of the blood, but it also dilutes and removes deleterious substances given off from the patient's body or accumulating around him from other producing sources.

The digestive system should be looked to. An accidental injury abruptly changes the habits of a patient's life, and the effects of the transition from full work to comparative inaction need to be thoroughly appreciated. It is a safe rule to give a dose of calomel and a saline purgative, partly for the sake of lowering the blood-pressure, but mainly to unload the bowels of effete material, the retention of which might lead to absorption of noxious substances (auto-intoxication), and thereby render the tissues more vulnerable to bacteria. While the patient is debarred from active muscular exercise he will require less food, especially animal meat, than a healthy person, *cæteris paribus*. The diet should consist of simple nutritious food-stuffs adapted to the digestive powers, and variously modified to meet any special indications. Alcohol is usually quite unnecessary, and had better be forbidden; in many cases it is responsible for the making of the wound and for concomitant digestive derangement, as well as other well-known complications which will demand appropriate treatment. In fine, the state of the stomach and other digestive organs should in no case be neglected.

When recovery from the anæmia attendant upon severe hæmorrhage is tardy, iron should be prescribed, and with this drug strychnine may sometimes be usefully combined.

Recognising the therapeutic value of contentment and cheerfulness, the surgeon will not overlook the importance of pleasant surroundings and genial influences in general.

(B) *Local.*—The redressing of wounds must be conducted on the same lines as the original treatment, the parts being fenced off with sterilised cloths, and no rule relating to the disinfection of hands, instruments, and materials relaxed. Exposure of the wound should also be as brief as possible. Assuming that asepsis has been secured in the case of a wound almost or quite closed, the dressings need not be changed for some days, since only harm can come of premature disturbance and uncovering: at the end of a week healing will have taken place, and stitches may be removed, dry gauze then being applied for a few days longer whilst the scar is consolidating.

Earlier attention will be required in various circumstances. Thus the appearance of discharge soaking through within twenty-four hours must be promptly met by packing over the spot a pad of absorbent wool secured by light bandaging; and it is advisable to change the whole dressing not later than the next day. Drainage tubes, too, should as a rule be taken out, and if the wound is satisfactory, left out, at the end of the first forty-eight hours. The onset of local pain or general febrile symptoms will also demand early examination of the wound; if, as is very probable, the trouble is due to pent-up exudate, this must be relieved by taking out one or more stitches and putting in a drain through a track made in the lower angle by a pair of sinus forceps or a director. Again, when a wound has been kept open by the free insertion of gauze, this should be withdrawn by gentle traction on the second day, and replaced by fresh gauze: to avoid bleeding, any very adherent portions of the material may remain a day or two longer.

So long as the wound shows no infective changes, a dry dressing is preferable. But if active inflammation ensues there must be no delay in freely opening up closed surfaces by cutting the stitches, and establishing a wide exit for discharges, which may be efficiently drained away by strips of gauze packed lightly within the wound and leading externally to layers of the same material wrung out of lotion. It is necessary to change wet dressings of this kind twice a day or oftener whilst suppuration is copious, if the septic condition is to be speedily checked. Whenever feasible, immersion of the wound and inner gauze in a bath of weak antiseptic has great advantages: it renders removal of the packing easier and less painful, and enables the lotion to penetrate into the recesses of the wound, where it displaces and dilutes the toxic substances. For cleansing infected parts that do not admit of immersion, irrigation under low pressure is serviceable, but forcible syringing must never be permitted. Continuous irrigation is sometimes of great value, the fluid being delivered as a slow stream flowing from a wick of gauze or worsted in contact with the highest point of the wound, the other end of the wick dipping into an elevated reservoir of lotion. Several precautions are to be taken,—the fluid must not splash upon the wound from a height, it must be kept warm, and it must be unirritating; normal salt solution answers the purpose very well.

With the decline of intense inflammation in a wound that has suppurated, the walls become covered with healthy granulations: these should now be purified as much as possible, and the opposite surfaces drawn together in order to hasten repair by secondary closure.

Such complications as stitch abscesses, sloughing of damaged tissues, and cellulitis, must be dealt with on the lines already laid down, the aim in all cases being the prompt and complete removal of harmful substances from the wound and its surroundings. It may be added that, in dressing septic wounds, the surgeon will find rubber gloves a great protection to his hands.

Wourali. *See* CURARA.

Wright's Coagulometer.—An instrument for determining the rapidity of coagulation of the blood.

Wright's Stain. — A methylene-blue stain for staining blood films; the red cells are made orange or pink, the nuclei of the leucocytes blue or dark lilac, the neutrophile granules lilac, the eosinophile ones pink, and the basophile ones deep blue, etc.

Wrisberg, Cartilages of. *See* LARYNX, EXAMINATION OF (*Laryngoscopy*).

Wrisberg, Ganglion of.—A ganglion in the superficial cardiac plexus.

Wrisberg, Nerve of.—The smaller sensory portion of the facial nerve; also, the cutaneous nerves of the arm.

Wrist-drop. *See* TOXICOLOGY (*Lead-Poisoning*); TRADES, DANGEROUS (*Plumbism*).

Wrist-Jerk. *See* TENDON-JERKS.

Wrist-Joint Injuries.

CONTUSIONS 543
DISLOCATION 543
FRACTURES 545
Colles' 545
Colles' Reversed 550
Epiphysary Separation . . . 551
Longitudinal Fracture of the Lower End of the Radius . . . 551
Fracture of Radial Styloid Process . 552
Fracture of Ulnar Styloid . . 552
Fracture of the Carpal Bones . . 552

See also AMPUTATIONS (*Wrist*); BRAIN, PHYSIOLOGY OF (*Functions of Cortex, Movements of Wrist*); FRACTURES (*Humerus*); OSTEO-ARTHRO-

PATHIES (*Wrist*); TENDON-JERKS (*Wrist-Jerk*); TOXICOLOGY (*Lead-Poisoning, Wrist-drop*); TRADES, DANGEROUS (*Lead-Poisoning, Wrist-drop*); WRIST-JOINT, DISEASES OF.

CONTUSIONS of the wrist joint and its neighbourhood are not uncommon, but most frequently fractures of the bones, either of the forearm or the carpus, result from them, and monopolise the attention of the surgeon.

In discussing certain of these fractures presently I shall consider contusion in some detail. Generally speaking, the covering of the bones is so closely adherent to them, and of so little thickness, that in simple injuries the contusions of the soft parts attract but little attention; in compound injuries the question of their vitality is of all importance in the determination of the treatment to be adopted in each particular case, whether it should be expectant, amputation, or excision.

In rare cases of contusion by soft and heavy bodies the bursal sac which invests the flexor tendons beneath the anterior annular ligament, and extends far above and below the limits of the ligament, suffers injury, and develops a traumatic bursitis, usually of moderate intensity, which yields in a short time to treatment by rest and cold lotions.

In former times, before the confidence in the safety of antiseptic surgery was developed, it used to be the daily practice to treat the tumour known as ganglion by a blow from the back of an octavo book with firm, smooth leather binding, and so to burst the cystic tumour and extravasate the fluid contents in the tissues around the tendon sheaths. This treatment, rough and ready, was in the majority of cases successful. I have, however, seen instances where, either from excessive force of the blow, or because the constitutional condition of the patient was unsuited to resist the effects of such injury, inflammatory troubles have followed, and have led even to the loss of the limb. A clean needle or tenotome in these days effects a more gentle and a safer division of the walls of these cysts.

The statistics of the medical and surgical history of the War of the Rebellion (1861-1865), American, are the most reliable that we can examine as a guide to the treatment of gunshot injuries of the wrist. In this war "the large majority of cases was treated by expectant conservative measures, with a comparatively low rate of mortality," 716 cases out of a total of 1496, or very nearly one-half, treated on expectant lines yielded a mortality of 7·9 per cent.

"To moderate inflammation by cold applications, by ice-dressings when available, to support the parts without constricting, bandaging upon a padded splint, to relieve inflammatory tension by free incisions as soon as suppuration took place, to remove all splinters and detached fragments, and finally, to institute passive movements of the fingers and hand at the earliest practicable moment, were the practical indications that were chiefly insisted on in the expectant conservative treatment of shot-fractures of the wrist." On the other hand, excision of the wrist recorded in 109 cases gave a mortality rate of 13·7 per cent; and again, amputations at the wrist for shot-injury gave as a result a death-rate of 10·6 per cent. In these days, with an antiseptic treatment efficiently carried out, and probably still of greater importance, with wounds inflicted by the bullets of modern warfare, conservative expectant treatment must be attended with even more favourable results, and the operations of excision and of amputation should show a proportionate decrease of mortality.

DISLOCATION of the wrist from injury is universally admitted to be extremely rare, but to be an injury which has real existence.

Writing in 1814 at the close of his short and original description of fracture of the lower end of the radius, Colles says: "During the last three years I have not met with a single instance of Desault's dislocation of the inferior end of the radius, while I have had opportunities of seeing a vast number of the fracture of the lower end of this bone."

We find in the lectures of Dupuytren, published some twenty years after the writing of Colles, which we have quoted, the following as the mature opinion of the great French surgeon:—

"Without denying absolutely the possibility of the dislocation backwards of the radio-carpal joint, although I have never met with it, one must admit, at least, that it is excessively rare, and that perhaps it never exists in consequence of a fall on the anterior part of the wrist."

When we read the writings of Desault, Boyer, and even Dupuytren, the difficulty that first meets us is the meaning that they attach to the term dislocation of the wrist. Is it dislocation of both radius and ulna from the carpus, or is it dislocation of the radius only or of the ulna only?

While each of the varieties here enumerated has been proved to exist, the actual number of possible dislocations must be increased by the varieties backwards and forwards, and possibly more. One must always bear in mind that dislocations uncomplicated by any fracture of the bones of the forearm or of the carpus are the rarest of all.

One of the very first to question the correctness of the assertion of Dupuytren, which would induce us to banish true dislocation of the wrist from the list of surgical casualties, was Voillemier. He says: "But still there is nothing else but the immense authority of this man, a species of fanaticism with which one accepts

his thought to set aside in one day the traditions of L. Petit, of Desault, of Boyer. · We are obliged to admit that these great masters of surgery, who have shown so much exactness in their observations, so much right sense in their judgments, and a spirit so simple in their descriptions, had all fallen into the same error, and have given the signs of a malady that they have never seen. Instead of waiting till facts could decide this question, an exaggerated enthusiasm, without appeal, judged it, and taxed as heresies all independent spirits who did not accept as an axiom the opinion of the surgeon of the Hotel Dieu."

With these observations Voillemier introduces his account of an accurately dissected dislocation of the wrist backwards. The category of such cases has grown since 1839, when Voillemier published his case. Some 70 fairly authenticated cases are now recorded, the greater number backward dislocations. Of the forward dislocations 7 at least have been proved by dissection.

I have never seen a recent traumatic dislocation in the living or dead; our collection contains no instance of the lesion except in association with fracture of the radius. I shall now quote an excellent description of the complete dislocation of the carpus:—

"Dislocation of the Wrist.[1]—W. W., aged twelve, was admitted 22nd March, having been knocked down by a cart a short time previously. He had two lacerated wounds of the scalp, and in connection with one of them a linear fracture of the posterior fossa of the base of the skull. There were no symptoms of brain injury. He complained of pain in the left wrist, but the parts around were so much swollen that the nature of the injury could not be made out. There were, however, two slight transverse abrasions on the fore and outer part of the limb about half an inch above the wrist joint. Evaporating lotions were applied and the swelling gradually diminished. The part then presented the following characters:—The whole of the hand was *displaced forwards* and slightly outwards; on the dorsal aspect of the limb the lower end of the radius and ulna formed a marked projection, the styloid processes, especially that of the radius, being very prominent, and the concave margin of the latter bone easily traced between them; anteriorly a somewhat pointed prominence could be felt in the middle line opposite the wrist joint. The deformity was easily removed by slight extension, but returned again almost immediately. There was no crepitus. Two straight splints were applied to keep the parts in position. On the eighth day after the accident the patient was seized with tetanus and died the following day.

[1] *Lancet*, 29th June 1878. Mr. Goodall's case, Birmingham General Hospital; recorded by Mr. Howard Lowe.

"*Dissection of the Wrist.*—The parts about the joint were much ecchymosed. The external lateral ligament was completely torn through. The anterior ligament was torn from its attachment to the scaphoid and semilunar bones, and presented a free border inferiorly between the styloid process of the radius on the outer side, and the cuneiform bone on the inner side—the ligaments uniting the semilunar to the cuneiform were torn through; the scaphoid and semilunar, carrying the hand with them, were displaced forwards, passing over the free border of the anterior ligament, which ligament came thus to intervene between these bones and the surface of the radius with which they articulate; the cuneiform was not separated from its connections, except those with the semilunar, and maintained its normal relations to the ulna and fibro-cartilage, though this part of the wrist joint was in a state of considerable flexion.

"*Remarks.*—This was a case of pure dislocation; there was no fracture of any of the bones, and no separation of the epiphysis of the radius. No clue could be obtained as to the direction of the force producing the dislocation, except from the position of the abrasions mentioned above, which seemed to indicate a force acting anteriorly on the lower end of the radius. The speedy return of the displacement observed was manifestly due to the fact that the dislocation was never really reduced, owing to the bones having passed in front of the anterior ligament of the wrist joint. The pointed prominence observed on the anterior aspect of the limb during life corresponded to the projection forward of the semilunar bone.

"Very recently the following concise and graphic account of a compound dislocation of the bones of the forearm forwards without any fracture makes the recognition of such injury essential, and is noteworthy for the success of its treatment.

"A Case of Compound Dislocation of the Wrist.[1]—A young man in emptying a cart of coal was lifting up the front end of it, when the horse quickly plunged forwards, whereby the weight of the coal and the moving cart was thrown with great force upon the palm of the right hand; as the elbow was fixed on the shaft a compound dislocation of the wrist resulted.

"When I saw the case I found the lower ends of the radius and ulna partially denuded of periosteum projecting 4½ inches into the palm of the hand. There was great hæmorrhage and considerable laceration of muscles and ligaments. The lower end of the radius and ulna were, in fact, stripped of all muscular and ligamentous attachments, and looked as if they had been scraped.

"The parts were washed well with a strong solution of creolin and the dislocation reduced under chloroform. The lacerated and torn

[1] *British Medical Journal*, 1st October 1898.

muscles, nerves, and tendons were brought together with catgut and the skin sutured. Free drainage was provided for, and the parts well dusted with boric acid and dressed with carbolised gauze, and the whole arm put into splints.

"The parts healed by first intention, and at the end of three weeks massage and passive movement were regularly persevered in with the happiest results—a perfectly movable joint with no deformity.

"WM. TIPLADY, L.R.C.P.E."

Newcastle-on-Tyne.

I quote these examples to prevent any error arising in the mind of any of my readers as to the reality of uncomplicated dislocations of the wrist joint.

THE FRACTURES OF THE BONES OF THE FOREARM

in the immediate neighbourhood of the wrist joint may be arranged as follows:—

Colles' fracture.

Colles' fracture reversed.

Epiphysary separation.

Fracture of the radial styloid process.

Longitudinal fracture of the lower end of the radius.

Fracture of the ulnar styloid.

Colles' fracture is commonly caused by a fall on the outstretched hand in walking or running; in falls from greater or less heights it occurs from the individual lighting on the hand or hands: in such falls as, for example, from the rigging of a ship to the deck.

I have seen the radii broken symmetrically. I have seen the injury symmetrical, too, as the result of the following accident:—A man was helping at the taking down of a builder's scaffolding; as he reached up his open hands to receive a heavy scaffold pole which was being lowered horizontally, the rope which let down the pole was suddenly let go when the pole was almost in his grasp. The sudden blow to the wrists broke the radii of both forearms. Again, I have seen the injury caused in one forearm thus: A surgeon driving in a low phaeton sought to save his knees from the kicks of his restive horse. To do so he stretched his arm and hand to lean on the leather dash-board of the carriage while he withdrew his knees as far as possible: in this attitude the horse struck the dash-board opposite to the point where the surgeon's palm rested. A Colles' fracture was the result. It is quite easy to produce the fracture in the dead body after the manner of Nelaton, experimentally, by sawing off the upper end of the ulna, disarticulated from the humerus, flush with the upper surface of the head of the radius. If the hand be placed prone on the table in full extension at the wrist, and a smart blow is struck with a mallet on the section of the ulna and the head of the radius simultaneously, the fracture takes place. In these varied ways the fracture which results in the living or the dead is seated within half an inch or thereabouts transversely in the radius above its carpal surface. In some examples the fracture may incline with slight obliquity from the dorsal to the palmar surface of the bone. One point is, I think, fully proved by the examination of a large collection of specimens which I have before me (120), that the break in the bone is never found to be as remote from the carpal surface as Colles located it. "This fracture takes place at about an inch and a half above the carpal extremity of the radius."[1] In making this statement Colles was probably led astray by mistaking the site of the springing of the dorsal tumour of the deformed wrist for the site of the fracture. At the time he wrote (1814) he had made no dissection of the injury.

Except in this one matter his account of the injury is unrivalled:—"The posterior surface of the limb presents a considerable deformity, for a depression is seen in the forearm, about an inch and a half above the end of the bone, while a considerable swelling occupies the wrist and metacarpus. Indeed, the carpus and base of the metacarpus appear to be thrown backward so much as on first view to excite suspicion that the radius has been dislocated forward. On viewing the anterior surface of the limb we observe a considerable fulness, as if caused by the flexor tendons being thrown forwards. This fulness extends upwards to about one-third of the length of the forearm, and terminates below at the upper edge of the annular ligament of the wrist.

"The extremity of the ulna is seen projecting towards the palm and inner edge of the limb; the degree, however, in which this projection takes place is different in different instances. If the surgeon proceed to investigate the nature of this injury he will find that the end of the ulna admits of being readily moved backwards and forwards. On the posterior surface he will discover by the touch that the swelling on the wrist and metacarpus is not caused entirely by an effusion among the softer parts; he will perceive that the ends of the metacarpal and second row of the carpal bones form no small part of it. This, strengthening the suspicion which the first view of the case had excited, leads him to examine, in a more particular manner, the anterior part of the joint, but the want of that solid resistance, which a dislocation of the radius forwards must occasion, forces him to abandon this notion, and leaves him in a perplexing state of uncertainty as to the real nature of the injury; he will, therefore, endeavour to gain some information by examining

[1] *Edinburgh Medical and Surgical Journal*, vol. x., 1814, p. 183.

the bones of the forearm. The facility with which (as before noticed) the ulna can be moved backward and forward does not furnish him with any useful hint. When he moves his fingers along the anterior surface of the radius he finds it more full and prominent than is natural; a similar examination of the posterior surface of this bone induces him to think that a depression is felt about an inch and a half above its carpal extremity. He now expects to find satisfactory proofs of a fracture of the radius at this spot. For this purpose he attempts to move the broken pieces of the bone in opposite directions; but, although the patient is by this examination subjected to considerable pain, yet neither crepitus, nor a yielding of the bone at the seat of fracture, nor any other positive evidence of the existence of such an injury, is thereby obtained. The patient complains of severe pain as often as an attempt is made to give to the limb the motions of pronation and supination. If the surgeon lock his hand in that of the patient and make extension, even with moderate force, he restores the limb to its natural form, but the distortion of the limb instantly returns on the extension being removed. Should the facility with which a moderate extension restores the limb to its form induce the practitioner to treat it as a case of sprain, he will find, after a lapse of time sufficient for the removal of similar swellings, the deformity undiminished. Or, should he mistake the case for a dislocation of the wrist, and attempt to retain the parts *in situ* by tight bandages and splints, the pain caused by the pressure on the back of the wrist will force him to unbind them in a few hours; and if they be applied more loosely, he will find at the expiration of a few weeks that the deformity still exists in its fullest extent, and that it is now no longer to be removed by making extension of the limb. By such mistakes the patient is doomed to endure for many months considerable lameness and stiffness of the limb, accompanied by severe pains on attempting to bend the hand and fingers. One consolation only remains, that the limb will at some remote period enjoy perfect freedom in all its motions, and be completely exempt from pain; the deformity, however, will remain undiminished through life. . . . Let the surgeon apply the fingers of one hand to the seat of the suspected fracture, and locking the other hand in that of the patient, make a moderate extension, until he observes the limb restored to its natural form. As soon as this is effected let him move the patient's hand backward and forward, and he will, at every such attempt, be sensible of a yielding of the fractured ends of the bone, and this to such a degree as must remove all doubts from his mind."

FIG. 1.—From a cast of an ordinary Colles' fracture.

Such is the description of the injury as Colles gives it. After an interval of some twenty years Dupuytren published his account, evidently without knowledge of the previous description of Colles, and suggests the fracture by crushing of the lower fragment as the leading type of the injury.

Every surgeon admits, I believe, the value of the researches of Professor R. W. Smith and of Voillemier in the pathology of these injuries, which have served to correct and complete the descriptions of Colles and of Dupuytren.

Since their views have been published we find authorities ranging themselves on opposite sides, according as they adopt the non-impaction theory of Smith, or the opposing doctrine of Voillemier, that all these injuries are fractures by penetration. Of late the number of recent dissections have forced most writers to admit the occurrence of each form. We have ample proof that the simple transverse fracture without impaction, the impacted, and the fracture *par écrasement* with a shattered lower fragment, are all possibilities. Intimately connected with the theory of impaction is the explanation of the variability of the symptom so much relied on in the diagnosis: crepitus. Yet any one who will take the trouble to investigate the statements contained in the accounts of recent cases which have been submitted to dissection may find that this symptom has been absent in each form of the injury. We cannot, therefore, rely on its absence as the proof of the existence of impaction in any given case, nor are we for the same reason to abstain from attempting reduction of the deformity for fear of undoing the impaction, or again rely on it as a safeguard against displacement, and so adopt a lax treatment.

In the study of the pathological varieties of the fracture one must be struck, in the examination of a large series of specimens, with the differences in the degrees and kind of displacement presented by individual cases.

Clinically the same varieties are observable. In the ordinary run of cases the diagnosis based on the familiar inverted spoon deformity (Fig. 1) is easy, so that to these cases the description of Pouteau applies: "There is, perhaps, no fracture more easy of recognition at a simple glance." The words of Voillemier in his reference to the examples which occur with little or no deformity are worth notice: "But one must be forewarned that in certain cases—and these are not very rare—of penetration but little

marked, fractures by tearing of the bone *par arrachement*, it is possible that there may be no change in the axis of the limb, no shortening, no mobility between the fragments, no crepitation, and only pain and swelling around the wrist; in fine, all the symptoms of a sprain."[1]

Here the author admits a group of injuries sufficiently common, in which impaction is absent or inappreciable—in which, too, crepitus and deformity are absent. If such cases be overlooked, as they too commonly are, the most extreme deformity establishes itself in a short time, as I have more than once observed. These facts are certainly inconsistent with the theory that deformity, the result of displacement, is solely due to impaction, and that muscular action takes no part in its production.

In opposing Voillemier's views Smith falls into the opposite error, in almost absolutely denying the occurrence of impaction. He says: "I am inclined to believe that the doctrine of fracture with penetration is untenable." He says, indeed, in another passage: "Until, therefore, the result of the examination of recent specimens can be adduced in support of the theory, I shall be inclined to believe that impaction is only apparent."

The photograph (Fig. 4) of the specimen on which Professor Smith based his rejection of Voillemier's theory of impaction shows the characters of a well-marked fracture, with displacement of the lower fragment, as in the group above referred to, recognisable at a simple glance, and without any penetration or impaction of the upper fragment into the lower. In such cases the lower fragment is displaced backwards, rotated on its own transverse axis backwards, and the whole piece is moved to the radial side with displacement of the radial styloid towards the upper extremity of the radius (Fig. 3). The degree of each of these displacements, more or less present in each specimen, varies. In fractures caused by great violence, such as a fall from a high building, the backward displacement may predominate, and the lower fragment may clear the upper; with it the hand and the wrist joint undergo extreme displacement, while the ulna, set free from its connection to the radius and hand alike, becomes dislocated through the integuments towards the inner border of the hand. In other specimens we see the rotation of the lower fragment backwards on its transverse axis, the salient feature of the specimen (Fig. 3, A). In some not very rare specimens the extremely limited development of all three displacements renders the deformity so small that even expert anatomical eyes may fail to appreciate it until close comparison is made with the macerated bone of the opposite side (Fig. 3, C). Such an oversight is made in a well-known osteological text-book,[1] in which an united Colles' fracture, with the minimum deformity, is represented as the normal bone. In such specimens the displacement most constant in its development is that of the radial styloid towards the upper end of the bone. This fact is of great importance clinically, because the altered relation of the radial styloid renders the line drawn from it to the ulnar styloid less oblique than in the uninjured limb, and by the appreciation of this change the diagnosis may be made certain in the living, in cases the description of which I have quoted above from Voillemier's paper, in which the most evident features of deformity are absent.

FIG. 2.

In discussing the subject of impaction, Voillemier puts in the forefront of his argument the constancy of the three lines of compact tissue seen in the antero-posterior section of united fractures (Fig. 4). He admits four possible modes of impaction: 1. The penetration of both walls of the upper fragment into the tissue of the lower without its being burst asunder; 2. The same mode of penetration, with crushing of the lower fragment—Dupuytren's fracture *par écrasement*; 3. The bending or hinging, so to speak, of the anterior walls, with impaction of the upper into the lower fragment posteriorly; 4. The reciprocal pene-

[1] *Clinique chirurgicale*, par L. Voillemier, 1842.

[1] Holden, *Human Osteology*, 6th ed., p. 56.

tration of the fragments, the upper into the lower behind, and the lower into the upper in front. Of these possible forms of impaction he believes the third to be the most common, founding his opinion, apparently, on the observation "that the impacted line is never double," an observation in which Smith agrees.

I am able to show that exceptions occur to this rule; further, that comminution is more common than Smith has estimated it to be, and that the paths of the fractures which occur in the lower fragment present a remarkable constancy in position. If I rightly interpret these

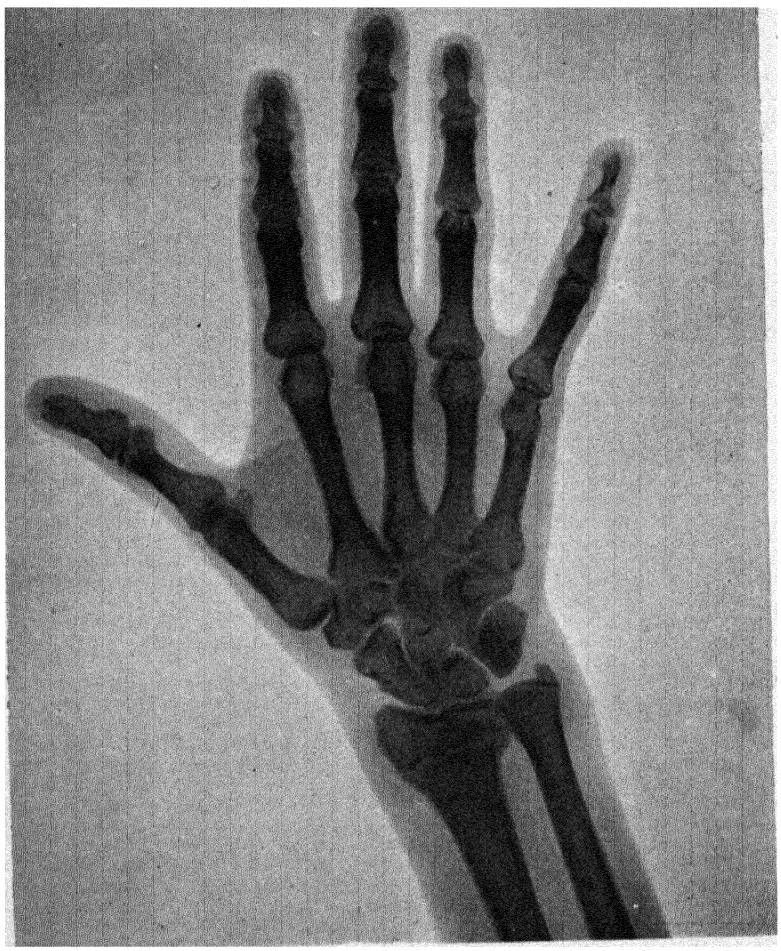


FIG. 2a.

appearances, the conclusion must be arrived at, that in comminuted fractures reciprocal impaction is the most common form of the accident.

In estimating the frequency of comminution of the lower fragment I apply the following test to the collection before me, which has now reached the number of 120—all Colles' fractures, transverse in the direction of the fractures. Some are recent injuries, a couple experimental, the vast majority old united fractures, in all the lower fragments displaced backwards, with more or less distortion of the carpal surfaces by rotation of the fragments backwards, and the most constant character, elevation of the styloid processes. In estimating the frequency of comminution of the lower fragment I accept as proof the extension of fracture through the articular cartilage of the carpal surfaces. These fractures are, of course, evident in recent specimens taken from patients who have died of other injuries. The traces of the fractures in the carpal surfaces are no less evident in united fractures, for all the world knows that the fractures of articular cartilage remain as permanent marks on the surfaces. Only one change in a few specimens can mask them—the supervention of chronic rheumatic arthritis in the articulation. Applying this test it is certain that I omit specimens which are examples of impaction, but in which the lower fragment has not been split through. In tracing these lines in united specimens a remarkable constancy in their course is appreciable, a constancy which suggests at once a constancy in their cause.

In twelve specimens the minimum fracturing of the carpal surface occurs (Fig. 5). A line of fracture passes in these through the articular cartilage of the ulnar facet, and turning into the carpal articular surface passes to its dorsal margin, so the posterior angle of the carpal surface next to the ulnar articular surface was broken off (Fig. 5, A, B). In six specimens the line of fracture, similar to that in the preceding twelve, exists, but there is added to it a line extending along the carpal surface further outwards in some, forming what has been called Barton's fracture (Fig. 5, C, D). In twenty-two specimens the lines seen in the two preceding groups are present, but they branch forward towards the anterior margin of the carpal surface (Fig. 5, E). In four of the five recent fractures obtained from cases fatal by other injuries, the same pattern of fracturing is seen; these fractures, *par écrasement*, are then only excessive extensions of the impacted fracture. In one specimen these fractures are associated with dislocation of the lower extremity of the ulna.

Of fractures which show no trace of lesion of the carpal articular surface of the radius there are seventy. Some of these, no doubt, presented some degree of impaction, but the majority were free from the complication.

There appears, then, sufficient ground for the assertion that the mode of comminution is constant, and that it results from the impaction of the upper into the lower fragment, taking effect first and chiefly on the side of the fragment next the ulna.

A further examination of these specimens shows that, where the fractures remain sufficiently distinct in outline to trace their details exactly, the impaction of the fragments is reciprocal (Fig. 7); that while the posterior surface of the upper pierces the lower, the anterior of the lower penetrates the upper, it may even lead to its splintering. This conclusion is at variance with the opinion of Voillemier; but I believe the explanation of the discrepancy between us is, that hitherto the examination of comminuted specimens has not been made with sufficient care. I believe that in many (Fig. 2) fractures no impaction whatever occurs, and again that in many injuries, Voillemier's third variety, the bending of the anterior wall with posterior impaction occurs. I cannot resist the conclusion to which this collection leads, that in comminuted fractures, which occur in proportion to injuries without secondary fractures of the carpal surface almost in the ratio 5 : 7, the characters are constant and the result of reciprocal impaction of the fragments.

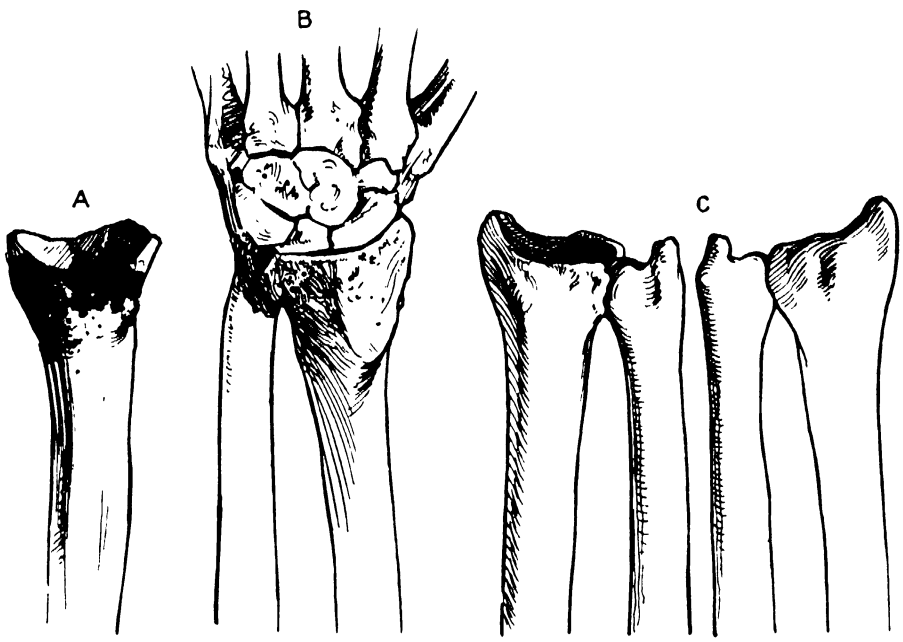


FIG. 3.

The following appear to be the chief complications of Colles' fracture so far as the specimens preserved in our collection illustrate them:—

1. Necrosis of the lower fragment.
2. Fracture of the shaft of the radius higher up.
3. Fracture of both bones of the forearm, also higher up.
4. Fracture of the styloid process of the ulna.

1. The fracture in which necrosis occurred is entirely exceptional; the injury was in all respects of the ordinary type, without excessive deformity or any wound. On the patient, of thirty-five years of age, resuming work at the close of the treatment, which lasted about five weeks, the limb suddenly swelled and abscesses formed, leading finally to amputation.

2. The fractures of the radius alone, higher up, are seen in two instances where the usual distortions of such injuries are absent, or at least very slightly marked; phenomena which I have myself noticed in this complication occurring in the living, the Colles' fracture, so to speak, taking precedence in establishing its deformity, while the fracture of the shaft was discoverable chiefly by its pain and local crepitus.

3. In another specimen, where both bones are broken, the radius very high up, the features of the fracture suggest its having occurred independently of the Colles' fracture.

4. In the instance of fracture of the ulnar styloid the characters of this complication are well seen (Fig. 2). These specimens were more numerous than the collection shows, for I have, in dissecting some now not characteristic specimens, noticed this lesion, but the little fragment of bone is lost in maceration or after the specimens dry. I am confident that in all the fracture occurs as in the examples preserved—a fracture of the styloid caused by traction on the lateral ligament of the wrist, free of direct relation to the attachment of the sacciform ligament; that this fracture unites in the majority by ligament only with very insignificant deformity. I notice these points merely to give expression to the opinion I hold as to this complication, that it is of practical importance only as explaining the occasionally severe pain observed at the position of the ulnar

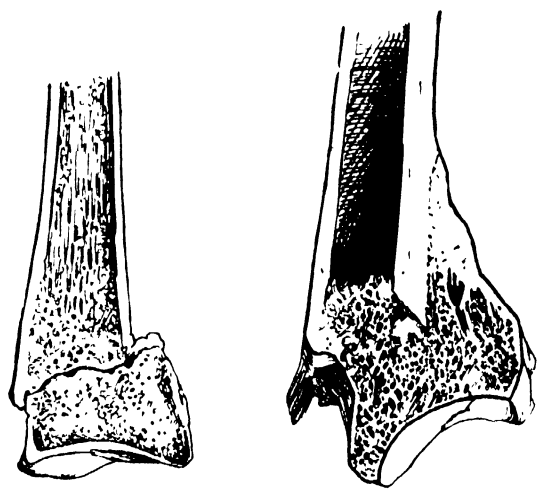


FIG. 4.

styloid in recent injuries, that it is quite unimportant in regard to the deformity or other leading phenomena of Colles' fracture.

The clinical complication of greatest gravity which I have seen in dealing with Colles' fracture is gangrene of the limb. I have twice seen this accident occur in cases which presented other grave injuries; in one I think over-severe efforts to diminish the deformity caused by the fracture precipitated the gangrene. In the other there was no special sign to give warning of the onset of gangrene, only the unusual complaints of pain in the limb on the evening and night of the second day.

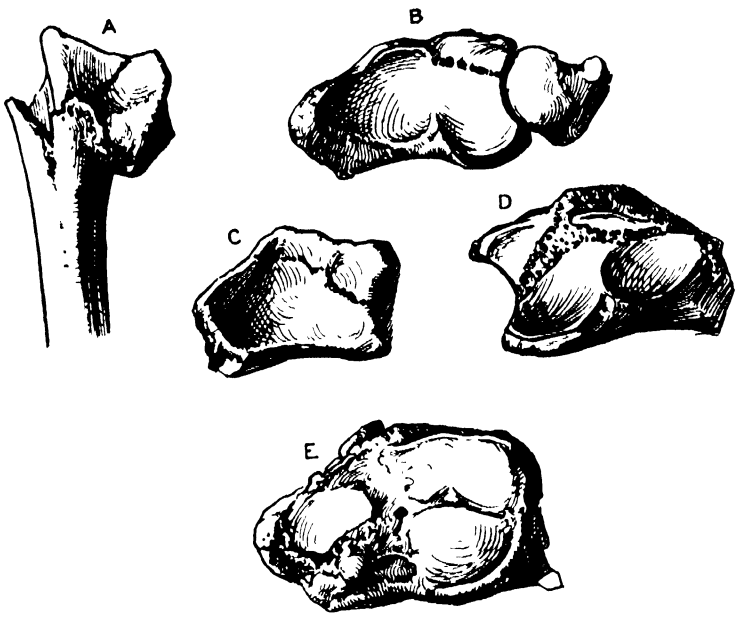


FIG 5.

The treatment of Colles' fracture should be directed, in the first instance, to the reduction of deformity as completely as it can be effected by the guarded use of traction. One must bear in mind that nearly one-half the cases are impacted fractures, and that it must in consequence be impossible to fully restore the form of the broken bone. The invariable existence of deformities in the examples preserved in our collection, and in every other that I have seen, goes to prove that one never succeeds in a perfect reduction. The reduction is a very painful proceeding for the patient, and I have many times found patients absolutely refuse to allow it to be carried out to the full degree. Many patients, particularly males, become faint when extension is made, and if the surgeon is wise, instead of suspending the reduction because of the patient's fainting, he will at once complete the reduction as far as he fairly can while the fainting lasts. I think if the injury the surgeon is called to treat is quite recent, he should have at his hand two flat wooden splints, well padded, long enough to reach from just below the elbow joint to the tips of the fingers. A pad of wool to be applied to the palmar surface of the forearm, thickest beneath the actual seat of fracture, should be placed on the anterior splint. As the reduction is effected the palmar splint is put in position, while extension is kept up by Colles' grip, then the dorsal splint is applied, and the apparatus fixed by a roller turned firmly but without excessive strictness. In a day or two, as soon as the degree of swelling of the soft parts has been estimated, and is seen to be on the decline, a more agreeable support for the limb is made of light poroplastic felt, moulded to the front and back; this can be shortened by cutting away the dorsal and palmar supports of the fingers and thumb, and by reheating, adjusted so as to fit the forearm and hand as the swelling subsides. Or instead of this material the limb may be lightly put up in plaster of Paris. Three or four weeks of support to the limb is best, and, as the fingers are freed by shortening of the splints, their motion early comes into play.

Colles' Fracture Reversed.—Of the rare injury, fracture of lower end of the radius, with displacement of the lower fragment forwards, the collection of the School of Physic contains seven specimens, with a cast of an eighth which is in the R.C.S.I. collection; of these, two agree with the description given by Professor Gordon; two, on the other hand, agree with the figure published by Voillemier. The relative number of these specimens proves the inapplicability of the name applied by Professor Gordon to this

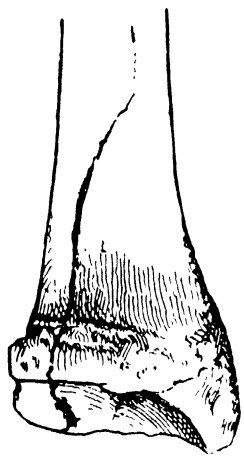


FIG. 6.

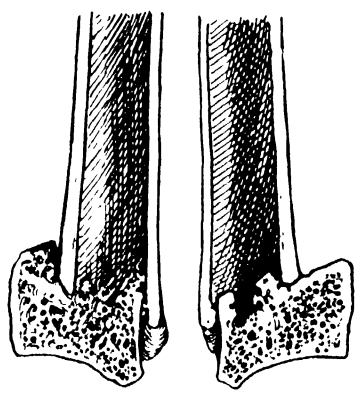


FIG. 7.

fracture, "articular," for we see here the fractures as remote from the joint as in ordinary Colles' fractures.

We possess also the original cast published by Professor Smith, and now casts of two cases observed in practice by myself. Of these specimens two may be regarded as conformable with Professor Gordon's articular fracture, being

fractures which pass obliquely from the dorsal margin of the carpal surface to the palmar surface of the shaft of the radius, three-quarters of an inch from the end of the bone. Of the remaining five, three were impacted with fractures of the carpal surface, after the manner of ordinary Colles' fracture; two presented no signs of such impaction.

The following is the history of an example of the injury observed by myself:—

A jockey, aged nineteen, was backing for the first time a three-year-old filly; she bucked and burst the girths, and sent saddle and rider into the air. The rider fell to the ground, striking first on the back of his right hand. I saw him the day following. The injury had been readily recognised by the deformity, the facility of reduction, and the distinct osseous crepitus felt on the reduction; immediately on the relaxation of the reducing force the deformity recurred. I took the patient into hospital and verified the details, and treated the patient until union was obtained.

It would seem, then, that Rhea Barton, and Mr. Callender, and Professor Smith were hitherto the only observers of the clinical details of the injury, and that the pathological facts bearing on it were not much more plentiful here or in America. I need not then apologise when I record the recent injury taken previous to reduction, and a series of eight pathological specimens obtained, one from the museum of the Royal College of Surgeons, to which it was presented by Mr Swan in 1885, and the seven which I have myself procured from the anatomical department of the School of Physic in Ireland. I think we may then fairly reject the conclusion arrived at by M. Lecomte, who states his opinions in a series of dogmatic assertions, of which this is the first:—"All the fractures of the inferior extremity of the radius are produced by a single and the same class of fall—falls on the palm of the hand."

It is, I think, clear that the reversed Colles' is the result of a fall on the back of the hand.

Epiphysary Separations.—The next group of our series includes two specimens of epiphysary separation, with which I have also the casts of the limbs of the patient. The third specimen I place in this group with less confidence, but I will presently state my reason.

The two recent specimens are taken from the limbs of a boy who died a few days after a fall from a height on his head, the head injury proving fatal.

The casts in these cases (Fig. 8, A) contrast, as many similar casts in our collection do, with the representations of the deformity (Fig. 8, B) in Colles' fracture, in the transverse features of their dorsal and palmar projections, and in the folding of the skin in the palmar depression, and again in the absence of abduction of the hand. If we add to these the easy reduction of the fracture and the facility of obtaining crepitus, and, still more remarkable, the facility of maintaining the reduced fracture in place, phenomena all long since pointed out by Smith, which I may state I have frequently observed and verified myself, there are, I think, ample grounds for questioning the recent statement of Professor Macleod, that "there are no distinctive signs by which a separation of it (the radial epiphysis) can be differentiated from an ordinary Colles' fracture." The united fracture I place in this group is remarkable when compared with all or any specimen of the collection of Colles' fractures, for, while there has been clearly a fracture at the usual seat, it alone presents the feature of a styloid process bearing its normal relation to the ulnar styloid, and a carpal surface almost without maldirection. There are faint traces of an ulnar fracture just

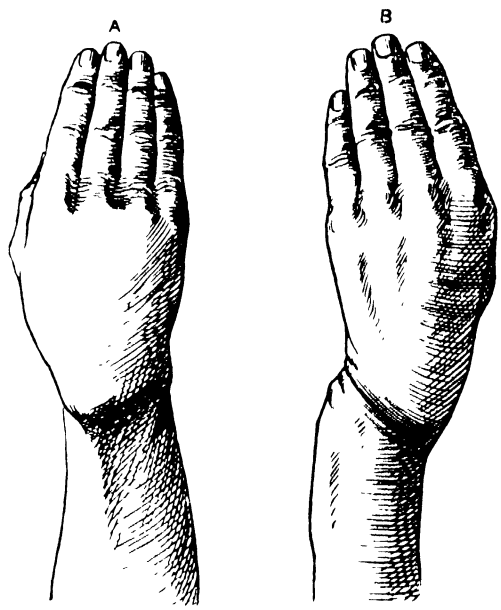


FIG. 8.

above its epiphysis, as often occurs with the radial detachment. The recent specimens, I should note, present fractures confined to the radius. On one side the lower fragment is comminuted; on the opposite, simple; a little scale of bone is detached from the diaphysis on the side which shows the comminution.

Longitudinal Fractures of the Lower End of the Radius.—I shall now briefly examine the longitudinal fractures of the lower end of the radius, and, I hope, show that they result from causes other than falls on the palm of the hand. The leading case which bears on this question is that recorded by Bigelow.

The catalogue of the Warren Museum contains this description of the specimen, 1035:—"The lower extremity of the radius, showing a stellate crack of the articular surface and extending upwards for more than an inch. From a patient who died of other injuries. At first there was only a complaint of lameness as from a sprain,

but after some days there were swelling and tenderness which, from their persistence, led Dr. B. to diagnosticate a stellate crack of the bone as a probable result of the fall, and of which he had met with a similar case two years previously. Dr. B. remarked that the bones of the wrist would act as a wedge upon the concave articular surface of the radius, and that this case would explain the persistence of some cases of sprained wrist." Case published in the *Medical Journal* (vol. lviii. p. 99), with a figure, and copied with the figure in Dr. F. H. Hamilton's work on *Fractures*, 1848.

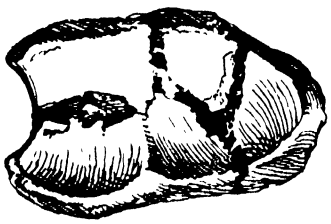
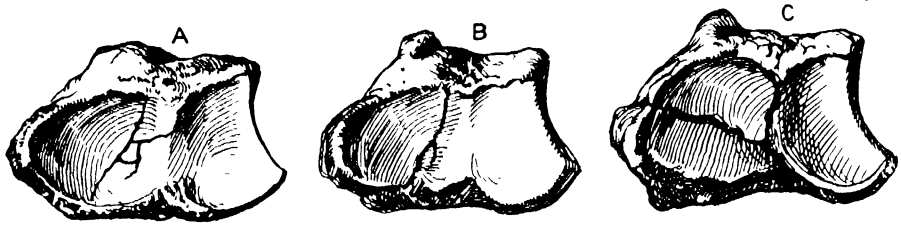


FIG. 9.

The details of the injury given here are very meagre, and leave us in doubt as to whether the patient fell on the palm of the hand or not. From the description and figure it appears that there was not any transverse fracture of the lower end as in Colles' fracture, and that the fissures were not, therefore, such as occur in the articular surface in nearly one-half of the cases of Colles' fracture.



IG 10

A series of radii which belong, one to the museum of the Royal College of Surgeons, and five to the museum of the School of Physic, present fissures that occurred during the lives of their owners. The earlier specimens that I obtained showed fissures of the articular surface only, and occupied my attention because of the difficulty in explaining their origin. This specimen (Fig. 9), placed in the museum of the college by the late Dr. Richardson, was the first which I saw that helped me to a solution of the question; it was damaged by the hand and forearm being caught in machinery, probably a cog-wheel; such is the note on the label.

I at once tried if I could produce such fractures on the dead bone. If one wraps a towel round the end of the dry bone, and presses with the heel on the carpal end of the radius as it lies on the ground, the bone yields and crepitus is felt; with the lighter degrees of pressure it is possible to break the bone only in the concave surface of the articulation, and to produce lines identical with those of these macerated specimens, strictly limited to the articular surface (Fig. 10, A, B, C). A greater degree of force extends the fissures upwards in the compact wall of the bone. If a man be trampled in a crowd, or if a weight of any kind falls on the wrist, such a damage can readily happen, still more if a wrist, as we often see, be rolled in machinery. It seems more rational to look for the cause of these fissures in such injuries than to attribute them to the wedge action of the carpal bones.

The experiment I have described is best made on bones in which maceration has removed the articular cartilage; in these the fissures of least degree will be seen in the articular layer of bone, but would be concealed in the bone covered by cartilage. This is illustrated by one of my specimens, where the cracks are evident in the shaft on both palmar and dorsal sides of the bone, and are traceable into the cartilage at each margin of the joint, but have not torn it.

Fracture of the Radial Styloid. — Of the fracture of the radial styloid I can only say that this specimen (Fig. 11) is a perfect example, remarkable for the downward displacement of the fragment, which has caused elongation of the outer border of the bone, probably as we see the inner malleolus displaced by traction of the internal lateral ligament of the ankle joint. I have never recognised the injury in the living, and this united fracture is the only example I have seen.

Fracture of the ulnar styloid occurs in a considerable number of cases of ordinary Colles' fracture in consequence of the strain communicated to it by the internal lateral ligament of the wrist. It is not unusual to find the fracture in the X-ray photographs of the injury.

The interest of the injury is derived from the fact that pain in position of the fractured process is so constant an attendant on the ordinary fracture.

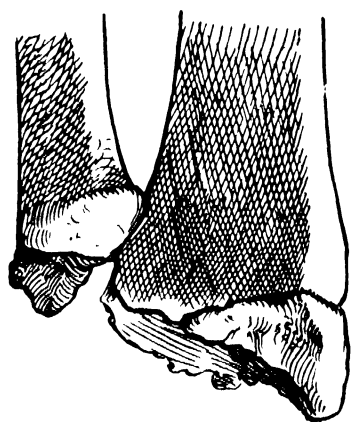


FIG. 11.

Fractures of the Carpal Bones. — Simple fractures of the carpal bones are of very rare occurrence, but as the use of the Röntgen rays becomes more general their great rarity will probably disappear. The scaphoid is the bone most often broken. Next to this is the semilunar, but were it of any practical advantage a complete list might be made out. Direct blows may produce any of these fractures. The cause, as far as I know, which breaks the scaphoid is over-extension of the wrist, as in the following

example: — A man of middle age and full strength was lowering a cask of pork into a cellar, letting it down a sloping plank. The cask proved too heavy for his hands pressed against it, and fell in spite of his resistance. At once he felt sharp pain in his wrist and noticed a projection at the back of the joint. He was presented to me next day in the accident room of Sir Patrick Dun's Hospital. I concluded that the tumour was either part of the scaphoid broken off and displaced, or there was partial dislocation of the bone unbroken. The small size of the tumour suggested that the case was one of fracture. I reduced the projection with a little manipulation, and when reduced there did not appear to be any tendency to recurrence of the projection. I did not get crepitus in any part of the manipulation.

I put a compress over the dorsal aspect of the wrist, and the next day had a skiagraph taken which shows the fracture very distinctly. I have never since seen the patient, so that I conclude he is free from trouble.

I have placed in our collection a fragment, nearly one-half of the scaphoid, which was given to me by my friend Dr. H. Fitzgibbon. He obtained it in this way:—A man had had his hand and wrist jammed in the cogs of a machine. The contusions behaved kindly for a time, but on the man's attempting to use the limb a swelling appeared at the anterior aspect of the wrist, which presently suppurated, and on the abscess being opened the detached and necrosed fragment of the scaphoid presented in the wound and was removed. In falls from a trapeze, in which the individual comes to the ground on his knees and over-extended wrists, the scaphoid may be broken. Of this injury I have representations of the Röntgen rays in two cases. Our collection contains a single example of the semilunar bone broken in several places without any lesion of the other bones either of carpus or forearm. One specimen of fractured cuneiform bone exists in Dublin; the cuneiform which corresponds to the radius represented in Fig 9: a cog-wheel injury.

I know of a single example of fracture of the pisiform bone (Stimson, 2nd edition, p. 666).

It would appear from the facts recorded with regard to the fractures of the bones of the first row of the carpus that union does not readily take place in this injury. As yet I know no example of osseous union having been observed.

Wrist-Joint Diseases.

1. Tuberculosis 553
   Symptoms 553
   Pathological Anatomy . . 554
   Treatment 554
      Medicinal 554
      Operative 554
2. Pyogenic Diseases 555
3. Gonorrhœal Arthritis . . . 556
4. Chronic "Employment" Arthritis . 556
5. Arthritis Deformans . . . 556
6. Charcot's Disease 556
7. Syphilis (*vide* Joints).
8. Rheumatism (*vide* Joints).
9. Tubercular Teno-Synovitis . . 556
10. Pyogenic Teno-Synovitis . . 557
11. Acute Teno-Synovitis . . . 557
12. Chronic Teno-Synovitis . . . 558
13. Tubercular Osteomyelitis of the Metacarpal Bones . . . 558

Tuberculosis.—The wrist joint holds a position midway between the shoulder and the elbow as regards its liability to tuberculosis. The disease in the elbow accounts for about 7 per cent of all joint tuberculoses, that in the wrist for 5 per cent, and that in the shoulder for 1½ per cent. This fact of its *comparative* rarity, coupled with the very insidious onset which characterises the disease in this joint, often leads to a delay in recognition of the condition and consequent serious permanent damage to the joint.

Two features are worthy of remark in this disease. Of all joint tuberculoses that of the wrist is perhaps the one most specially liable to be associated with phthisis. And the second point is that, though it occurs in children, it is essentially a disease of adult life; many patients suffering from it are no longer young, and some of them are old. I amputated the hand in a severe case in which the patient must have been between sixty and seventy years of age.

Symptoms.—Swelling is an early symptom. It is more marked on the dorsal surface, but even on the palmar surface comparison with the other wrist shows that the normal hollows are filled up; there is "a fulness and smoothness of outline, both on the palmar and dorsal aspects, which, even when it is slight, is very characteristic" When the disease has existed for some time the carpus may be found displaced somewhat forwards; the wrist slightly flexed, or the forearm, hand, or fingers in a straight line; the thumb applied to the hand, and the fingers held close together, though in some cases they may be widely separated. Atrophy of the muscles of the forearm is usually found early and well marked. With the flat forearm, the bulging, thick wrist, and the thin, tapering, glistening fingers stuck together, the disease presents a notable and unmistakable picture. Flexion and extension may seem at first sight to be unrestricted in an early case, but even at the beginning of the disease the limits of these movements will be found on careful examination to be narrowed. The same may be said of pronation and supination, and of course later in the disease only slight movements are possible. Pain is often absent for a considerable time, but in the later stages may be extreme.

Tenderness will be found early over the posterior and outer aspect; while grasping the fingers and forearm, and pressing the bones of the wrist together—care being taken neither to flex nor extend the joint—may elicit pain and indicate a correct diagnosis at an early stage of the disease. Grasping each finger in turn, too, and pressing it and its metacarpal upwards, so as to "crowd" the corresponding carpal bones, may give some indication of the position of the diseased areas.

Lateral mobility is a very important symptom. It is due to softening of the ligaments by the disease, and is quite an early symptom. It is made evident by grasping the forearm and the hand while they are in a straight line, and moving the hand laterally. The symptom is virtually pathognomonic of carpal tuberculosis.

As the disease progresses the joint becomes so insecure that the patient supports the fingers with the other hand. Abscesses may form, the resulting sinuses opening more frequently on the dorsal than on the palmar surface.

Cause and Pathological Anatomy (*see* JOINTS). —Invasion by the tubercle bacillus is often determined by some minor trauma, such as a sprain or over-exertion. Owing to the number and small size of the bones in the joint it is difficult to decide whether the disease commences more frequently in the bone or synovial membrane, but probably most cases are primarily synovial. Tillmanns holds the opposite view. Primary osseous deposits are found in the end of the radius, and occasionally in the second and third metacarpal bones. Sometimes the joint is invaded from a tubercular teno-synovitis of the neighbouring tendons. The ligaments are early invaded and softened.

Diagnosis.—The disease is to be distinguished from tubercular teno-synovitis and from arthritis deformans. In teno-synovitis there is indeed a chronic bossy swelling, but pain is conspicuous by its absence, and the movements of the wrist and of the fingers continue entirely, or almost entirely free. In arthritis deformans the atrophy of the forearm is not comparable with that in tuberculosis, and later all mobility is absent.

Treatment.—The means of treatment at our disposal are immobilisation by a splint, Bier's congestive bandage, iodoform injections, and operative procedures.

(*a*) *Immobilisation by a Splint.*—In this connection three things must be remembered:—Firstly, that stiffness in the fingers is apt to be produced by adhesions forming round the tendons passing over the joint. Secondly, that in a normal hand the grasp of the fingers is weakened if they and the wrist are in the same line with the forearm,—to get the full power the wrist must be partly extended; therefore a wrist anchylosed in a straight line or in flexion will have the use of the fingers impaired, even though the tendons may be quite free. Thirdly, the thumb must not be allowed to become stiff, applied to the hand, where it will be useless, but must be free to drop. Lister's well-known splint is the best for the purpose. It reaches from the elbow to the heads of the metacarpals, has a pillow of cork fixed at the lower end, and is of a breadth only to support the metacarpals of the fingers. It is applied to the palmar surface, the cork pillow bends back the wrist while the fingers are left free for movement. The thumb drops over the side of the splint, which should be provided with a small support on its under surface, so that the thumb may hang free when the arm is laid down, as, *e.g.*, during the night. To prevent it slipping round this splint should have a vertical piece of wood on one or both sides near the elbow. Rest maintained in this way may alone suffice to produce an absolute cure; but the splint must be worn for months.

(*b*) *Bier's Congestive Bandage.*—If improvement is not marked under treatment by a splint, the congestive bandage and injection of iodoform may be added. If needful the fingers and hand may be bandaged in the ordinary way, the wrist is left uncovered, and immediately above it the forearm is bandaged tightly with elastic webbing applied over a protecting layer of lint, so as to produce considerable venous congestion. The fingers should be watched to ascertain that the bandage is not too tight. It may be taken off for six hours each day, or may be left on continuously for weeks. I have seen remarkably good results following this treatment in disease of the wrist and ankle. The splint may be used at the same time.

(*c*) *Injection of Iodoform.*—After the congestive bandage has been used for some four weeks it may be discontinued and injection of iodoform begun. As the carpal bones are usually much softened they can be penetrated without difficulty. A strong, narrow syringe filled with a 10 per cent emulsion of iodoform in glycerine should be entered on the ulnar side below the styloid process, pushed across to the radial side, and then gradually withdrawn while the injection is made. The injection is thus parenchymatous as well as intra-articular (Senn). When the injections are repeated every ten days to three weeks, they may be made on the dorsal aspect at suitable points; Bier's bandage may be reapplied a day or two after the injection. This treatment has been very successful.

(*d*) *Operative Procedure.*—It should be distinctly understood that tuberculosis of the wrist joint is peculiarly amenable to treatment by immobilisation, and that the surgeon who is dealing with an early case may confidently look for gratifying results from the patient use of a splint alone, or that aided by the congestive bandage and injection of iodoform. Few cases, then, should require operation.

Incision.—There is no doubt that simple incision and drainage will succeed in arresting the disease in cases which have resisted "expectant" treatment. The incisions should be made so as to avoid injury to tendons, vessels, and nerves, and the wounds should be kept open by gauze packing or drainage tubes. They should be made over the most swollen, softened, or tender parts.

Arthrotomy and Curetting.—This should be tried before formal excision is resorted to, for reasons which will be found in the following paragraph. Incisions should be made over diseased areas—usually on the dorsum—or sinuses should be opened up, and all tubercular bone and synovial tissue removed by Volkmann's spoon and scissors. Iodoform should be dusted in, the spaces packed with gauze, and a Lister splint applied. Assiduous pursuit of the disease in this way will usually be rewarded by results which will far surpass those that follow excision.

Excision of the Wrist.—A review of the statistics of this operation will convince any impartial critic that the results which follow it are distinctly disappointing. The mortality, of course, is "insignificant," but the functional results are poor, and this, of course, has to be considered in deciding for excision as against amputation. I do not deny that isolated cases occur in which the surgeon has the gratification of observing an almost perfect use of the hand as the result of excision, but such cases are the exception. Often the hand can do no more than merely hold articles, and these light ones. Possibly this is often due to a formal resection of the joint being too long delayed.

The best method is that by a *single incision on the ulnar side* (Heron Watson). It is very efficient, as access to the parts is free, and it is much easier of performance than Lister's or other methods. An incision about three inches long is made down to the bone on the ulnar side of the forearm towards the palmar aspect, over the lower end of the ulna, the wrist, and part of the fifth metacarpal bone. After the lower end of the ulna has been cleared, fully an inch of it is removed. The way is opened at once for easy and thorough removal of the disease. As each bone of the carpus is met, the tendon can be easily separated from its surfaces without damage; the carpus is removed piece by piece, the trapezium being left if sound. The bases of the metacarpal bones are removed, being turned out of the wound if needful. The metacarpals towards the ulnar side may be dealt with before the radial side of the carpus is attacked; in this way each successive step is made easy. The end of the radius is removed with bone forceps. Few vessels require ligature. The wound is partially stitched and drained by a tube, or it may be packed with gauze. The forearm and hand are bandaged to a Lister splint. Early passive movement of the fingers must be attended to.

Amputation.—Excision should not be performed in cases in which the patient is old or in weak health, or is the subject of well-marked phthisis; nor in which the disease is too widespread to admit of complete removal, or has involved the tendon sheaths in tuberculosis with suppuration and sinuses. In such cases amputation is the proper practice; it should be performed by the modified circular method, or any other that is suitable, as close above the wrist as possible.

Pyogenic Diseases.—These result either (1) from infection through the blood-stream, as in (*a*) osteomyelitis of the radius and ulna, or possibly the metacarpal bones; or (*b*) in pyæmia: or else (2) from without, as in the direct infection of wounds, operation or accidental, or from the spread of the organismal inflammation from a septic teno-synovitis of the tendons of the wrist.

Suppurative arthritis from osteomyelitis of the neighbouring bones is not nearly so common in the wrist as in other joints, as the knee and ankle (*see* Joints); but *pyæmic suppurative arthritis* quite commonly occurs in the wrist in cases of pyæmia. The joint swells often without giving the patient much pain, the skin may not be inflamed, and indeed the surgeon may notice the condition only when taking the patient's hand to examine the pulse. *Treatment.*—The joint should be incised and drained, and the limb bandaged to a splint with the hand in dorsiflexion. Wonderfully good movement may be retained by the joint, especially if the tendon sheaths have not been involved.

Septic arthritis following suppurative teno-synovitis is unfortunately not a rare sequel to a neglected or inefficiently treated "suppurating whitlow." The *symptoms* will depend on the type of the original inflammation and its organism. But once the joint is affected the severity of the symptoms is increased; the pain becomes very severe, the swelling and œdema of the wrist and hand increase, and the skin is reddened. The constitutional symptoms correspond. If untreated the joint will soon be disorganised; in a few days lateral movement will show that the ligaments are softened, the slightest movement causes intense pain, and the bones, denuded of their cartilage, can be felt to grate on one another. The sheaths of the flexor tendons, of course, are the usual source of the disease *Treatment.*—As soon as the invasion of the joint is recognised it should be freely opened, the site of the incisions being determined partly by the spots of greatest tenderness, and partly by considerations of access and drainage; then the joint can be readily opened up and drained on the inner side just below the styloid process of the ulna. If possible there should be continuous irrigation

with a weak antiseptic, while the limb is placed in a splint with the wrist dorsiflexed. In a severe case little movement may be ultimately expected. *Infection of the joint from wounds*, operation or accidental, is followed by similar symptoms, and demands the same treatment.

Gonorrhœal Arthritis (*see* Joints).—This disease does not occur so frequently in the wrist as in the knee or ankle, but the possibility of its occurrence should be borne in mind lest it be mistaken for rheumatic arthritis, acute or chronic. Since it very seldom in any joint results in suppuration, it should be considered apart from pyogenic affections. Since some stiffness may result, it is most important that the hand should be dorsiflexed in the splint already described.

Chronic "Employment" Arthritis.—Madelung has described this condition, which, though not rare, is at the same time apt to be overlooked, at least in its earlier stages, when treatment is of more avail than later. It is a form of chronic inflammation met with mostly in young people, and especially in those whose wrists are subjected to violent or excessive movements—washerwomen, workers in certain trades, boxers, tennis and cricket players. Sometimes it occurs as a change at the radio-ulnar joint, where the ulna, apparently from relaxation of ligaments, projects prominently backwards, and the carpus is displaced forwards. In others the firmness of the radio-carpal joint does not seem to be impaired, but there is a disturbance of growth. The articular surface of the radius is altered so that, apparently in consequence of violent movements mostly in the direction of flexion, growth is arrested on the palmar aspect, and the dorsal edge overhangs; the carpal surface of the radius becomes oblique, and the carpus is subluxated forwards and cannot be replaced in its normal position. Slight pain, at first complained of, afterwards ceases, some disability alone remaining. *Treatment.*—The excessive movement must not be given up, and a leather "bracelet" should be worn for months.

Arthritis Deformans (*see* Joints). — The wrist is not infrequently the seat of this disease, which bears an importance to the surgeon on account of the similarity between some of its features and those of tuberculosis. In both there is a chronic disease with pain, swelling, and stiffness of the joints. Hence it is well to remember in this connection two features highly characteristic of tubercular disease of the wrist. They are marked atrophy of the forearm and early lateral mobility of the joint.

Charcot's Disease (*see* Joints.)—The wrist is but rarely affected with this disease. As it sometimes appears in locomotor ataxy before the tabetic symptoms, there is need for care to avoid an error in diagnosis. Were it to be a case of the mild type, or were the disease, beginning with milder symptoms, to pass into the severe form with swelling, grating of the bones, and marked lateral mobility, the disease —especially with this last symptom—might be mistaken for tuberculosis were it not for one symptom. The remarkable absence of pain and tenderness, so extraordinary a feature of Charcot's disease, ought to make a mistake impossible.

Tubercular Teno-Synovitis (*tubercular tendo-vaginitis*, "compound ganglion").—This is a local tuberculosis which affects the synovial sheaths of the tendons both on the flexor and extensor surfaces of the wrist. Though undoubtedly infrequent in comparison with joint tuberculosis, tubercular tendo-vaginitis cannot be called rare in this region.

Symptoms.—The first symptom to be noticed is usually a *swelling* on the back or front of the wrist. It is a more sharply defined one than the swelling occurring in tuberculosis of the joint, is often oblong with rounded ends, running parallel with the tendons. Sometimes the swelling is fairly round, but it often consists of "compartments," lateral offshoots spreading from the central mass; hence the name "compound ganglion." If it occur on the front of the wrist it may be "hour-glass" in shape, the annular ligament causing a constriction between a mass in the palm and one over the upper part of the wrist. The swelling may be movable transversely with the tendons, but not longitudinally. According as it is of the fungus or hydrops variety, it will feel semi-elastic or fluctuating, and in the latter case especially the presence of "rice-bodies" may impart a characteristic sensation as they are pressed from one part to another of the swelling. *Pain* is by no means a marked symptom; in fact, it is often absent for a long time after the swelling has attained a large size. *Movement* of the fingers similarly may for long be quite free, though as the disease progresses it may destroy the tendons and impair the use of the fingers. Eventually the wrist joint may be invaded, or abscesses may form and open spontaneously with disastrous results from the consequent sepsis.

Pathological Anatomy.—Two forms of this disease are usually described:—(*a*) The tendon sheaths are distended with synovial fluid, their walls are not greatly thickened with granulations, but they are frequently hung with numerous hard white masses, the *corpora oryzoidea*, or "rice-bodies" ("melon seeds"). These vary in size up to that of a pea, are flattened and ovoid; some hang loosely packed from the wall of the sheath, while others float free in the fluid. Though tubercle bacilli are rarely found in them, they produce tuberculosis in animals inoculated with them. (*b*) In the second variety the swelling is due mainly to massive fungous granulations which enormously increase the thickness of the sheaths, there is

little fluid, and rice-bodies are wanting. As a matter of fact these forms may be found combined. The tendon itself is eventually invaded; as one dissects the thickened sheath off in the operation for the condition, the tendon is sometimes found to be so slender as to require care not to rupture it; while in some cases the glistening white fibres will be followed up till at last they are lost in the tubercular mass, there is an actual gap in the continuity of the tendon. After the condition has been apparently cured by excision of the sheath, it may recur in adjacent sheaths, and eventually invade the joint itself. I excised the sheaths and a huge swelling the size of an orange from the common extensor tendons of the wrist in a young man who made no complaint except of the swelling; two years later it recurred in the extensors of the thumb, and was again successfully removed; after another interval of two years the disease attacked the wrist joint, but was eventually successfully combated by the congestive bandage and iodoform injections.

Diagnosis.—The "compound ganglion"—a term which should be entirely abandoned for "tubercular teno-synovitis"—ought usually to be easily distinguished from the simple ganglion. The following are the points of distinction:—The ganglion usually appears as one single round tumour, without accessory compartments as in the tubercular disease; should the ganglion be "compound" in shape, however (which is seldom the case), its tenseness and firmness, especially when the wrist is extended, serve to distinguish it from the much softer fluctuation or else more boggy feeling of the other; and, lastly, if required, the diagnosis may be absolutely confirmed by a puncture which evacuates in the case of a ganglion the characteristic fluid resembling soft glycerine jelly, while unmistakable synovial fluid or even minute rice-bodies will escape from the tubercular swelling.

Treatment.—It may be that the congestive bandage and iodoform injections will prove as efficacious in the form of tuberculosis as they prove in joint tuberculosis, but as yet no treatment has been followed by results that can compare with *excision of the affected sheaths.* The operation is always an extensive and prolonged one. An incision should be made longitudinally over the swelling and considerably beyond it at each end, or else a flap with its convexity to one side should be raised so as fully to expose the diseased area. After separating the tumour from the healthy tissues by blunt dissection, the operator splits the sheath longitudinally for the entire length of the disease, and then with fine scissors or the knife dissects it away and cuts it off from the healthy tissue at each end. If a gap be found in a tendon the two ruptured ends may sometimes be sutured in the ordinary way by the splitting method. The wound is then closed, with or without a small drain, as is thought advisable. The splint, which is required for the first ten days, should at the end of that period be discarded; passive movements are begun, and the patient is encouraged to move his fingers. Perfectly free movement is to be looked for.

Pyogenic Teno-Synovitis.—By far the commonest cause of this condition in the tendon of the wrist is "whitlow," septic infection of a wound of a finger, though it may follow a wound of a tendon sheath. The violent inflammation which ensues is very apt to produce necrosis of the tendon, or, if the disease stop short of that, firm adhesions may form which render the corresponding fingers useless. The disease as it occurs in the fingers has already been described (*see* "Hand"). The *symptoms* here are of the same character; the palmar tendons are, of course, much more frequently affected. *Treatment.*—As soon as the presence of pus in the sheaths is suspected they should be laid freely open. The sites for incision should correspond to the tender areas, and the highest point invaded by pus at or above the wrist can be determined with precision by noting the highest spot of acute tenderness on pressure. The wound should be washed out with sublimate lotion and drained by tubes. Whenever the wounds begin to heal, voluntary and passive movements of wrist and fingers should be commenced at once, and be so assiduously persevered with that all stiffness, which can possibly be avoided, may be prevented.

Acute Teno-Synovitis (*non-pyogenic*).—This disease may be considered as occurring in three forms: (*a*) Dry crepitating teno-synovitis; (*b*) Dry plastic teno-synovitis; and (*c*) Acute serous teno-synovitis (Watson Cheyne).

(*a*) *Dry crepitating teno-synovitis* is apt to occur as an acute disease after prolonged and excessive use of the hand and fingers, in the work of washerwomen, pianists, and others; in these it occurs mostly on the extensor aspect. The *symptoms* are subacute pain on pressure and on movement, slight stiffness, and occasionally slight swelling; in addition, when the tendons move a characteristic sensation of friction is produced (as "when two pieces of silk are rubbed one over the other") palpable both by the patient and surgeon. Spontaneous cure is the rule, but relapse is common. *Treatment.*—The hand should be placed on a Lister splint for a week, and if this does not suffice, a blister will be found in this affection specially useful; as it heals, the moderate compression produced by a firm bandage over wool will aid the cure.

(*b*) *Dry Plastic Teno-Synovitis.*—This is the much more serious variety which results in adhesions, following Colles' fracture, suppurative teno-synovitis, and similar conditions, though it may arise in rare cases without a trauma, when, of course, it is *called* gouty or

rheumatic. Apparently it may exist without actual invasion of the sheaths by infective organisms. Acute pain and great stiffness of the fingers are the chief *symptoms*; in the first few days of the disease soft adhesions may be felt to break down on movement of the fingers. The adhesions, if left to themselves, will eventually become so firm as to render the fingers useless. The *treatment* consists in resting the hand in a sling for a week and applying hot fomentations in the acute stage; after that has passed, at the end of the week, passive movements and massage should be begun *and should be continued* till patience and perseverance are at length rewarded. If the surgeon should encounter the condition at a later stage than the first, repeated attempts must be made to break down adhesions under nitrous oxide anæsthesia, with massage and movements in the intervals.

(*c*) *Acute Serous Teno-Synovitis* (*see* "JOINTS, GONORRHŒAL ARTHRITIS").

CHRONIC TENO-SYNOVITIS occurs in two forms, usually in the palmar sheath:—

(*a*) *Simple Chronic Teno-Synovitis.*—The only *symptom* in this form is a painless, slowly growing swelling, which hardly interferes at all with movement of the wrist or fingers. In the palm it sometimes extends above and below the annular ligament, under which the fluid can be pressed from one compartment to the other. The wall of the sheath is not appreciably thickened; it contains a clear pale yellow fluid. *Treatment.*—The swelling should be incised above the annular ligament, and a drainage tube inserted and kept in as long as needful; passive movements should be begun gently after a week.

(*b*) *Villous Teno-Synovitis.*—This is an affection which might be mistaken for tubercular teno-synovitis, but is a distinct disease, resembling arthritis deformans in joints. The tendon sheath is markedly thickened and lined with villous projections, and is distended with fluid. The *symptoms* in this form are the presence of a swelling which fluctuates, and at the same time gives the sensation produced by the outgrowths; the free fluctuation and absence of tubercular symptoms will help to distinguish it from tuberculosis. *Treatment.*—A somewhat severe operation is required, at least if the sac extend above and below the annular ligament. The ligament must be divided, and the whole sac dissected out; the ligament is then sutured and the wound drained. Rest and passive movements are combined in the later stages as before.

TUBERCULAR OSTEOMYELITIS OF THE METACARPAL BONES.—This is a chronic tubercular osteomyelitis which frequently affects the metacarpal bones of children, and still more commonly the phalanges of the fingers (tubercular dactylitis). The disease attacks the medulla, producing the usual tissue of tuberculosis, while the compact shell is gradually eaten away from within; at the same time the periosteum deposits new bone on the surface, with the result that the tube of bone has the appearance of having been expanded from within. When the subperiosteal deposit is slight, or is wanting, the shell becomes as thin as parchment, and eventually gives way, leading to abscess and sinus formation. *Symptoms.*—The patient is usually a weakly child, and for long the only symptom noted is a gradually increasing swelling seen best on the dorsal surface of the hand; the fusiform or "bottle-shaped" character of the swelling is not, of course, so evident in a metacarpal bone as in a phalanx. Little pain or tenderness is complained of till the superficial parts become inflamed, when, in addition, the swelling will increase rapidly, the skin will redden and break down, and a sinus will form leading to the centre of the bone. *Treatment.*—Treated at an early stage cases of this disease may be absolutely cured. The congestive bandage and a splint should first be tried. If these fail, iodoform emulsion should be injected into the bone. These cases are admirably suited for this, as the bone can be easily pierced by the injecting syringe and the whole cavity filled with the emulsion. If the disease has progressed too far to be arrested by these means the bone should not be excised; the swelling should be incised for its whole length, the cavity of the bone thoroughly scraped, swabbed, if needful, with undiluted carbolic acid, and packed with gauze. The usual constitutional means for combating tuberculosis are peculiarly successful too.

Writer's Cramp, Palsy or Spasm.

See NEUROSES, OCCUPATION (*Etiology*); SPASM (*Varieties*).

Writing.

See BLIND, TRAINING AND EDUCATION OF (*Braille Writing-Frame*); HYPNOTISM (*Experimental Phenomena, Automatic Writing*); MIRROR-WRITING.

Wry-Neck or Torticollis.

VARIETIES 558
ETIOLOGY 559
TREATMENT—
Operative 560
Subcutaneous Method . . 560
Open Method 560
After-Treatment 561
SPASMODIC WRY-NECK 561
Treatment 562

See also DEFORMITIES; HYSTERIA (*Contractures*); HYSTERIA, SURGICAL ASPECTS OF.

THE term torticollis (*torqueo, tortum*, twist; *collum*, neck) is applied to any vicious position of the head of a more or less persistent nature, whatever its cause.

It is usual to state that the condition occurs in two forms, the congenital and the acquired, though, personally, I have never seen an instance of the former. That is to say, I have never met with a case of wry-neck in which it was affirmed that *as soon as the infant made its entrance into the world the medical attendant noted that its head and neck were permanently twisted* It is quite possible, however, that if a fœtus were tightly packed in the uterus with an insufficient amount of liquor amnii, and with its head rigidly bent over to one side, it might offer an example of congenital wry-neck. Such cases must be, at any rate, of very rare occurrence. But it is not at all an uncommon thing for a nurse or a parent to call the doctor's attention to the fact, when the infant is two or three days old, that it holds its head towards one shoulder. And a year or two afterwards, if an operating surgeon were closely questioning the mother as to when the deformity was first noticed, it is quite likely that the mother would affirm that it was discovered "directly after birth," and the temptation to call it congenital might, in the circumstances, be almost irresistible.

One frequently sees an infant a few days old persistently bending its head down to one shoulder, turning the face towards the other side, and showing by flinching or crying when the root of the neck is fingered that there is much tenderness there. The meaning of all this is that a sudden and violent movement of the head, as the child was being born, caused the sterno-mastoid muscle to be torn across; blood was poured out into the muscle-sheath; the neighbouring nerves were pressed upon, and the infant could not bear the part being fingered.

In some cases the muscle tears across as the result of the medical attendant or midwife pulling upon the feet, but it may be met with in infants whose entrance into the world has been so easy that neither nurse nor midwife was called upon to help. In a considerable proportion of the cases the children have entered the world breech first; possibly there is a greater strain put upon the muscles in these circumstances. In fat-necked infants the tumour may pass unnoticed for days or weeks, but, as the neck grows, the lump can scarcely fail to attract the attention of the mother.

The sequence of events is probably as follows:—The muscle is torn across by sudden strain, and a considerable effusion of blood and a local myositis ensue; interstitial granulation tissue is formed, and this is shortly converted into fibrous tissue, which, undergoing contraction, produces a permanent cicatricial contraction in the muscle.

Time after time one learns on inquiry that the child with wry-neck had in its early infancy a tender swelling in the muscle which gradually became hard and painless. Not many years ago such tumours were thought to be evidence of congenital syphilis, but inspection of one by Dr. Frederick Taylor revealed their exact nature. A surgeon is never justified in assuming that because a tumour of the sterno-mastoid disappeared when the child was being treated by mercury it was necessarily syphilitic.

As a rule both parts of the muscle—the sternal and the clavicular—are implicated in the shortening, though sometimes only one is concerned. And it often happens that when only one part of the muscle apparently needs division, after the surgeon has cut it he finds that the remaining part also has to be severed.

As a result of the permanent shortening of the muscle the mastoid process is drawn down towards the top of the sternum, so that the ear of the affected side approaches the shoulder, whilst the chin and face are turned towards the opposite side. And when the attempt is made to place the head and face in an improved position, the rigid muscle is brought out into strong relief, and the shoulder is drawn up to meet the ear.

The shoulder of that side is permanently raised, and from the constant dragging of the shortened muscle the due development of that half of the head and face is checked, so that the parietal eminence, the half of the frontal bone, the malar bone, and that side of the lower jaw may be markedly smaller than they should be, whilst the ear and the commissure of the eyelids and of the lips may be dragged downwards. A similar arrest of development may affect the sides of the vertebræ, so that a lateral curvature of the cervical column is produced, the concavity being directed to the affected side, with, in several cases, a secondary curvature in the opposite direction in the dorsal region of the spine.

Torticollis Anterior.—In certain very rare instances symmetrical contraction follows rupture of both sterno-mastoid muscles, with the result that the chin is rigidly drawn down towards the top of the breast-bone. Both muscles being equally shortened the head cannot be inclined to either shoulder, nor the face turned to the right or the left, so the deformity cannot be called *double wry-neck*. A very suitable name for it, however, is torticollis anterior.

Persistent deviation of the head and neck may also be the result of inflammation of the cervical glands; of a shortening of the sterno-mastoid following suppurative inflammation of that muscle or of the tissues near it; of contraction of the scar following a severe burn of the neck; of periostitis of the cervical vertebræ; of tuberculous disease of the vertebræ themselves, or of the synovial joints between the base of the skull and the first and second cervical vertebræ. (Hysterical contraction of a sterno-mastoid is unlikely to occur without other

suggestions or manifestions as to its exact nature.)

Wry-Neck due to Vertebral Disease.—Of all the foregoing conditions the most important is tuberculous inflammation of the cervical vertebræ or of the joints in the highest region of the neck. (*See* SPINE, SURGICAL AFFECTIONS OF (*Spinal Caries*), vol. ix. p. 298.) The symptoms of tuberculous disease of the spine are not infrequently misinterpreted and are ascribed to "rheumatism," but the deformity may be distinguished from ordinary wry-neck by the fact that pressure upon the top of the head causes distress, and that even in a limited degree the patient is unable to move the neck or to shake or nod the head; and that there have been for many weeks, or even months, complaints of obscure pains in the head, neck, or chest, the result of pressure upon the roots of the nerves passing out through the diseased region of the spine. In wry-neck due to disease of the vertebræ no movement is permitted except with apprehension and pain, whereas in the other case movements are free and painless within certain limits. To mistake cervical caries for common wry-neck may involve very serious consequences.

TREATMENT OF WRY-NECK.—For the treatment of wry-neck which is due to permanent shortening of the sterno-mastoid nothing short of a cutting operation can suffice, and the sooner that it is efficiently carried out the better. The treatment by drugs, electricity, or massage, can end in nothing but disappointment.

Operation for Wry-Neck.—The child should be anæsthetised and placed upon an operating table, or upon a folded blanket laid upon a low chest of drawers or a narrow table. The shoulders should be slightly raised on a firm pillow, and, whilst the head is drawn towards the opposite side, a nurse should pull down the arm of the affected side so as to render the muscle tense and prominent. The head should be held and steadied by the chloroformist, so that the assistant may have both his hands free. It is hardly necessary to say that the skin of the neck, and everything that is to come in contact with the wound, should be aseptic.

Division of the sterno-mastoid may be performed either by subcutaneous tenotomy or by what is known as the open method.

The subcutaneous method consists in making a puncture in the skin at the root of the neck, close to the side of the contracted tendon, and introducing a blunt-ended tenotomy knife through the puncture and beneath the band, which is then divided by a sort of sawing motion up towards the skin. Traction being made upon the head, the tendon yields in a definite and obvious manner. The movements of the knife are checked just before the skin is reached, the actual progress of the knife being estimated partly by the finger placed on the skin over the point of section.

After the one piece of the muscle—the sternal origin, for instance — has been divided, and further traction is made on the head, the remaining portion of the muscle is generally found to be in need of division, although it may not previously have appeared to be shortened.

The clavicular head is then divided in a manner similar to the preceding, and generally through a separate skin-wound. In a small proportion of cases, however, only one portion of the sterno-mastoid may require section.

The operation being finished, a small pad of lint is fixed over the punctures, the head being left in the old position until they are soundly healed.

Subcutaneous tenotomy was introduced in the days when suppuration commonly followed the infliction of an open wound on account of septic micro-organisms having been allowed to contaminate the freshly cut surfaces. The smaller the wound the less the chance of septic infection. Division of the muscle through the smallest possible wound was, therefore, almost a necessity a few years ago. But the subcutaneous method had this great disadvantage, that when the knife was working out of sight and in a very dangerous area the surgeon could not know exactly what he was dividing. And so it came about that a large anterior or external jugular was sometimes wounded, with an alarming, if not a fatal result. Walther states that whilst performing the subcutaneous operation Robert cut the external jugular vein, and that Volkmann wounded the internal jugular. I also once had an alarming experience whilst operating by the subcutaneous method at The Children's Hospital a good many years ago, the child nearly dying either from entrance of air into a wounded vein, or from grave shock which was produced in some other manner. At any rate, there was so great a welling-up of blood that I had immediately to apply firm pressure over the spot and send the child back to bed with the tenotomy unfinished. And it was with no little apprehension that I set about the completion of the operation a few days later. However, no further trouble ensued, and the child was in due course completely cured.

The open operation has now almost entirely taken the place of the subcutaneous method. First of all the skin at the root of the neck is carefully prepared and rendered aseptic. An incision, long enough to give free access to the deeper parts, is then made over the contracted muscle about a finger's breadth above the upper border of the clavicle, the superficial fascia, platysma, and deep fascia being divided layer by layer, and bleeding vessels being secured by clip-forceps. Then the origin of the muscle is reached, and its fibres are cut through until the

layer of the cervical fascia which lies beneath it is fully exposed. This also may need some touches of the knife before the head can be drawn into the improved position.

All the rigid bands of muscle and fascia being divided, a wide gaping wound is made, in the depths of which the surgeon may see the subclavian vein heaving with the respiratory movements, to say nothing of the swollen jugulars and their tributaries. If in the course of the operation a branch of vein has to be divided, it is first leisurely and securely dealt with, for the surgeon sees every structure before he cuts it, and has ample opportunity for recognising it. And this, surely, is a great comfort.

Before the wound is sutured it is well to have the head vigorously bent over to the other side, whilst the face is made to rotate to the affected side in order to stretch all the shortened structures to the utmost, and thus to begin the corrective treatment whilst the child is under the anæsthetic. Force thus vigorously applied is extremely helpful.

When every resisting band has been divided with the point of the knife or scratched through with a director, and all bleeding has been stayed, the skin-wound is closed with an aseptic suture of horse hair or silk-worm gut, and dressed with a seal of gauze and collodion; or an aseptic pad is secured over the place by a bandage which runs round the neck and under the armpit, and the child is put to bed with the head steadied, in a slightly improved position, between two large sand pillows.

Sometimes after the sterno-mastoid has been divided, and bands of the deep fascia have been scratched through or efficiently stretched, the border of the trapezius or of some other cervical muscle may be found short and rigid, but this complication can easily be put right subsequently by massage.

It is occasionally urged that, in the treatment of wry-neck in a girl who will eventually have to wear low-necked dresses, the open method is not advisable, in that it will be followed by a permanent and possibly an unsightly scar. But, in my opinion, this is a matter of comparatively small importance when the question of the effectual and safe performance of a necessary and a delicate operation is at stake. As regards the possible disfigurement of the scar by a keloid thickening, this is likely to occur only in a tuberculous subject. And should it occur, it may be trusted to disappear as the child grows in years and in health.

As regards the respective merits of the subcutaneous and the open operations, I would say that for some years I practised the subcutaneous method, but that I have long since entirely discarded it for the open operation, and I would not revert to it under any consideration. Redard goes so far as to maintain that the great progress which has been realised in the treatment of wry-neck is chiefly due to the general adoption of the open method.

In certain cases resection of a piece, or even of the whole, of the sterno-mastoid has been practised. Personally, I have never seen a case in which I could have thought so severe a measure to be necessary or justifiable.

The after-treatment should be begun as soon as the stitches have been removed, which is, as a rule, well within the week, though if for any cause the healing of the skin-wound has been delayed, stretchings and manipulations must be postponed. However, if the division of the muscle has been thoroughly carried out, the divided ends having been left, perhaps, far asunder, the delay of a few days is of no serious import.

The operation having been thoroughly performed, no splint "brace" or apparatus of any kind is needed, not even in the case of a young adult. If the patient be a very young child the rectification of the position of the head and the straightening of the cervical column must be gradually effected by the surgeon's hands, and in this he may obtain much assistance from a competent nurse or an intelligent parent. At night the child should be made to lie upon the affected side, with the head so placed upon a thick and firm pillow that even during sleep the treatment is not entirely remitted.

If the child be old enough to walk he should be encouraged to go about with a shot-can, or some other weight, hanging in his hand so as to pull down the shoulder to the utmost, and he should also be made to incline his head over to the opposite side. And many times during the day this drilling may be supplemented by firm massage and manipulations of the neck. Rigid retentive apparatus of all sorts would be prejudicial, in that it would prevent the needful exercise of the muscles.

The after-treatment demands long-continued care and patience; it is obvious that deformities extending secondarily to the bones themselves cannot be corrected by the mere cutting of a contracted muscle.

If the patient be capable of taking an intelligent interest in the after-treatment of his case, he and his surgeon will be able to plan all sorts of games or exercises which will have the effect of stretching the tissues on the short side of his neck, and hurrying on the complete effacement of the deformity.

Spasmodic Wry-Neck

As the result of central nervous irritation in adult persons, the sterno-mastoid may be implicated in persistent rhythmic contractions, which, by violently jerking the side of the head and neck, may cause considerable pain and distress, or seriously interfere with the usefulness and comfort of the patient. Spasmodic wry-neck has to be distinguished from hysterical contrac-

tion of the sterno-mastoid, which, however, is more likely to occur in a youngish woman.

Treatment. — As regards the treatment of spasmodic wry-neck all drugs, inclusive of arsenic and strychnia, are disappointing, and electricity has not been found of service. Unfortunately, time, the great healer, passes these cases by, and nothing remains but for the surgeon to resect a portion of the spinal accessory nerve. Possibly he may also have to take away branches of the cervical nerves entering the muscle in order to ensure complete relief.

For *resection of the spinal accessory nerve* a three-inch incision is made from the tip of the mastoid process down the anterior border of the sterno-mastoid. The edge of the muscle is lifted well up, and the posterior belly of the digastric muscle exposed; the nerve then is seen passing from beneath it into the deep aspect of the sterno-mastoid. It may be necessary to slacken the muscle and turn it well out in order to expose the nerve, which is, however, a large and conspicuous trunk. On pinching it in the forceps spasmodic contractions of the sterno-mastoid and trapezius result, and reveal its identity beyond a doubt.

On the first occasion of the spinal accessory nerve being resected for the relief of spasmodic wry-neck, the surgeon (de Morgan) sought for it at the posterior border of the sterno-mastoid, and, following it upwards and forwards through the muscle, divided it above the point of entrance, which is on a level with the angle of the lower jaw. The usual operation now, however, is to seek the nerve at its entrance into the muscle. There is no great difficulty in the operation if the surgeon makes a long incision, beginning it as high as possible, and lifting the muscle well up.

Excision of the posterior branches of the cervical nerves is rarely called for in the treatment of spasmodic wry-neck, and it should not be undertaken unless it is certain that the spinal accessory nerve has undoubtedly been resected, and that that measure has proved insufficient.

Wunderlich's Law.—The temperature curve in typhoid fever with its ascending oscillations (morning and evening).

Wutzer's Operation. — A radical operation which used to be employed in cases of inguinal hernia, the scrotum being invaginated as far as the internal inguinal ring by means of a hollow cylinder of wood carrying a concealed needle, by which the various parts are transfixed.

Wutz's Valve. *See* URACHUS (*Valve Preventing Passage of Urine from Bladder into Urachus*).

Xanthelasma. *See* XANTHOMA.

Xanthin. *See* LEUCOCYTHÆMIA (*Symptoms, Urine*); LIVER, PHYSIOLOGY OF (*Supply of Proteids, Dioxypurin*); PHYSIOLOGY, EXCRETION (*Urine, Nitrogenous Substances*); URIC ACID (*Chemical Characters, Dioxypurin*); URINE, PATHOLOGICAL CHANGES IN (*Nitrogenous Constituents, Xanthin Bases*); URINE, PATHOLOGICAL CHANGES IN (*Calculi*).

Xanthinuria.—The presence of an excessive quantity of xanthin in the urine.

Xantho-.—In compound words *xantho-* (Gr. ξανθός, yellow) means yellow; *e.g.* the *xanthochroi* (fair or yellow-haired races), *xanthochromia* (a yellow discoloration of the skin), *xanthopathy* (any skin disease characterised by a yellow discoloration).

Xanthocreatinine or Xanthokreatinin. — A leucomaine or poisonous alkaloid ($C_5H_{10}N_4O$), in the form of yellow crystals, found in muscle.

Xanthocyanopia or Xanthocyanopsia. — Red-green blindness. *See* COLOUR VISION (*Congenital, Partial*).

Xanthodermia. *See* XANTHOMA.

Xanthoma. *See also* DIABETES MELLITUS (*Symptoms, Skin*); TUMOURS OF THE SKIN (*Xanthoma Palpebrarum*). (SYN.: *Xanthelasma, Vitiligoidea.*)—*Definition.*—A fibro-fatty neoplasm forming yellow plates or nodules in the corium (Crocker). Two forms of the disease are recognised—Xanthoma planum and Xanthoma multiplex.

XANTHOMA PLANUM.—This is the more common form of this rare affection. It is characterised by the occurrence of small, flat pin-head and finger-nail size plaques, which vary in colour from a pale yellow to a much darker shade. They are circumscribed in character, are situated in the corium, and are very little if at all raised above the surface; the usual site is the eyelids, more especially near the inner canthus, from which they may spread to involve the surrounding skin. With a lens the patches may often be seen to consist of an aggregation of small yellow granules, which usually have a central pinkish puncture. There are little or no subjective symptoms.

XANTHOMA MULTIPLEX (*Xanthoma tuberosum*) is a more rare and also a much more serious affection. The most characteristic sites are on the extremities, especially on the parts subjected to pressure. The nodules vary in size from a hemp seed to a pea, but by fusion they may form a swelling as large as an orange. Occasionally the mucous surfaces of the mouth, of the respiratory and gastro-intestinal tracts, are involved, as also the surfaces of the peritoneum,

endocardium, and larger arteries. The genital region, palate, œsophagus, spleen, trachea, and cornea have all been recognised as seats of the disease. Papular and tubercular lesions may coexist with the plain lesions above described, and scarcely differ from the latter save in a greater development. The lesions are whitish or yellowish papules, plaques, and tubercles, circumscribed in contour, covered with an unaltered epidermis, and determinable by palpation as having greater consistence than the flat macules. In rare cases the tubercles may coalesce to form sessile or pedunculated tumours, which are firmer as a rule than the smaller lesions (Hyde). In some cases the disease is accompanied by a general yellowish discoloration of the skin. Disfigurement is the chief symptom complained of. Occasionally pricking sensations are present. The *etiology* of the disease is quite unknown. The *pathology*, according to Chambard, is as follows:—There are two processes going on, an increase of connective tissue and a fatty degeneration or deposition, the results of a chronic inflammatory process, in the soft plaques the fatty change, and in the nodules the connective tissue growth predominates, being greatest in the larger and firmer ones.

The diagnosis is easy if regard is had to the peculiar yellowish or saffron-like hue of xanthoma. Multiple dermoid cysts may simulate.

Treatment. — Erasion and excision are the usual methods of treatment. Electrolysis is of great service in some cases; and various caustic applications have been recommended.

Xanthoma Diabeticorum. — This is an extremely rare affection. It is very fully described by Crocker, from whose description the following account is given. The eruption consists of dull red discrete or confluent papules quite firm to the touch, from a line to a quarter of an inch in diameter, well defined at the margin and roundish or obtusely conical. On the top of many of them, but not of all, is a yellowish or yellowish-white head, which looks like a pustule, but is really solid, and some of the papules are dotted or streaked with red from dilated vessels; a red areola is sometimes seen. Itching, pricking, or tenderness is generally felt in the lesions. The most common situations are the buttocks, elbows, and knees, where they are generally confluent, and may form tumours, though the papular origin is generally still discernible. They have also been seen on the trunk, on the extensor surfaces generally, on the mucous membrane of the mouth, on the face, scalp, and bend of the ankles. The eruption comes out rather suddenly at first upon the extensor aspect of the limbs, especially the forearms, and then more gradually in other parts; after remaining stationary for some time —months or even years—the papules begin to disappear, leaving no trace behind them, or while some disappear others come out; or again they may disappear entirely for a time and then break out once more.

Etiology. — Males are affected much more than females. The disease is one of adult life. Most cases are accompanied by true diabetes mellitus.

Pathology.—The diseased process is anatomically of the same nature as ordinary nodular xanthoma, but with more inflammatory phenomena and less connective tissue growth. With regard to its pathogeny, in diabetes as in jaundice, disorder of the liver exists, but the clinical facts show that derangement short of that necessary to produce either diabetes or jaundice may yet produce xanthoma.

Diagnosis.—Those who separate this form of xanthoma from all others base the difference between them upon the following points (Hyde):—

1. In xanthoma of glycosuria the sudden evolution and involution of the cutaneous lesions.

2. The firmness and solidity of the latter as distinguished from the softness of the ordinary forms.

3. The inflammatory character of the glycosuric as distinguished from the hypertrophic changes in the other variety.

On the other hand, some authorities urge that the glycosuria is simply an initiatory cause, which explains the differing symptoms of xanthoma in the two classes of cases. Surveying the literature of xanthoma, they find patients without diabetic symptoms suffering from atrocious pruritus and most of the special features claimed as peculiar to diabetic xanthoma of glycosuria. The general conclusion arrived at is that it is difficult to determine what are the relations, if any, between these two forms of xanthoma.

The treatment of the disease, medicinal and dietetic, is mainly that of glycosuria.

Xanthoproteic.—Related to xanthoprotein (a yellowish substance produced by the action of nitric acid on proteids); the xanthoproteic test or reaction is the fact that an orange colour is produced when ammonia is added to a protein heated with nitric acid. *See* Physiology, Protoplasm (*Products of Decomposition*).

Xanthopsia.—Yellow vision, as in cases of jaundice. *See* Jaundice (*Symptoms, Yellow Vision*).

Xanthosis.—A morbid condition in which the skin or a mucous membrane has a dirty yellow colour, as in cases of carcinoma and in chronic inflammation of the nose. *See* Nose, Chronic Inflammation (*Xanthosis*).

"Xaxa."—Acetyl-salicylic acid as prepared by Burroughs Wellcome & Co.; dose, 5 to 20

grains. "Xaxaquin" is the quinine salt of the same; dose, 3 to 6 grains.

Xenia.—Telegony as it is met with in plants (Gr. *ξενία*, the state of a guest). *See* HEREDITY (*Telegony*).

Xeno-.—In compound words *xeno-* (Gr. *ξένος*, a guest or stranger) means strange or foreign, *e.g. xenogenesis* (heterogenesis).

Xenomenia.—Vicarious menstruation, or periodic hæmorrhage from some other part of the body than the uterus taking the place of the menstrual discharge. *See* MENSTRUATION (*Vicarious*).

Xenon.—A gas forming a very small proportion of the atmospheric air; it has only recently been differentiated from nitrogen.

Xero-.—In compound words *xero-* (Gr. *ξηρός*, dry or parched) means dry, e.g. *xerotripsis* (dry friction).

Xeroderma or Xerodermia. *See* ICHTHYOSIS (*Symptoms, Xerodermia*).

Xeroderma Pigmentosum. *See also* SKIN, PIGMENTARY AFFECTIONS OF (*Neoplastic Type, Xerodermia Pigmentosum*); TUMOURS OF THE SKIN (*Carcinoma*); X-RAYS (*Therapeutics*).—A rare disease which commonly commences in the first or second year of life and progresses continuously. It commences with freckle-like pigmentations, surrounded in a year or two by telangiectases; this is followed by a gradual disappearance of the latter and the occurrence of cicatriform depressions; there is finally a diffuse atrophy of the skin. New pigment spots continue to form, so that a given case usually presents all types and grades of lesions. The general health seems to be unaffected. The *etiology* of the disease is quite unknown. The *pathology* probably begins as a proliferation of connective tissue in the papillary layer with involvement of the vascular endothelium, followed in some points by retraction and in others by both ectasis and new formation of vessels. Kaposi, who first named and described the disease, considers that the irregular accumulation of pigment is consecutive to the vascular changes. The diagnosis has to be made from scleroderma and lepra maculosa. The prognosis is guarded; there is a distinct tendency for the disease to assume a malignant (sarcomatous) form. Most cases succumb in from ten to twenty years. *Treatment* is purely palliative.

Xeroform.—An odourless powder containing oxide of bismuth and tribromphenol; it has an antiseptic action; dose, 7½ grains.

Xeroma. *See* XEROPHTHALMIA.

Xerophthalmia.—A dry and thickened state of the conjunctiva occurring as a sequel to long-continued conjunctivitis of a severe type.

Xerosis. *See also* CONJUNCTIVA, DISEASES OF (*Degenerations, Xerosis Conjunctivæ*); CORNEA (*Kerato-malacia, Xerosis bacillus*).—Xerosis is a term applied to a condition of skin intermediate between ichthyosis simplex and keratosis pilaris. The skin is dry, harsh, and void of the natural moisture and lubrication. The skin surface is finely scaly, and in some instances the passage of the hand over the skin surface will cause separation of scales in a fine shower. In other cases the scales are more definitely adherent. The eruption is most frequently seen in the extremities, but all parts of the skin may be involved. The disease is met with in all grades, being sometimes only indicated by a very dry finely scaly condition of the skin of the face, and at the other extreme by a state of "goose skin" appearance, characteristic of mild cases of ichthyosis. The treatment and prognosis are similar to those of ichthyosis, to which article the reader is referred. One word, however, may be said *re* the etiology of this affection, supplementary to what has been written *re* the etiology of ichthyosis. The present writer believes that xerosis, ichthyosis, and allied disorders are disorders largely due to some defect in metabolism of fats in the body. Further, that this defect is closely related to a defect in the bone-marrow function. And, further, that both of these defects are in turn related to, and in great part dependent on, a chronic auto-intoxication from the alimentary tract, or one of the other free mucous surfaces of the body. This view may or may not be correct. In all cases, however, the practitioner is directed to a close investigation of the functions of the free mucous surfaces of the body, more especially the oro-, gastro-intestinal, and respiratory mucous membranes, with the view of correcting any defect that is there discernible on careful investigation.

Xerostomia or Xerostoma.—Abnormal dryness of the mouth; aptyalism. *See* SALIVARY GLANDS, DISORDERS OF (*Xerostomia*).

Xiphi- or Xipho-.—In compound words xiphi- or xipho- (Gr. *ξίφος*, a sword) means relating to the ensiform cartilage of the sternum; e.g. *xiphicostal* (relating to the ensiform cartilage and to the ribs), *xiphodynia* (pain in the ensiform cartilage), etc.

Xiphisternum.—The ensiform cartilage of the sternum; the xiphoid cartilage or process or appendix of the sternum.

Xiphodymus or Xiphodidymus.—A teratological type in which two fœtuses are united so that their pelvic and thoracic cavities are fused; there are usually

two legs, but there may be a third (rudimentary) lower limb.

Xiphopagus.—A teratological type in which two fœtuses are united by the tissues stretching from the lower end of the sternum to the umbilicus. *See* TERATOLOGY (*Thoracopagous Double Monsters*).

X-Rays, Finsen Light, and High Frequency Currents.

RÖNTGEN RAYS, THEIR NATURE AND PRODUCTION 565
IN DIAGNOSIS 571
IN TREATMENT 574
Skin Affections 574
Tumours 576
Other Affections . . . 577
THE FINSEN LIGHT. 578
Its Advantages and Disadvantages . 579
HIGH FREQUENCY CURRENTS . . . 580
Mode of Production 580
Therapeutics of 582

See also BLADDER, INJURIES AND DISEASES (*Calculus Vesicæ, Diagnosis*); BONE, DISEASES OF (*Chronic Osteomyelitis, Diagnosis*); CONJUNCTIVA, DISEASES OF (*Various Conditions, Conjunctivitis from Dazzling*); CORNEA (*Injuries, Foreign Bodies, Diagnosis*); FAVUS (*Treatment*); HEART, SURGERY OF (*Foreign Bodies*); HIP-JOINT, INJURIES OF (*Epiphyseal Separations, Diagnosis*); HYDATID DISEASE (*Diagnosis*); MEDIASTINUM (*Growths, Diagnosis*); ORBIT, DISEASES OF (*Injuries, Diagnosis of Foreign Bodies*); OSTEO-ARTHROPATHIES (*Pulmonary, Diagnosis*); PLEURA, AFFECTIONS OF, SURGICAL (*Empyema, Diagnosis*); RHEUMATISM, RHEUMATOID ARTHRITIS (*Morbid Anatomy*); RODENT ULCER (*Treatment*); SHOULDER, DISEASES AND INJURIES OF (*Dislocations, Diagnosis*); SKIN, TUBERCULOSIS OF (*Lupus Vulgaris, Treatment*); STOMACH, SURGICAL (*Foreign Bodies, Diagnosis*); TEETH (*Exostosis, Diagnosis*); TUMOURS, INOPERABLE, TREATMENT (*Phototherapy*).

Their Nature and Production

THOUGH seven years have elapsed since Prof. Röntgen made his wonderful discovery, very little has comparatively been since added to our knowledge as to their physical nature or as to our means of generating them.

Though their nature is still unknown, evidence has been accumulating in favour of Sir George Stokes' theory, that they are due to irregular impulses in the ether comparable acoustically to a noise. Thus it has been shown that they travel with the velocity of light (Blondlot), but they differ from ordinary light in not being capable of regular reflection, refraction, or polarisation;[1] they can, however, excite fluorescence, affect a photographic plate, and discharge an electrified body, but these are the chief properties of violet and ultra violet light, so that the Röntgen rays more closely resemble ultra violet light than ordinary visible light.

There are altogether four chief qualities of radiation which are used in medicine, and they can be tabulated as follows:—

1. Radiant heat rays (invisible).
2. Ordinary light.
3. Ultra violet light (invisible).
4. Röntgen rays (invisible).

The Effluve, or brush discharge, produced by high frequency currents has some of the properties of ultra violet light. About three of these forms of radiation a good deal is known; thus if we examine the spectrum of white light, we find it consists of seven colours which are arranged with the radiant heat rays and the ultra violet rays as follows:—

Invisible.—Radiant heat.

Visible Spectrum.—Red, orange, yellow, green, blue (Finsen rays), indigo (Finsen rays), violet (Finsen rays).

Invisible.—Ultra violet (Finsen rays).

The rays are arranged in this table according to their wave lengths; thus the radiant heat rays have the longest wave length and the ultra violet the shortest. Our knowledge is not at present sufficiently definite to permit us to assign a position in this table to the Röntgen rays, but they have, as we have seen, the leading properties of the invisible ultra violet rays. They differ chiefly in their powers of penetration. Ultra violet rays have but little penetrative power as regards our tissues, and are absorbed by the thinnest film of blood, while the Röntgen rays pass through our skin and soft structures with great facility, but throw shadows of the bones. Transparency to Röntgen rays is a question of density; all light bodies are transparent and all heavy bodies are opaque. In the following table of densities an attempt has been made to roughly indicate in order the relative transparency of bodies, but it must be remembered that transparency and opacity are only questions of a degree, and that a thin film of an opaque body would permit some of the rays to go through, while a thick film of a transparent body would cast a shadow.

Quite opaque.—Platinum, mercury, bismuth, lead, silver.

Opaque.—Copper, iron, zinc.

Semitransparent. — Flint glass, aluminium, quartz, crown glass, calculi of lime oxalate, calculi of phosphates, bone, calculi of uric acid.

Transparent. — Biliary calculi, cutaneous tissue, tendons, arteries, muscles, blood, veins, nerves, brain, pus, urine, milk, water, human fat, wood, etc.

The above table is one of the order of relative transparency; thus calculi of oxalates are more opaque than calculi of phosphates or of urates.

[1] Blondlot has recently shown that the rays are polarised.

The order of transparency is not always the same as the order of density; thus uric acid calculi are more dense than calculi of phosphates, but are more transparent (Swain). Biliary calculi have much the same transparency as the skin, and are therefore very difficult to photograph *in situ* unless there be a thick mass of them. In actinic effect the Röntgen rays are far behind violet and ultra violet light; it has been calculated that they have only one-sixtieth the actinic effect of a candle. In producing fluorescence and in discharging electrified bodies they are, however, more powerful; they cause barium platinocyanide to fluoresce with a yellowish green, while ultra violet light causes it to shine with a brilliant green. The Röntgen rays will discharge a positively or a negatively charged body; the ultra violet light, unless of high refrangibility, will only discharge a negatively electrified body provided with a clean zinc or other special metallic surface, and will only do this at a comparatively short range. Further, the Röntgen rays appear to have no bactericidal effect, while ultra violet light can kill cultures of the staphylococcus pyogenes aureus in four seconds.

Lastly, they differ in their clinical effect; ultra violet light produces a transient hyperæmia or erythema, and sometimes some pigmentation, the granulomatous tissue of lupus has in time its vitality destroyed and undergoes degeneration (Macleod), but under no circumstances does destruction of healthy tissue follow.

The Röntgen rays, if pushed, or in susceptible people, will produce severe dermatitis and intractable ulcers; changes in the nails, falling off of the hair, and pigmentation are common. The Röntgen ray dermatitis is a cumulative effect, and it may not come on until a week or more after the cessation of the exposures, hence great caution is necessary. A patient may have had half-a-dozen exposures of five to ten minutes each, and may go away with apparently no risk of dermatitis, but in a few days' time may suffer from severe smarting and burning sensations. The skin becomes red, then dusky purple and œdematous, and it may give way and be followed by an indolent suppurating sore. This dermatitis may be prolonged, and it will not yield to remedies.

The pathological changes are mainly confined to the blood-vessels, which become dilated and degenerated; thrombosis follows; and dense bands of connective tissue are formed. The hair follicles and sebaceous glands disappear. *See also* p. 576.

Mode of Production of the Rontgen Rays

The rays, so far as we are at present aware, can only be produced by passing electrical discharges through highly exhausted vacuum tubes. In time to come we may, perhaps, be able to make use of the radiation of certain uranium compounds, or of radium; their bodies yield a constant radiation akin to the X radiation, but in feeble amount.

At present we require electricity of high potential and properly constructed vacuum tubes.

There are two chief methods for producing the electricity; the one method is to make use of a static influence machine, the other is to use an induction coil.

There are several kinds of influence machine, the Holtz, Voss, Wimshurst, Pidgeon, and Gaiffe.

In this country the Wimshurst is in most use, but it is probable that the Pidgeon will displace it.

Whichever machine be chosen it is essential that the plates be of large size—20-36 inches in diameter—and many in number. A Wimshurst (Fig. 1) machine for this purpose might consist of ten 24-inch plates, but larger plates would be an advantage. The plates are best made of a special quality of glass. Gaiffe uses ten ebonite

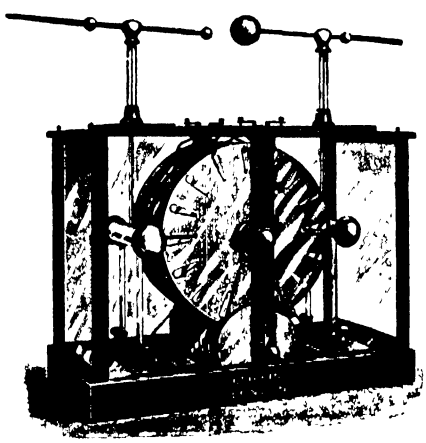


Fig. 1.—Wimshurst influence machine. Griffin.

plates, and the advantage of this is that there is no risk of breaking the plates by a too rapid rotation; thus owing to the great rate at which the plates can be revolved the output is considerably greater. The objection to the use of ebonite is that it is liable under the influence of light to lose its surface insulation, and also to warp, and therefore for a permanent installation, and where portability is not concerned, glass is to be preferred.

The terminals of the ordinary vacuum tube are not very suitable for use with a static machine; it is better to have a tube with ball-shaped terminals, and to allow a small spark gap to intervene on either side between the wires attached to the influence machine and the terminals of the tube. A good deal will depend upon the proper arrangement of these spark gaps, and their lengths should be varied until the tube yields the best radiation; sometimes the tube will work better without them. For Gaiffe's influence machine a special tube with cylindrical terminals called the Chabaud-Villard is supplied. These sources of difficulty, together with the trouble often

occasioned by the capricious yield of the influence machine, are absent when an induction coil is made use of. The Leyden jars of the influence machine should be disconnected, otherwise a succession of very bright flashes will be produced instead of a continuous discharge. An advantage of the influence machine is that, unless punctured by a stray spark, the life of the tubes is longer, because the current passed through them is smaller and the heating effect less. There is also an absence of flickering, so that for screen work they are less tiring to the eye. It is also an advantage to be able to do without accumulators or primary batteries, and to generate one's own electricity; still, on the whole, most operators, including the writer, prefer the induction coil.

If the induction coil method be preferred, a set of accumulators, or a primary battery, or a connection to the electric lighting mains will also be required.

The smallest induction coil which will be found of much use is one with a six-inch spark,

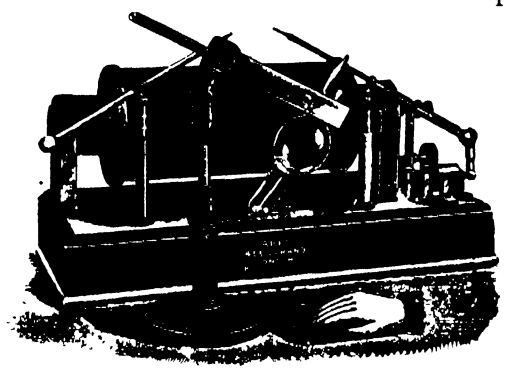


FIG. 2.—Induction coil. Turner's *Med. Electricity*.

a more satisfactory instrument will be one with a 10-inch spark (Fig. 2), and coils with 18- or 24-inch sparks are sometimes used. A great deal depends upon the quality and construction of the coil, and the instrument should be suited to the source of electric supply; thus a coil wound to run off a few accumulators will not yield the best effects when fed by a high voltage current from the mains. A good maker of coils is Apps, 433 Strand, London. When electrolytic interrupters are used it is sometimes an advantage to be able to vary the self-induction of a coil; this can be done by altering the connections of the layers of the primary winding. Schall, 55 Wigmore Street, London, supplies a German induction coil in which the terminals of the four layers of the primary wire are brought outside (Fig. 3), and which can be connected together either—

1. All in series,
2. All in parallel, or
3. Two layers in series, and the two groups in parallel.

In the first case the self-induction is at a maximum and the coil is suitable for soft tubes; the current at make is completely suppressed.

In the second case the self-induction is at a minimum, the current at make is not completely suppressed, but the full length of spark at break is obtained, and the coil is suitable for hard tubes.

In the third case an intermediate condition obtains.

In addition to the coil a current interrupter will be required; the old-fashioned Neef's hammer or vibrating spring which usually forms part of the coil can be used when the coil is fed by a few accumulators, and when intense effects are not required, but it cannot be used when the coil is attached to the electric light mains, and under all circumstances a separate motor interrupter is advantageous; still it is better not to buy a coil unless it is provided with a Neef's hammer, for the latter is often useful as a stand-by, and it is also the most portable of interrupters.

The chief forms of motor interrupters can be classified as follows:—

1. Mercurial { Dipping contact, Mackenzie Davidson, mercury jet or turbine.
2. Electrolytic { Wehnelt, Simon Caldwell, Campbell Swinton.
3. Rubbing contact between solids { Dawson Turner, Contremoulins-Gaiffe.

All these interrupters can be used either with

FIG. 3.—Arrangement for altering self-induction of coil. Schall.

high voltage currents from the mains or (excepting the electrolytic group) with low voltage currents from accumulators.

1. The simplest motor-driven mercury interrupter is the one with a vertical dipping contact (Fig. 4).

The advantage of this instrument is that it is simple, it is easily kept in order, and it only requires a small amount of mercury. It is, however, less rapid than the succeeding instruments.

In the Mackenzie Davidson interrupter (Fig. 5) the contact is made by an inclined rotating paddle which alternately dips into and out of the mercury. The paddle shaft is driven by a band attached to the motor shaft so as to insulate it from the motor, and to permit the motor to be fed by the accumulators that are supplying the induction coil. This instrument having a rotating instead of a reciprocating motion can be run very rapidly, and it is one

The order of transparency is not always the same as the order of density; thus uric acid calculi are more dense than calculi of phosphates, but are more transparent (Swain). Biliary calculi have much the same transparency as the skin, and are therefore very difficult to photograph *in situ* unless there be a thick mass of them. In actinic effect the Röntgen rays are far behind violet and ultra violet light; it has been calculated that they have only one-sixtieth the actinic effect of a candle. In producing fluorescence and in discharging electrified bodies they are, however, more powerful; they cause barium platinocyanide to fluoresce with a yellowish green, while ultra violet light causes it to shine with a brilliant green. The Röntgen rays will discharge a positively or a negatively charged body; the ultra violet light, unless of high refrangibility, will only discharge a negatively electrified body provided with a clean zinc or other special metallic surface, and will only do this at a comparatively short range. Further, the Rontgen rays appear to have no bactericidal effect, while ultra violet light can kill cultures of the staphylococcus pyogenes aureus in four seconds.

Lastly, they differ in their clinical effect; ultra violet light produces a transient hyperæmia or erythema, and sometimes some pigmentation, the granulomatous tissue of lupus has in time its vitality destroyed and undergoes degeneration (Macleod), but under no circumstances does destruction of healthy tissue follow.

The Rontgen rays, if pushed, or in susceptible people, will produce severe dermatitis and intractable ulcers; changes in the nails, falling off of the hair, and pigmentation are common. The Röntgen ray dermatitis is a cumulative effect, and it may not come on until a week or more after the cessation of the exposures, hence great caution is necessary. A patient may have had half-a-dozen exposures of five to ten minutes each, and may go away with apparently no risk of dermatitis, but in a few days' time may suffer from severe smarting and burning sensations. The skin becomes red, then dusky purple and œdematous, and it may give way and be followed by an indolent suppurating sore. This dermatitis may be prolonged, and it will not yield to remedies.

The pathological changes are mainly confined to the blood-vessels, which become dilated and degenerated; thrombosis follows; and dense bands of connective tissue are formed. The hair follicles and sebaceous glands disappear. *See also* p. 576.

Mode of Production of the Rontgen Rays

The rays, so far as we are at present aware, can only be produced by passing electrical discharges through highly exhausted vacuum tubes. In time to come we may, perhaps, be able to make use of the radiation of certain uranium compounds, or of radium; their bodies yield a constant radiation akin to the X radiation, but in feeble amount.

At present we require electricity of high potential and properly constructed vacuum tubes.

There are two chief methods for producing the electricity; the one method is to make use of a static influence machine, the other is to use an induction coil.

There are several kinds of influence machine, the Holtz, Voss, Wimshurst, Pidgeon, and Gaiffe.

In this country the Wimshurst is in most use, but it is probable that the Pidgeon will displace it.

Whichever machine be chosen it is essential that the plates be of large size—20-36 inches in diameter—and many in number. A Wimshurst (Fig. 1) machine for this purpose might consist of ten 24-inch plates, but larger plates would be an advantage. The plates are best made of a special quality of glass. Gaiffe uses ten ebonite

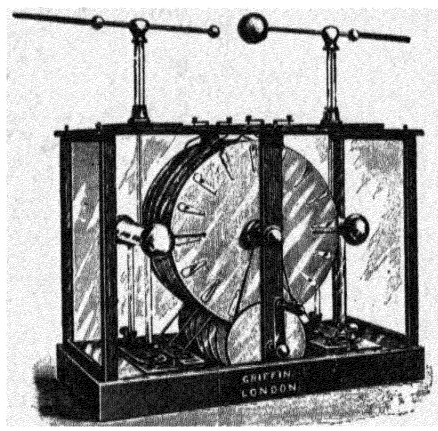


Fig. 1 Wimshurst influence machine. Griffin.

plates, and the advantage of this is that there is no risk of breaking the plates by a too rapid rotation; thus owing to the great rate at which the plates can be revolved the output is considerably greater. The objection to the use of ebonite is that it is liable under the influence of light to lose its surface insulation, and also to warp, and therefore for a permanent installation, and where portability is not concerned, glass is to be preferred.

The terminals of the ordinary vacuum tube are not very suitable for use with a static machine; it is better to have a tube with ball-shaped terminals, and to allow a small spark gap to intervene on either side between the wires attached to the influence machine and the terminals of the tube. A good deal will depend upon the proper arrangement of these spark gaps, and their lengths should be varied until the tube yields the best radiation; sometimes the tube will work better without them. For Gaiffe's influence machine a special tube with cylindrical terminals called the Chabaud-Villard is supplied. These sources of difficulty, together with the trouble often

occasioned by the capricious yield of the influence machine, are absent when an induction coil is made use of. The Leyden jars of the influence machine should be disconnected, otherwise a succession of very bright flashes will be produced instead of a continuous discharge. An advantage of the influence machine is that, unless punctured by a stray spark, the life of the tubes is longer, because the current passed through them is smaller and the heating effect less. There is also an absence of flickering, so that for screen work they are less tiring to the eye. It is also an advantage to be able to do without accumulators or primary batteries, and to generate one's own electricity; still, on the whole, most operators, including the writer, prefer the induction coil.

If the induction coil method be preferred, a set of accumulators, or a primary battery, or a connection to the electric lighting mains will also be required.

The smallest induction coil which will be found of much use is one with a six-inch spark,

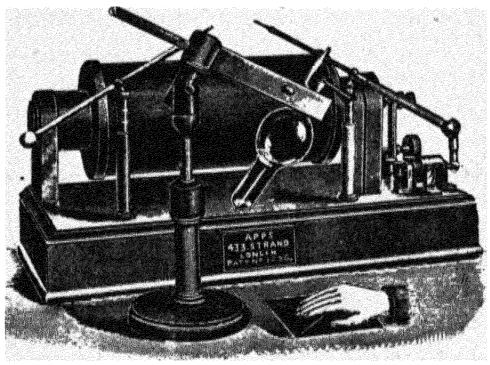


FIG. 2.—Induction coil. Turner's *Med. Electricity.*

a more satisfactory instrument will be one with a 10-inch spark (Fig. 2), and coils with 18- or 24-inch sparks are sometimes used. A great deal depends upon the quality and construction of the coil, and the instrument should be suited to the source of electric supply; thus a coil wound to run off a few accumulators will not yield the best effects when fed by a high voltage current from the mains. A good maker of coils is Apps, 433 Strand, London. When electrolytic interrupters are used it is sometimes an advantage to be able to vary the self-induction of a coil; this can be done by altering the connections of the layers of the primary winding. Schall, 55 Wigmore Street, London, supplies a German induction coil in which the terminals of the four layers of the primary wire are brought outside (Fig. 3), and which can be connected together either—

1. All in series,
2. All in parallel, or
3. Two layers in series, and the two groups in parallel.

In the first case the self-induction is at a maximum and the coil is suitable for soft tubes; the current at make is completely suppressed.

In the second case the self-induction is at a minimum, the current at make is not completely suppressed, but the full length of spark at break is obtained, and the coil is suitable for hard tubes.

In the third case an intermediate condition obtains.

In addition to the coil a current interrupter will be required; the old-fashioned Neef's hammer or vibrating spring which usually forms part of the coil can be used when the coil is fed by a few accumulators, and when intense effects are not required, but it cannot be used when the coil is attached to the electric light mains, and under all circumstances a separate motor interrupter is advantageous; still it is better not to buy a coil unless it is provided with a Neef's hammer, for the latter is often useful as a stand-by, and it is also the most portable of interrupters.

The chief forms of motor interrupters can be classified as follows:—

1. Mercurial { Dipping contact, Mackenzie Davidson, mercury jet or turbine.
2. Electrolytic { Wehnelt, Simon Caldwell, Campbell Swinton.
3. Rubbing contact between solids { Dawson Turner, Contremoulins-Gaiffe.

All these interrupters can be used either with

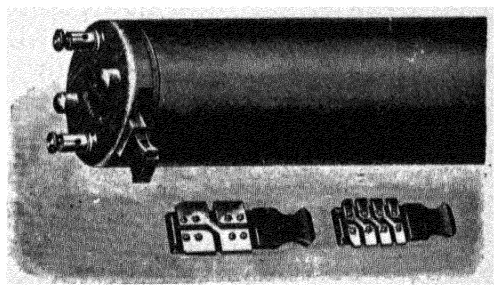


FIG. 3.—Arrangement for altering self-induction of coil. Schall.

high voltage currents from the mains or (excepting the electrolytic group) with low voltage currents from accumulators.

1. The simplest motor-driven mercury interrupter is the one with a vertical dipping contact (Fig. 4).

The advantage of this instrument is that it is simple, it is easily kept in order, and it only requires a small amount of mercury. It is, however, less rapid than the succeeding instruments.

In the Mackenzie Davidson interrupter (Fig. 5) the contact is made by an inclined rotating paddle which alternately dips into and out of the mercury. The paddle shaft is driven by a band attached to the motor shaft so as to insulate it from the motor, and to permit the motor to be fed by the accumulators that are supplying the induction coil. This instrument having a rotating instead of a reciprocating motion can be run very rapidly, and it is one

of the best interrupters known. A disadvantage is the very large amount of mercury required.

In the mercury jet interrupter (Fig. 6) there is a small centrifugal pump below, which throws up a constant fountain of mercury along which

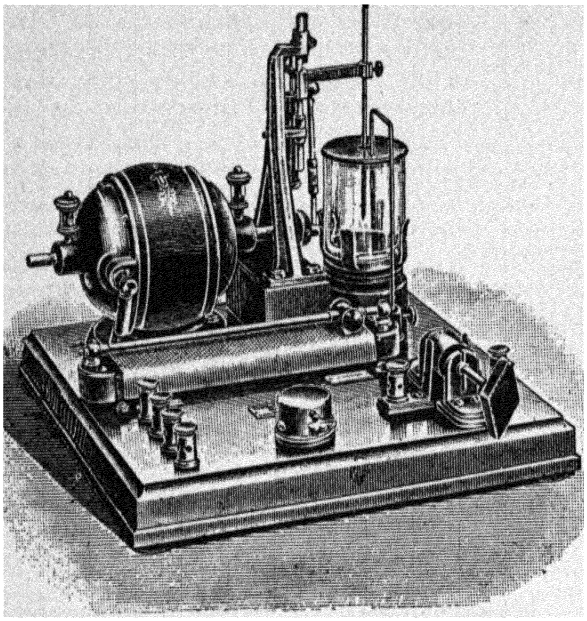


FIG. 4.—Dipping contact interrupter. Schall.

the current passes; the current is broken by the interposition of two insulating fibre arms whose plane of rotation is at right angles to the direction of the jet. This is also an excellent interrupter, but it suffers from the disadvantage of requiring a large amount of mercury. This makes it very heavy: in fact, the complete instrument is as much as a man can conveniently carry.

All the above-mentioned mercurial interrupters suffer from certain defects which appear to be inseparable from the use of mercury; the mercury becomes oxidised, pulverised, and churned up with the paraffin or alcohol so that the insulating properties of the paraffin are impaired and a complete break is not obtained; further, in the case of the mercury jet, the emulsified mercury is thrown up and deposited upon the insulating fibre supports, which have accordingly to be cleaned —not a pleasant operation while a patient is waiting. Care must, therefore, be taken to keep the mercury in these instruments clean by washing with water or dilute sulphuric acid.

2. A new group of interrupters were first introduced by Dr. Wehnelt (Fig. 7), and we owe modifications of the original instrument to Simon Caldwell, Campbell Swinton, and others.

The principle of the instrument is electrolytic. Into a large jar of dilute sulphuric acid are placed a good-sized lead plate and a small platinum wire, which can be made to project through a tight-fitting aperture; the diameter of the platinum wire and the amount of projection are important, and in later instruments three or more platinum wires of different diameters (1, 2, and 3 mm.) are provided (Fig. 7). Any one of these can be used as desired to suit the conditions of the voltage. The lead plate must be made the kathode, and the platinum wire the anode. The interrupter can be used when the voltage of the current supplied is not less than thirty (fifteen accumulators), and it can be used directly from the electric light mains (100-230 volts). No mechanical interrupter can approach this one in the rapidity of the interruptions, and the discharge at the secondary terminals takes the form rather of a continuous flame than of a succession of sparks (Fig. 8). It is thus exceedingly powerful. The condensers of the coil should be detached during its use. It suffers from the disadvantage of not being wholly reliable; sometimes it will not work steadily, it tires and the acid grows hot, and the powerful discharges set up overheat the anode of the tube. The interruptions are perhaps due to bubbles of gas produced by electrolysis at the anode; the spheroidal state may also play a part. The tiny bubble of gas will cut off the current, but as soon as the current stops the bubble is absorbed and the current starts again. In other forms of this break the small platinum wire is replaced by a lead plate, and the interruptions are produced by forcing the current to pass through a small aperture in a plate of porcelain which divides the cell into two parts. The

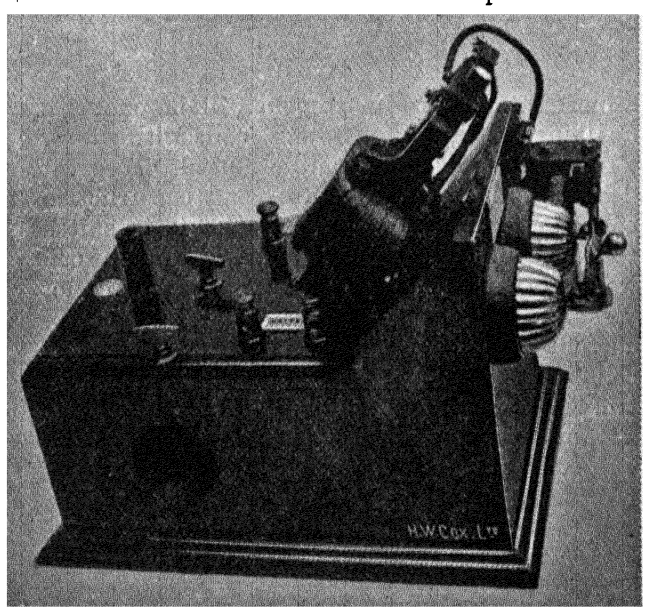


FIG. 5.—Mackenzie Davidson Interrupter. Walrh's *Rontgen Rays*.

Wehnelt break is of service when it is desired to give a very short exposure, as in photographing the teeth.

3. More recently rubbing contact interrupters between solids have been introduced by Dawson Turner and Gaiffe.

In the Dawson Turner break (Fig. 9) there

are two rollers of brass or copper, which are alternately brought into contact and separated by a small electric motor; as the rollers rotate

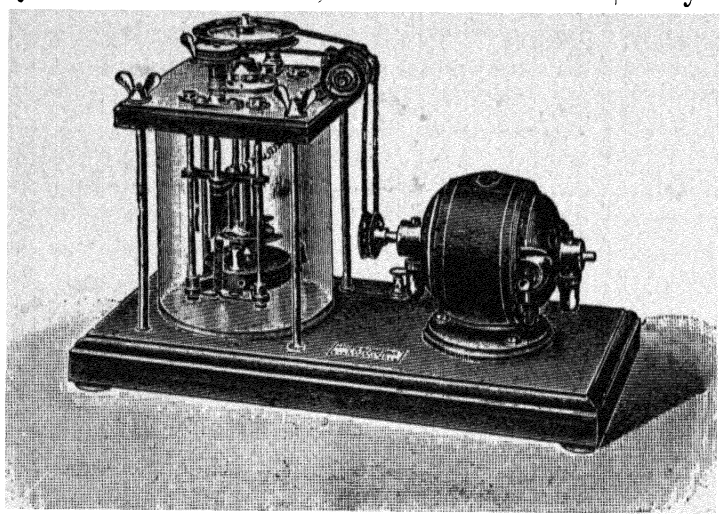


FIG. 6.—Mercury jet interrupter. Turner's *Med. Electricity.*

the break is constantly made at a fresh point, and the burning away is distributed evenly all round. The rollers must be immersed in paraffin, alcohol, or water. If the noise made by this interrupter be not objected to, it will be found a satisfactory and portable instrument; there is no possibility of short circuiting, and there is no mercury to be oxidised and thrown into waves.

Contremoulins and Gaiffe have constructed a rubbing contact interrupter between copper and

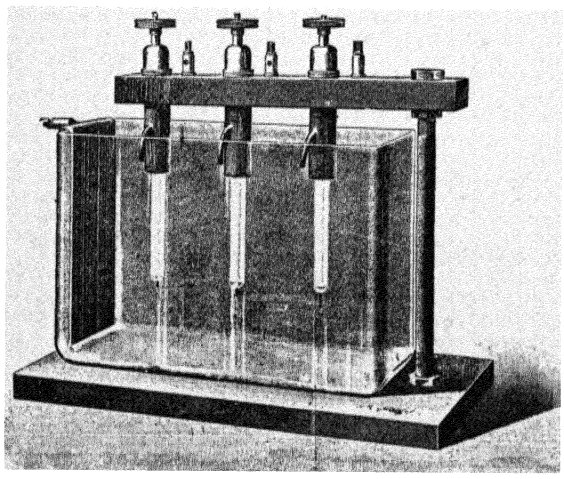


FIG. 7.—Wehnelt interrupter, new pattern. Schall.

carbon. A tube of copper is divided longitudinally into two halves, and is rotated by a motor; two carbon brushes are pressed by springs against the surface of the copper, the current will pass when both brushes are on the same half of the copper, and will be broken when they are not. By moving one of the brushes the contact can be increased or diminished. The apparatus is immersed in alcohol or paraffin. This is a silent instrument, but as the burning away always occurs at the same place, the brushes and copper cannot last very long.

The supply of electricity for the coil may be obtained from primary cells, from accumulators, or by a branch circuit attached to the electric light mains. Primary cells should, if possible, be avoided; if, however, circumstances compel their employment, five to ten large bichromate or Bunsen cells are the best; the bichromate cells (Fig. 10) may be purchased in sets of five provided with a lifting arrangement, and the zincs should only be allowed to remain in the acid solution during use of the coil. The solution will require frequent renewal, and the zincs must be kept well amalgamated. Bunsen cells give a more continuous current, but they have to be set up before and dismantled after every use. Accumulators are much to be preferred; five to ten will be found sufficient; the "q" type of the Electrical Power Storage Company or the lithanode are so sealed as to be fairly portable. Accumulators can only be employed when there is some means at hand for recharging them. If the electric light mains

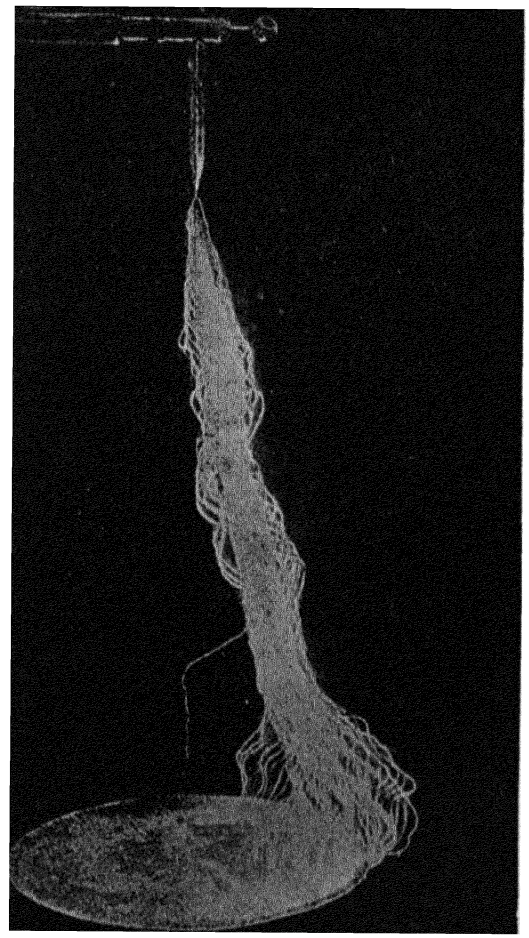


FIG. 8.—Wehnelt spark. Turner's *Med. Electricity.*

(continuous current) are laid on, the coil may be fed by placing it on a branch circuit with a sufficient resistance, but one of the high voltage breaks will be required. Schall sells complete

resistance boards for this purpose (Fig. 11). Though this is the simplest and least troublesome method, it is not so advantageous as the use of accumulators: a larger current strength can be obtained from the latter and a higher voltage from the former, and the large current strength is to be preferred; further, the Neef's hammer is available with accumulators.

Rontgen ray tubes have been recently improved in strength and durability, and in means of keeping the degree of vacuum constant. In its simplest form the tube consists of a cylinder or bulb of glass through the ends of which platinum wires pass; the latter are continued as aluminium rods. One of these, the kathode, terminates in a small concave mirror; the other, the anode, is a thick disc of platinum inclined at an angle of 45° to the axis of the kathode, and at a distance a little beyond the principal focus of the concave mirror. The tube is exhausted to a very high vacuum. The glass of which the tube is made must be quite free from lead. When the discharge from an induction coil is passed in the right direction through such a tube, that part of the wall of the tube which is opposite to the anode will shine with a greenish yellow phosphorescence of hemispherical form; if the tube be wrongly connected the green light will be patchy and irregular.

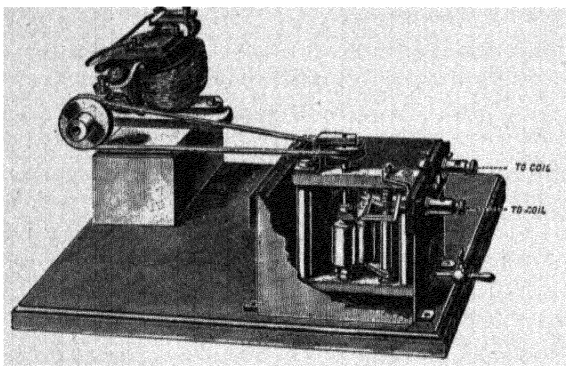


FIG. 9.—Dawson Turner interrupter Turner's *Med. Electricity.*

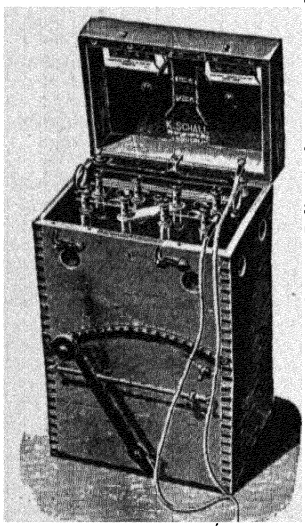


FIG. 10.—Bichromate battery with lifting arrangement. Schall.

Modern tubes have three electrodes and sometimes arrangements for varying the vacuum. The Chabaud-Villard tube for Gaiffe's influence machine is provided with such a means. A good maker of tubes is Hirschmann of Berlin (Fig. 13). At one end of the tube is an arrangement for admitting air; and at the other end one for withdrawing air; either can be used by causing the electricity to pass solely through the particular arrangement. Such a tube is a great advantage, for most tubes unless punctured become gradually useless through their electrical resistance rising owing to a too high vacuum. Another good maker of tubes is Mueller of Hamburg; his tubes are sold by Cox of Cursitor Street, London, and by Leslie Miller, Hatton Garden, London. To Mueller's tube was awarded the gold medal of the Rontgen Society in 1901. The conditions of this competition serve to bring out what are considered to be the important points in a Rontgen ray tube. These are:—

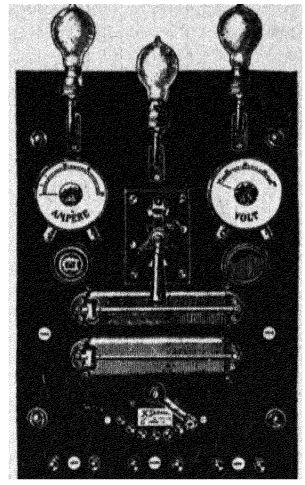


FIG. 11.—Switchboard for supplying coil from the main. Schall.

(1) Definition.
(2) Penetration.
(3) Photographic effect.
(4) Price.

The predominant requirement was that the tube selected as the best should be practical. About 75 per cent of the tubes failed to satisfy the required tests for definition, and in the opinion of the jury the attention of makers of tubes should be drawn to this defect, because unless a tube defines well, all other advantages it may possess are to a great extent valueless for most of the purposes to which X-rays are put.

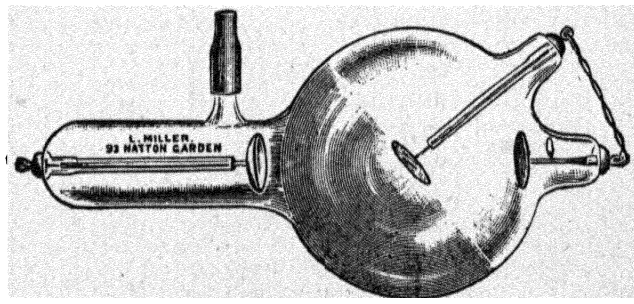


FIG. 12.—Rontgen ray tube. L. Miller.

The tubes which stood the test for definition were subjected to a very severe trial for penetration and photographic effect, and the jury were glad to be able to report that a much higher standard of excellence was attained in this respect than in that of definition. The element of price did not require very serious consideration. The prices of the tubes sent in varied from £4, 5s. to 16s. Many of the high-priced tubes were eliminated by the first test, and the tube which best satisfied all the tests

applied by the jury was the one sent in as Cox's Record tube, made by C. H. F. Mueller of Hamburg (*Archives of the Röntgen Ray*, August 1901). The only fault the writer has to find with these tubes is that the resistance seems to increase rather rapidly. The only satisfactory way of reducing the resistance of a tube is to lower the vacuum, and if the tube is not provided with a means for letting in more air it should be returned to the maker, who will first allow it to refill with air and then re-exhaust it. A strictly temporary method of lowering the resistance of a tube is to heat it

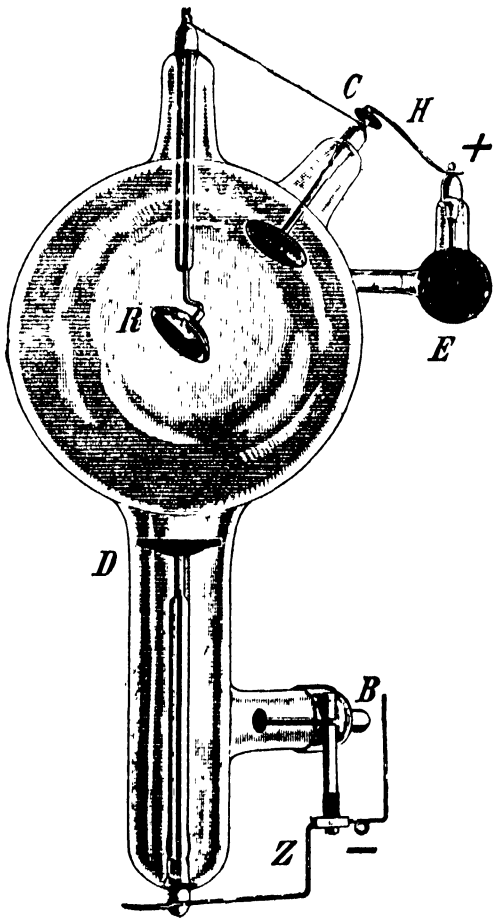


FIG. 13.—Röntgen ray tube with arrangement for altering the vacuum. Hirschmann.

with a large spirit flame; the flame should be passed boldly and rapidly all over the tube; this remedy only serves to aggravate the existing disease, for though effectual for the moment the resistance will be found afterwards to be higher than ever; other methods suggested for lowering the resistance are not satisfactory.

Tube-holders, fluorescent screens, and photographic plates are also required. Convenient tube-holders are supplied by Schall (Fig. 15); a table tube-holder should be about 14 inches in height and its arm about 2 feet in length. One of the best makers of fluorescent screens is Johnson and Matthey of Hatton Garden, London; a convenient size is 14 × 16 inches. The screen is coated with barium platinocyanide on one side and with black paper on the other; the coated side should be protected by a glass or celluloid cover.

Any good make of photographic plate will suffice; some of those the author has used are those supplied by Lumière, Edward's Cathodal, Paget, Imperial, and Ilford, and Wrotten and Wainwright. It is important to stick to the same plate and developer. The plates should be fresh and be coated with a thick film. The author (see *Electrician*, February 1897) has not found any advantage result from using very rapid plates; though a shorter exposure may suffice, the resulting picture is not so good.

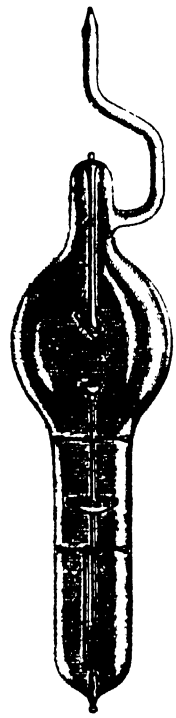


FIG. 14.—Röntgen ray tube with movable cathode. Dawson Turner.

To protect the plates from light they should be enclosed in light-tight envelopes, or in a specially made case, and the object to be photographed should be placed in close contact with the envelope, with the anode of the tube directly above the centre of the object and twelve or more inches away. The film side of the negative should be uppermost. After the exposure, which may vary from a few seconds for the hand to four or five minutes for the pelvis and hip-joint, the plates are developed in the ordinary way. Hydroquinone is to be preferred to pyrogallic acid, because it produces hard negatives, and a slow development is recommended; in fact it is customary on the Continent to dilute the developer some thirty times with water, and to put the plate aside in a box for some three-quarters of an hour or more, and if the negative be still too thin to add a little normal strength developer. The satisfactory development of Röntgen ray negatives requires a great deal of care and considerable experience, and this is particularly the case in the skiagraphy of obscure bone diseases and of the thorax and abdomen.

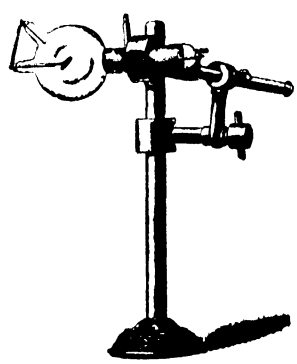


FIG. 15.—Tube-holder for tube.

THE RÖNTGEN RAYS IN DIAGNOSIS AND TREATMENT

The marvellous power of the Röntgen rays of penetrating our tissues, and of rendering them, so to speak, transparent, led obviously to their application in surgical and medical diagnosis;

that they would have an important place in therapeutics was not until comparatively recently even suspected, and yet it is probable that they are likely to be more serviceable to mankind in the latter respect than in the former.

For diagnostic purposes the use of the fluorescent screen or of skiagraphy is employed.

When the screen is to be used the room should be quite dark, hence I prefer, if possible, to do screen work at night, for the pupil is then also more dilated. Screens are sometimes enclosed in light-tight boxes which fit closely to the eyes; these are called cryptoscopes, and they permit of the use of the screen in broad daylight; still, for difficult cases, it is much better to darken the whole room.

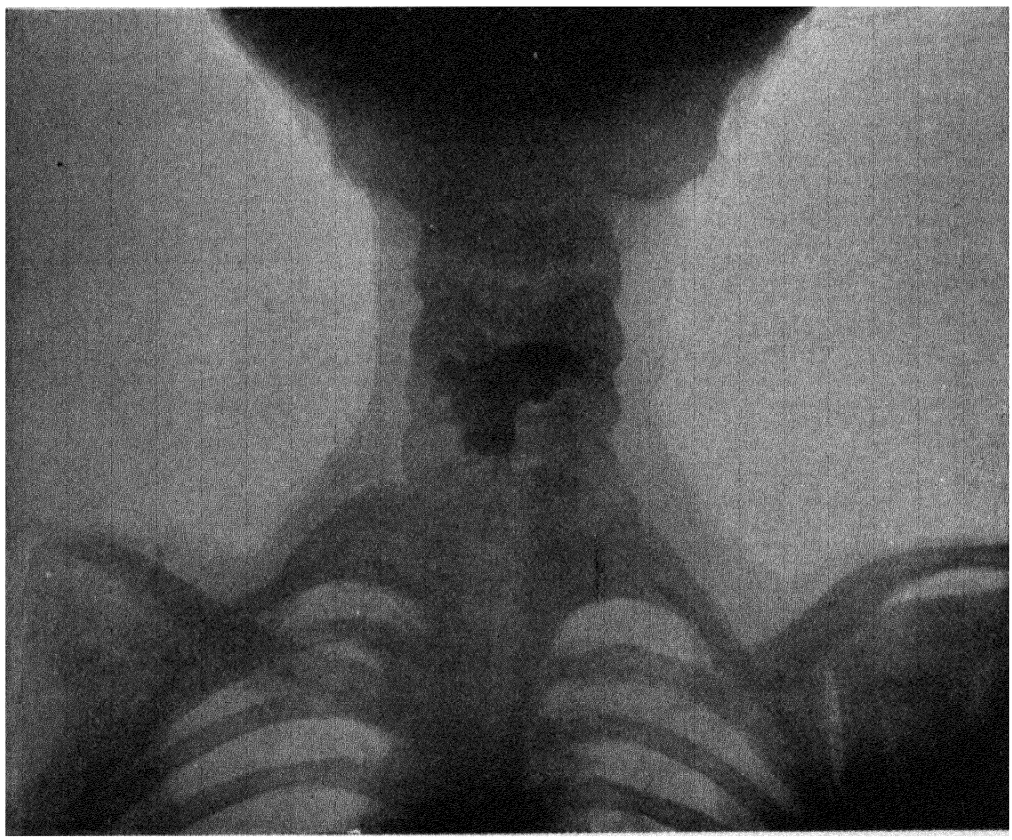


FIG. 16.—False teeth in œsophagus.

The object to be examined should be placed between the screen and the tube and as close to the black side of the screen as possible; a little practice with the hand will soon give the operator the requisite skill, and show him how to obtain the best effect without distortion of the shadows. If the tube has a low resistance equal to about a two-inch alternative spark gap, the skin will throw a somewhat dark shadow, but if the resistance of the tube be high, the skin will be very faintly outlined, and even the bones will be transparent. For children, for the thinner parts of adults, and to bring out soft structures, a tube of low resistance should be used; for other cases a medium hard tube. By referring to the table on page 565 a good idea can be formed as to the kind of foreign body that will be visible in the tissues; thus a fragment of glass might be seen in the wrist or hand, but might be invisible in a thicker part. A small piece of lead might, however, be located in no matter how thick a part. The exact location of foreign bodies, such as a needle or a bullet, in an extremity is most easily effected by examining say the arm from two different positions at right angles to one another, and making four marks on the skin; these marks are made by holding the end of a probe in the direct line of the shadow of the foreign object, the light is then turned up and the position of the probe marked. It should be remembered also that the nearer the foreign body is to the screen the smaller and the more defined will be its shadow. For more exact localisation the author's apparatus and method may be used (Turner's *Medical Electricity*, 3rd edition, p. 344), or that of Dr. Mackenzie Davidson, who has done more than any one else to make localisation precise and accurate (*Archives of the Röntgen Ray*, May 1898).

Examinations of a patient may be very rapidly made by means of the screen; the extremities and the thorax present little difficulty to a practised eye; when, however, the hip-joint or abdomen are in question it will be better to trust to photography, and in all obscure cases the screen examination should be followed by one or two photographs. The thorax should be examined both with the screen on the sternum and on the back, and the patient should be completely stripped; the ribs, vertebral column, and scapulæ will be seen, and the heart less definitely; the pulsations of the heart can be counted and the rise and fall of the diaphragm observed. Phthisical consolidations throw a denser shadow than the normal lung, and give rise to a peculiar marbling and mottled appearance. Cavities betray themselves as light areas with dark borders unless

they contain pus, when they are uniformly dark. According to Dr. Hugh Walsham an earlier diagnosis of the presence of tubercle can be made by the Röntgen rays than by ordinary physical signs. Amongst other conditions which can be noticed are fibroid phthisis, emphysema, pleurisy with effusion, intra-thoracic tumours, and displacement of the heart and liver. The smallness of the heart and the diminished excursion of the diaphragm in phthisis should be noted. Screen examinations are more valuable than photography in many of these cases, particularly in the differential diagnosis of aneurysm (Fig. 17), in which case a uniform pulsation may be seen, the margin of the shadow can be compared with the dull area, the patient can be examined at once both from the front and from the back and obliquely.

FIG. 17.—Aneurysm of ascending aorta.

Photography by X-rays is a more difficult and troublesome operation. There is the question of the distance of the tube, the position of the tube and plate, and the length of the exposure; further, there is the difficulty of proper development. Nothing but practice in these procedures will give successful results. To take the case of the photography of renal calculi; it will be seen by reference to the table on page 565 that oxalates and phosphates will be most easily shown. Mr. W. H. Shenton has published in Guy's Hospital Reports an analysis of 200 of such photographs with the following results:—

Cases in which the rays and surgeon found calculi, 28.

Cases in which the surgeon found calculi, but the rays did not, 8.

Cases stated not to have calculi by the rays, and operated on with negative results, 11.

Cases in which the rays found calculi and the surgeon did not, 2.

In 39 cases the rays were correct, and in 8 they were wrong.

Mr. Shenton's technique is to take the photograph in the prone position with the tube underneath the couch and the negative on the patient's back. A preliminary use of the screen is made, the tube is placed six inches from the abdomen, and an exposure of a minute or less is given.

The author's practice in the Edinburgh Royal Infirmary is as follows:—The patient must first be prepared by having the bowels thoroughly emptied, he is then placed in the supine position with the negative under his back, the centre of the negative should be about under the umbilicus; the respiratory movements of the abdominal contents are limited by a light flannel binder, the anode of the tube is placed over the umbilicus and about 8 inches away, and an exposure of two minutes is given. It is better, if possible, to take three negatives, and depending upon the stoutness of the patient, to give the first an exposure of one minute, the second of two, and the third of three minutes. The patient's knees should be brought up into a vertical position, and a medium or somewhat soft tube used. For the hip-joint a harder tube (for an adult) and a longer exposure of from three to five minutes are required. The negative should be supported in a case or on a flat board.

Fluorescent screens to shorten exposures have been used, and are frequently eulogised by dealers, but are unreliable and give granular negatives with want of sharp definition. Most operators, therefore, avoid them. They are generally made of finely powdered tungstate of

calcium gummed on to a flexible material; the prepared side should be placed against the film side of the negative, and both placed in the light-tight envelope together.

Curious markings in the form of wavy lines and streaks sometimes appear upon Röntgen ray negatives, and have not so far been satisfactorily explained, and this is another reason for avoiding the use of anything likely to impair the clearness of the result.

The value of the Rontgen rays in surgical affections of the bones and joints is well summed up by Dr. David Walsh in *The Röntgen Rays*, p. 132:—"To sum up; in dealing with bone injuries, successful radiography, compared with previous methods, offers the following advantages: It substitutes speed, accuracy, and finality for delay and doubt; it affords exact evidence, that may confirm or modify the diagnosis of the surgeon; it may furnish both grounds for prognosis and hints for treatment; it may save the patient the pain of useless, and perhaps dangerous manipulations as well as the shock of anæsthetics; it provides a permanent record of the precise nature of an injury; it may prove a safeguard for the patient and for his medical attendant both in the present and the future; and, lastly, it can hardly fail to be of value for teaching purposes."

FIG. 18.—Congenital dislocation, left hip-joint.

It may be pointed out that considerable practice is required for rightly interpreting Röntgen ray photographs, and that medical men would do well in such cases to call in the aid of an expert; more is usually to be learned from the negative than from the print, a little of the detail and contrast is lost in each transference, and in the print there is also lateral inversion; thus, if the left shoulder joint be photographed the negative will show it as the left, but the print will show it as the right. The rays are also of great value in the diagnosis of disease of the bones; in tubercular osteitis there is usually a lighter centre in the shaft due to rarefaction and a denser shadow on each side due to osteoplastic processes under the periosteum; in syphilitic osteitis the usual shadow is uniformly dense, due to a sclerosis, but in some cases of syphilis the bones are quite remarkably transparent, as in a case exhibited by the author at the Edin. Med. Chir. Society, Dec. 4, 1901. In tubercular joint disease it is common to see a general haziness and want of outline, and in advanced cases of wrist-joint disease the carpal bones may be quite transparent. Cartilaginous outgrowths and callus throw very faint shadows, so do gummata and sarcomata, but if they become ossified they will be distinctly revealed.

Dislocation and deformities require no special mention. Acromegaly is a condition which exhibits characteristic changes.

Röntgen Ray Therapeutics.—The number of the diseases which have been subjected to treatment by the X-rays is a constantly increasing one; they have been used in skin diseases, rodent ulcer, malignant disease of all kinds, phthisis, pulmonary and laryngeal, trachoma, tubercular glands and joints, sinus and fistula, spinal cord diseases, and for the relief of pain.

The skin diseases in which they have been found most serviceable are:—

Lupus, eczema chronic, favus, sycosis, hypertrichosis, lupus erythematosus, mycosis fungoides, xeroderma pigmentosum, etc.

Lupus.—The Rontgen ray treatment of lupus was preceded by the Finsen light treatment. "So long ago as September 1899, Dr. Finsen had treated 350 cases of this disease with a satisfactory result in 345. This result has been so certain and constant that there is even reason to doubt the accuracy of the diagnosis of lupus vulgaris when this method of treatment appears to be ineffective. The light treatment will only cure lupus of the skin, the hard palate, the front part of the septum nasi, tongue, and the mucous membrane of the cheek" (*Brit. Med. Journal*, Sept. 30, 1899).

When the disease is more deeply situated the Röntgen rays, in consequence of their penetrative power, can be used, but the rays can also be used for the superficial cases; in fact, it would nowadays appear highly probable that the rays will supersede the Finsen light entirely in the treatment of lupus. The rays produce their effect much more quickly, and that over a much larger area; an exposure of five minutes to the rays has as much or more effect as a fifteen-minute exposure to the Lortet and Genoud lamp, or an hour's exposure to the original Finsen lamp. It is, however, claimed that the cosmetic effect of the light is better than that of the rays; this is doubtful, but a more serious disadvantage of the rays is the risk there is of setting up a serious and intractable dermatitis. There are two methods of treating lupus by Röntgen rays: the one method is to expose the part to the rays alone; the other method is to combine the action of some medicament with that of the rays; this if applied in suitable cases much hastens the reaction and shortens the treatment.

If the first method be adopted a suitable mask of lead foil or of thin lead is so cut as to shade the parts that it is not desired to affect, and the patient so protected is placed about four to eight inches away from a medium or somewhat soft tube; an exposure of about five minutes is then given; this may be repeated daily for a week, and then three times a week, careful watch being kept for any signs of erythema. This usually requires about one hour's total exposure, but it may in susceptible cases and with hot tubes require less, while in other cases it may require much more. At the first sign of erythema treatment should be suspended, and the patient told to return in a week or fortnight, when the treatment will be resumed. This is at once the safest method of employing the rays, and the least painful and troublesome to the patient. The treatment in severe cases will have to be prolonged even for months, and there is a strong tendency to recurrence.

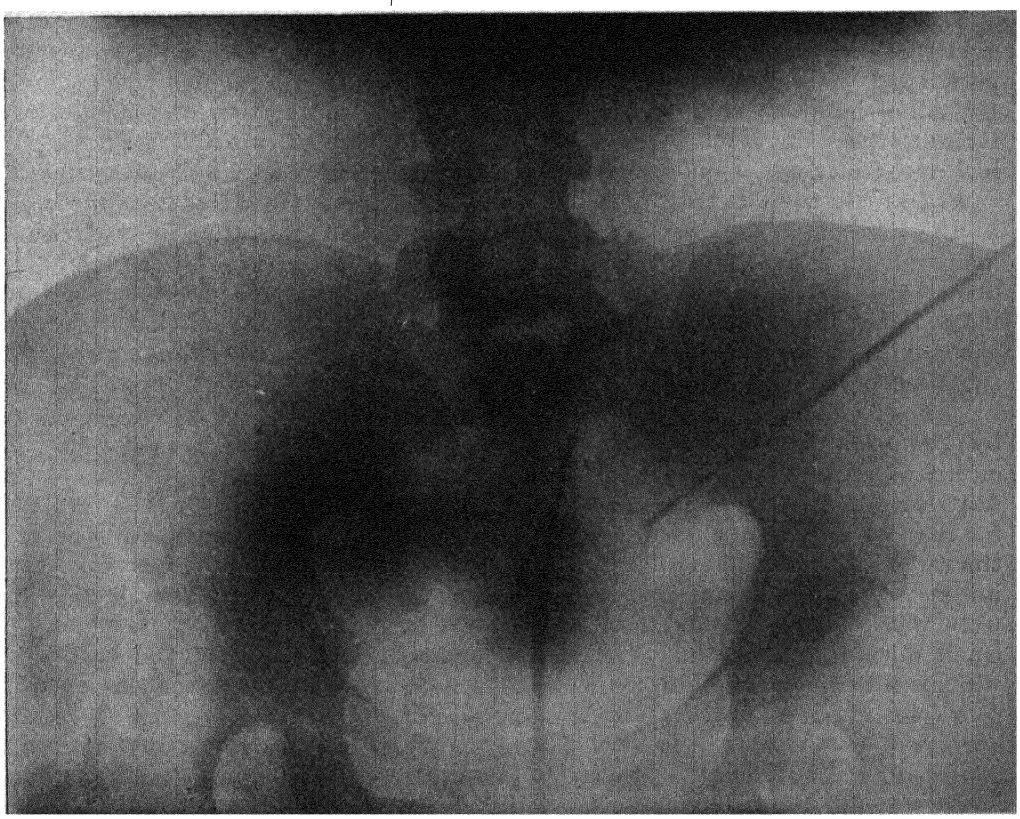


FIG. 19. Pelvic sinus with probe.

In the second method the affected part is first painted with tincture of iodine or carbolic lotion 1-20, or pure carbolic acid or Jarisch's ointment, and then protected and exposed as before. Considerable reaction follows in a day or two attended by pain, swelling, and some sloughing; the treatment is suspended, and the ulcer covered with zinc ichthyol salve muslin renewed once a day. After the ulcer has healed the treatment, if necessary, can be resumed and repeated until the patient is cured. Recurrence is usual. Dr. Lowe (*Archives of the Röntgen Ray*, Feb. 1903) thinks that lupus affecting a fair skin yields more rapidly to the rays than when it affects a dark skin; the time required is increased in proportion to the amount of pigmentation. As in rodent ulcer, so in lupus, the character of the cicatrix is all that can

be desired—soft, white, and yielding, with no tendency to contract and cause distortion of the face or eyelids, as so frequently happens when operative procedure is undertaken. For Röntgen ray dermatitis, *see* p. 566. Dr. Norman Walker showed a case of lupus with marked ectropion to the Edin. Med. Chir. Society in Nov. 6, 1901; under the influence of the Röntgen rays the exposed mucous membrane changed into what was, to all practical purposes, skin.

Other Skin Diseases. — In the other skin diseases enumerated above encouraging results have been recorded by various observers, but, except in favus, enough statistics have not yet been gathered to determine the proper place of this treatment.

The intractable diseases, favus, mycosis fungoides and xeroderma pigmentosum, appear to be particularly amenable to the Röntgen rays, and Dr. Allan Jamieson exhibited cases of the two latter to the Edin. Med. Chir. Society which he had apparently cured by this treatment.

Rodent Ulcer.—The treatment of rodent ulcer by the Röntgen rays is usually very successful; many such cases have now been recorded. Sometimes the action of the rays may be reinforced by a course of the Finsen light, as in the following case exhibited by the author at the Med. Chir. Society in Jan. 1902:—

Case.—A man, aged fifty-five, recommended by Professor Annandale, was admitted to the electrical department of the Royal Infirmary on 4th December 1901. The patient had suffered for the last eight years from a rodent ulcer on the right side of the nose which had been treated unsuccessfully in many different ways. The ulcer was bean-shaped and about the size of a small mussel shell, covered with brown crusts, which when removed left an irregular depression ⅛ to ¼ inch in depth, with a raw greyish granular surface and shelving edges. Treatment by Röntgen rays was commenced on 4th December 1901, with exposures of five minutes daily. Slow but steady improvement followed until 10th January 1902, when the X-ray treatment was replaced by exposures of from two and a half to five minutes to the condenser spark Finsen light (*see* p. 579) through rock salt three times a week. Three days after the first exposure marked improvement was observed, and after three exposures the ulcer had almost disappeared. After five exposures the patient returned home, the only trace of the disease that remained consisting of some slight scales towards the upper margin of what had been the ulcer (Turner's *Med. Elect.*, p. 373).

This case is important because it gives an indication of what may be a more useful mode of treatment than either the Röntgen rays or Finsen light alone. The same procedure is recommended by Schiff in cases of lupus. It will be found in cases of rodent ulcer that under treatment by the X-rays the surface dries up, the advancing margin disappears, and the ulcer ultimately heals over by granulations. In many cases of rodent ulcer the pressure necessary for the treatment by Finsen light cannot be borne, and the same is true of ulcerating cases of cancer, but the X-rays can always be made use of. Cases have been recently recorded of the successful treatment of this disease by high frequency currents (*see* p. 582). Mr. Mayon has recently contributed to the Röntgen Society some remarks on the treatment of rodent ulcer of the eyelids by X-rays (*Archives of the Röntgen Ray*, January 1903); he regards the action of the X-rays as due to the inflammatory process which they set up. If a section of rodent ulcer after it has just begun to react to X-rays be examined, we find the clumps of epithelial cells which make up the growth are surrounded with enormous numbers of leucocytes. In a section of rodent ulcer which has nearly healed we find a mass of granulation tissue organising, with healthy epithelium growing in from the edges; scattered here and there among the infiltration leucocytes are epithelial cells of the growth in various stages of degeneration. The whole process is purely inflammatory, the inflammation being increased in the neighbourhood of the growth owing to the irritant action of the rodent epithelial cells. Mr. Mayon's reasons for this are—

1. These are the only histological changes found.
2. The sections showed direct invasion of the clumps of rodent cells by leucocytes.
3. The clumps degenerated from without inwards towards their centre; that is to say, the cells in contact with the leucocytes degenerate first, the innermost cells showing no change at that time.

In treating a rodent ulcer, Mr. Mayon removes portions of the growth from time to time to see if there are any active cells of the growth present, and when they have all disappeared to cease the treatment.

Cancer and Sarcoma.—It may be claimed that the X-rays now have a definite place in the treatment of malignant disease, and that is in inoperable external cases, but evidence is accumulating that they may also be found of value in internal cases. The clinical results of the treatment are:—

1. To remove or diminish the pain.
2. To loosen adhesions and to cause the disappearance of the contracting and tightening sensation of scirrhus.
3. To cause an ulcerating and sloughing surface to take on a healthy and healing aspect, and to diminish in size through the growth of epithelium from the edges, and even, as in two cases of the author's, to become completely covered with healthy skin.
4. To dissipate and remove cancerous and sarcomatous tumours.

Twenty-five cases have been under the writer's care, and of these 15 affected the mamma; 2 affected the tongue; 2 affected the scalp; 1 affected the penis and scrotum; 1 affected the cervix uteri; 1 affected the rectum; 1 affected the larynx; 1 affected the glands of the groin; 1 affected the side of the neck.

Nearly all these were for various reasons inoperable cases, the derelicts of surgery, and therefore all the more difficult to treat successfully, and yet all but three of them showed improvement, and some of them remarkable improvement. The breast cases were the most successful, and the tongue the least. The following is an interesting case:—Inoperable ulcerating scirrhus of four years' duration of the left mamma in a woman aged forty-nine, with enlargement of the axillary glands. The ulcer was obliquely placed, and measured 3½ inches by 3 inches; the patient complained of pain, stiffness of the left arm, and insomnia. An exposure of five minutes to the X-rays was given every third day. After five applications the pain and insomnia had gone, and the patient felt better and stronger; the ulcer appeared to be shallower, but was still partly covered with a greenish slough. Rapid improvement continued, and after twenty-two exposures the ulcer was entirely covered with a thin vascular skin. Treatment was now suspended. Three months later the patient was in the same condition, but the skin appeared to be a little thicker.

Another case was very similar, a large ulcerating surface completely skinning over after nineteen applications, and continuing to improve after the cessation of the treatment.

In a case of Dr. Ritchie's of large sarcoma of the side of the neck, recommended to the author for treatment, seventeen exposures were followed by the complete disappearance of the external tumour; the growth, however, continued internally, and dermatitis interfered with the exposures, and the patient died.

The difficulties of satisfactorily introducing the X-rays into the rectum and vagina have led the author to prefer the use of high frequency currents for such cases; in fact, there is a strong tendency now to substitute the latter for all cases of malignant disease, whether internal or external.

Dr. W. B. Coley has published an important paper on the treatment of sarcoma by the Röntgen rays, and discusses the following conclusions amongst others:—

1. That the results in the cases thus far treated prove that the Röntgen rays have a remarkable inhibitory action upon the growth of all forms of malignant disease, and this is especially true of sarcoma.

2. That this action, in many cases of even far advanced and inoperable malignant disease, may result in the total disappearance of the tumours, often without any breaking down of the tissues, the new growth being apparently absorbed.

The Röntgen rays have a very marked influence upon the pain and nearly all types of malignant tumours, causing in many cases entire relief.

Dr. Clarence Skinner (*Journal of Advanced Therapeutics*, New York, October 1902) describes 33 cases, including carcinoma of the uterus (8 cases), carcinoma of the breast (8 cases), and others of epithelioma and osteo-sarcoma. The clinical data are summarised as follows: complete disappearance of the malignant process has apparently been secured in 3 cases; permanent reduction of the tumour in 13; temporary reduction with subsequent increase in 1; permanent arrest of growth in 2. There was no effect positively demonstrated upon the size of the lesions in 14. Complete permanent relief of pain in 14 cases, complete temporary relief in 2, partial relief in 8, no relief whatever in 4, and in 5 there had been no pain.

The author is inclined to recommend that the exposures should not follow one another too rapidly; his best results have been obtained with two or even with one seven-minute exposure a week; further, the treatment should be interrupted from time to time, for the patient will continue to improve, the therapeutic effect of the ray being like its effect in producing dermatitis cumulative. The very greatest care should be taken in malignant cases to avoid setting up a dermatitis.

Phthisis; Tubercular Glands; Sinus.—The Röntgen rays have been tried in cases of phthisis pulmonalis and laryngea by a few observers. Ten cases have been under the author's treatment; 4 improved, the others were unaffected. Dr. P. R. Turnure (*New York Medical Record*, 7th February 1903) treated 2 cases of tuberculosis of the larynx, 1 for four and the other for six weeks without improvement.

The results are more favourable in tubercular glands and joints. A large number of the former have been under treatment by the Röntgen rays in the electrical department of the Edinburgh Royal Infirmary, and in most cases with beneficial results; it is, in fact, rare to meet with a case which does not improve. Good may also be anticipated in the case of tubercular joints. The following case of Professor Annandale's will illustrate this:—Case: patient aged twenty-seven, suffering from tubercular disease of the right elbow. The elbow became swollen and very painful after a fall. As it did not improve under rest Professor Annandale excised it. The wound has never healed properly, and there is still pain and considerable swelling. After a month's exposure to X-rays three times a week of five minutes' duration there was decided improvement, the movements at the elbow were fairly good, there

was practically no pain, and little discharge. Professor Annandale expressed himself as greatly pleased with the result.

Sometimes chronic ulcers and sinus improve under X-ray exposures; the vascular changes, leucocytosis, and growth of connective tissue produced by the rays are likely to be beneficial. Sjogren and Sederholm (*Archives of the Röntgen Ray*, August 1901) report 4 cases in which the cause of ulceration was uncertain; the only point in common between them was that they had resisted other methods. Two of these were cured and 1 greatly benefited. Dr. Berry Hart (*British Medical Journal*, 31st May 1902) describes 2 cases treated in the electrical department of the Edinburgh Royal Infirmary, in which obstinate callous sinuses in the abdominal wall healed up—1 after a single application of the X-rays for photographic purposes with a probe in the sinus, the other after three exposures (Fig. 19). The author has observed similar good results in some cases, but in others, particularly in fistulæ, no beneficial effects. High frequency currents are likely to be of more service in such cases.

Trachoma.—Mr. M. S. Mayon and Dr. David Walsh have reported cases of trachoma cured by exposure to the Röntgen rays. Mr. Mayon (*Archives of the Röntgen Ray*, January 1903) states that the final result to the lid is most satisfactory. Instead of the white, puckered conjunctiva gained by other methods, a supple, non-contracted, non-scarred conjunctiva, with no obliteration of the fornices, unless they are already gone before treatment, is produced, similar to the soft, supple scar in the skin following by this form of treatment in rodent ulcers, as compared with the dense cicatrix of excision.

As regards the effect on the pannus, it often clears with great rapidity, especially if recent, and it is the common thing for patients to say that they see more clearly from almost the first exposure. But even dense corneal opacity will often clear considerably, and in one case of extensive destruction a cicatrisation of the cornea, in which at the commencement of treatment the patient could only see shadows, in two months she could count fingers three feet away.

The chief advantages of this treatment are:—

1. It leaves considerably less deformity.
2. It is a painless treatment.
3. The pannus clears more thoroughly.

Diseases of the Spinal Cord.—Dr. Byrom Bramwell has had some nervous cases, mostly suffering from locomotor ataxy, treated with the Röntgen rays to the lumbar and lower dorsal region, in the electrical department of the Edinburgh Royal Infirmary. He exhibited one patient to the Edin. Med. Chir. Society; this patient had derived marked benefit from the treatment and had put on weight. The writer tried the rays in an old case o myelitis without effect.

Neuralgia.—The Röntgen rays may be tried for this: they will nearly always relieve the pain of cancer, and the author has seen cases of sciatica and even of toothache relieved. This relief of pain was strikingly brought home to me in a case of severe and intractable toothache sent to me for diagnostic purposes by Mr. Watson, L.D.S. Several photographs of the suspected teeth (exposures from two to ten seconds) were taken, but when the patient returned in a few days' time he told me that his persistent pain had disappeared shortly after the exposures. Stembo (*Archives of the Rontgen Ray*, January 1901) narrates cases of neuralgia which were relieved as a rule after the third application.

The Finsen Light and High Frequency Currents

By consulting the table on page 565 it will be seen that the Finsen light consists chiefly of the blue, indigo, violet, and ultra violet rays of the spectrum. Such rays are given off by the sun and by certain artificial lights, such as the arc light and that of a condenser spark, particularly in the two latter cases if iron poles or electrodes be used. The original large arc light used by Prof. Finsen has been superseded in this country by small arc lights placed close to the patient and separated from the diseased area by two diaphragms of quartz and a layer of running water. This arrangement is necessary to cool the light by absorbing the radiant heat rays. A type of such a lamp is the Lortet-Genoud or its London hospital modification (Fig. 20). To render the part to be treated anæmic the patient presses it against the outer quartz diaphragm, and the part is thus brought within 2 or 3 inches of the light. To supply the lamp with electricity it must be attached to the electric light mains with a sufficient resistance in series to bring the current down to about seven to ten ampères. A number of these lamps have now been in operation in the United Kingdom for some time, and it is found that an exposure of some ten to twenty minutes is sufficient to produce the necessary reaction. With the original Finsen large arc light the exposures lasted an hour or more, and the area affected was one-quarter the size.

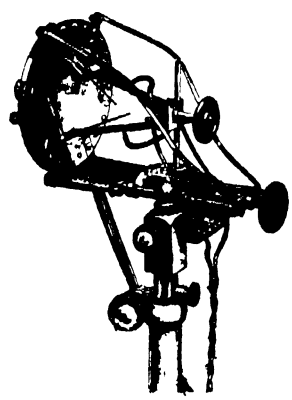


Fig. 20.—London hospital lamp. Marshall and Woods.

More recent arc lights use iron poles (Dr. Bang's lamp, Fig. 21), or carbon poles with cores of iron dust (Dr. Strubel's lamp, Fig. 22). The

advantage of using iron is that its spectrum is richer in ultra violet rays. Dr Strubel's lamp possesses the further advantage of requiring less resistance in the circuit and of giving double the light; it consists of two small arc lights placed compactly together; as each light requires about 45 volts' pressure, the two will require 90 volts, so that they can be run on a 100-volt circuit with but little added resistance.

FIG. 21.—Dr. Bang's lamp. Dermo pattern. Sanitas Co.

The *British Medical Jour.*, 4th Jan. 1902, quoting a German authority, states "that Dr. Bang's lamp need not be larger than an ordinary tablespoon, compressing and supporting apparatus included, and that its cost would be about £3. While the light from an ordinary arc lamp can kill cultures of the staphylococcus pyogenes aureus in 4½ minutes, the light of Dr. Bang's lamp, under similar conditions, can kill them in less than four seconds, thus giving a bactericidal power sixty times stronger than that of the ordinary arc lamp. With his apparatus in both healthy and lupoid skins a marked light erythema of many days' duration may be produced with five ampères of current over an area 4 inches square in five or even in three minutes, and as strong as that hitherto obtainable with sixty ampères of current (original Finsen light) in seventy-five minutes."

The disadvantage of all the arc light sources

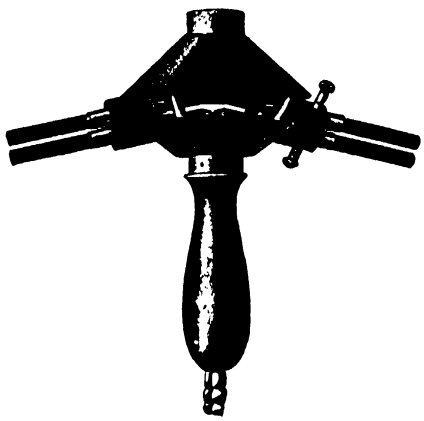


FIG. 22.—Dr. Strubel's lamp. Schall.

of the Finsen rays is that their sources radiate not only the Finsen rays, but the whole of the rest of the spectrum as well, including the infra red or radiant heat rays, and for this reason the light has to be cooled by running water, and the water and quartz windows absorb an appreciable quantity of the active Finsen rays, and their interposition also prevents the diseased area from being brought quite close to the light. What is required is a cold source of light which radiates only the blue violet and ultra violet rays, and the light of a condenser spark more nearly answers this purpose. This spark is obtained by joining the outer and inner coatings of a condenser to the secondary terminals of a large induction coil, and causing the condenser to discharge between two iron electrodes. This lamp—introduced by Prof. Görl (Fig. 23)—can be obtained from Schall, 55 Wigmore Street, or from Leslie Miller, 93 Hatton Garden, E.C. The lamp is contained in a metal case, and its open end is closed with a diaphragm of rock salt, ice, or quartz which is pressed against the affected area. The author's experiments on the transparency and opacity of bodies to ultra violet light showed that pure polished rock salt was more transparent than either quartz or ice (Turner's *Medical Electricity*, 3rd edition, p. 366). Glass, mica, ebonite, and gelatine are opaque; the opacity of glass is to be particularly noted; ultra violet rays may be present in abundance inside a Röntgen ray tube, but they will all be stopped by the glass. Rock salt is twice as transparent as ice, and from twenty to thirty times as transparent as quartz. The rock salt should be cut into the form of a convex lens and should be polished. It must be kept quite dry; a film of moisture upon it destroys both its transparency and its polish. It may be cleaned with absolute alcohol and polished with chamois leather.

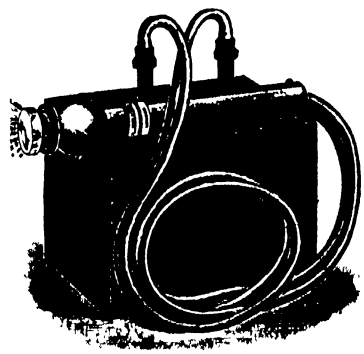


FIG. 23.—Condenser spark lamp. Turner's *Med. Electricity*.

An objection to the use of this lamp is the crackling noise of the spark discharges; it is also said that its action is more superficial than that of the arc light; despite of that we have reduced the exposure with this lamp to from two and a half to five minutes in the electrical department of the Royal Infirmary, Edinburgh.

The Finsen light has been used chiefly in the treatment of lupus (*see* p. 575); that it is a specific for superficial cases of lupus vulgaris there can be no longer any doubt, hundreds of such cases are on record. The disadvantage is the great length of time required in the treatment, and the smallness of the area exposed at one time. In consequence of these drawbacks there is a strong tendency to treat cases of lupus with the Röntgen rays instead of with the Finsen light (*see* p. 575), the area treated is much larger, and the time required and the expense less. To the employment of the Röntgen rays there is, however, attached the risk of a dermatitis, while such a sequela is not to be feared with the use of the light.

Care must be taken in treating cases with the

Finsen light to see that the pressure upon the diseased area is sufficient to express the blood; the use of ice as a compressing medium has the advantage of aiding the expression of blood by its chilling effect. The length of the exposure will be governed by the particular lamp selected, but it should be sufficient to produce a reaction of slight erythema with some smarting and tingling.

The cases of lupus for which the light is particularly suitable are those in which the patches are small and well defined, and in positions such as over bony prominences where the necessary pressure can be exerted. As a result of repeated exposures the granulomatous tissue of lupus has its vitality destroyed and undergoes degeneration; any bacilli present are killed. The writer has treated cases in which there was a lupus patch on each side of the face, the one side with the Röntgen rays, and the other side with the Finsen light, and he has always found that the improvement was more rapid and general on the side treated by the rays. The following is a case in which the light treatment had a very rapid effect. Case: Lupus of four years' duration affecting the right ala of the nose, and consisting of a small, punched-out patch about a quarter of an inch in depth; eleven exposures of two and a half minutes each to a large condenser spark lamp with a rock-salt compresser sufficed to bring about a cure. To assist in the production of a reaction applications of pyrogallol, acid nitrate of mercury, or iodine may be tried.

In the case of lupus erythematosus the Finsen light appears to be often of more service than the Röntgen rays. In a case of well-marked lupus erythematosus of two years' duration which failed to yield to X-ray treatment, the writer has observed distinct improvement follow the application of the Finsen light.

When the necessary pressure can be borne the light can also be used for rodent ulcer, and Dr. Finsen of Copenhagen had many successes with this treatment. The light is also of service in the treatment of superficial cancerous nodules. In a case of Mr. Cotterill's of multiple scirrhus (exhibited at the Edinburgh Medico-Chirurgical Society, 7th May 1902) ultra violet light was employed by the author with excellent effect to the cancerous nodules, which rapidly disappeared under its use.

High Frequency Currents

Though these currents have been known for about a dozen years under the name of Tesla or d'Arsonval, they have only recently become prominent in practical medicine. Introduced first by Tesla and Elihu Thomson, they owe to d'Arsonval their physiological and therapeutical importance. These currents differ principally from those developed by an alternating current dynamo or an induction coil in the fact that the electromotive force or pressure is exceedingly high, and the alterations or reversals (frequency) inconceivably rapid. A good induction coil may develop an electromotive force or difference of potential of 50,000 or more volts, and if provided with a Wehnelt break the number of its interruptions may reach 1000 per second, but the electromotive force of the high frequency currents may reach a million or more volts, and the frequency may attain many millions per second. This frequency is occasioned by the oscillating character of the discharge of the Leyden jars; the jar is first charged by the induction coil or static machine, and when the potential has been sufficiently raised it discharges across the spark gap, and this discharge is of an oscillatory nature. Rushes or surgings of current occur first in one direction and then in another with a frequency of alternation quite unapproachable by any mechanical means.

Mode of Production of the High Frequency Currents

An induction coil or a static machine may be used from charging the two Leyden jars; the former is the best and most reliable arrangement, the output from the latter, unless very large and very rapidly rotated (*see* Gaiffe's machine, p. 566), being insufficient to keep the jars fully charged. The two inner coatings of the Leyden jars (Fig. 24) are connected to the secondary terminals of the induction coil, and the spark discharge may be allowed to occur either between two especially supplied balls enclosed in a box to deaden the noise and shade the light, or between two balls attached to the ordinary spark electrodes of the coil.

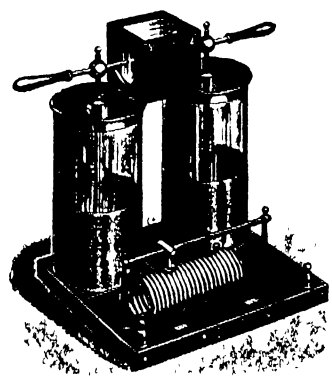


Fig. 24.—High frequency apparatus. Schall.

The outer coatings of the jars are attached one to each end of a solenoid of thick copper wire.

This is all the apparatus that is absolutely necessary, but the effects will be very much improved if the resonator of Oudin be added. Dr. Oudin's resonator (Fig. 25) consists of some two hundred feet of copper wire wound round a large framework of insulating material. When a proper length of the wire of the resonator is placed in parallel with the solenoid, the former, to use an analogy, acts the part of the sounding board of a musical instrument, and vibrates in unison with the solenoid so as to greatly reinforce the electromotive force. When the two are in proper accord, the upper turns and the

terminal of the resonator will give off a very powerful violet-coloured effluve (best seen in the dark). The resonator should be provided with a sliding contact to which the wire from one end of the solenoid can be attached, and this contact should be moved up and down until the best effects are produced; to obtain a more exact adjustment the resonator should be capable of rotation. In some instruments the solenoid is suppressed altogether, and the lower turns of the resonator take its place. Before the invention of the resonator the usual method adopted for producing high frequency currents was to allow the Leyden jars to discharge through the primary of an oil induction coil (*see* Turner's *Medical Electricity*, p. 144); d'Arsonval has quite recently reintroduced this method in his bipolar induction coil, but which uses air instead of oil as the circulating medium. This is a bulky instrument, consisting of a cylinder on which the secondary wire is wound, and round it, but separated from it by a layer of air, are a few turns of a thick primary wire supported by insulating legs; the latter is arranged to slide along and parallel to the axis of the secondary wire. This apparatus is supplied by Gaiffe. The one pole of the secondary wire is attached to the electrode for the patient, the other to earth or also to the patient. Dr. Macintyre has introduced a special device for reversing the current as it comes from the condensers, so as to make the arrangement more suitable for use with a static machine.

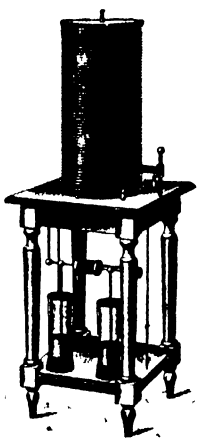


FIG. 25.—High frequency apparatus. Schall.

Dr. Oudin (*Annales d'électrobiologie*, July-August 1900) makes the following remarks as regards d'Arsonval's bipolar coil:—"When we wish to administer general high frequency treatment to a patient, I do not think that we must attach much importance to this *bipolar* character, which must perforce exist when the efflux from one end of a single resonator is directed upon an individual. For at every instant when a positive influx passes to such a capacity as the body of a patient, the capacity has induced upon it a negative potential, and *vice versa*. But I think that in making local applications we get a zone of greater density when a bipolar coil or resonator is used."

There are three chief methods of applying the high frequency currents for purposes of treatment:—

1. "Autoconduction."
2. Condensation.
3. Direct local applications, either by *a*, the effluve; or *b*, intercalation of the patient in the circuit by the direct contact of suitable electrodes.

1. In "autoconduction" the solenoid is made (Fig. 26) large enough to enclose the patient without touching him, and the patient is placed inside; he may either stand or recline according to the direction of the long axis of the solenoid, which may be vertical or horizontal, and arranged round a couch. The patient forms the secondary of a gigantic induction coil and will be the seat of induced currents.

2. In "condensation" the patient forms one of the plates of a condenser or Leyden jar; for this purpose a couch is provided, the back and lower part of which is lined with metal, and carrying a terminal by which it may be attached to the resonator or to one end of the solenoid. Over the metal is placed an insulating material such as felt, and over that the ordinary cushions. The patient reclining on the couch is connected to earth or to the other end of the solenoid by grasping an electrode attached to a chain; the other end of the chain may pass to a gas or water pipe, or to the solenoid, or to both.

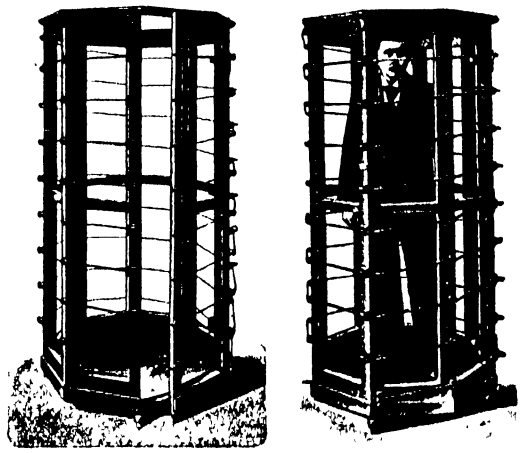


FIG. 26.—Autoconduction. Schall.

The felt and cushions form the dielectric, and the patient the outer or earthed coating of the condenser. At every electrical oscillation the patient will become charged and discharged, and the charges will vary in sign.

3. The "Effluve." A pointed conductor attached to the resonator is approached to the patient; the conductor must be held in an insulating handle. As the point approaches a violet "effluve," starting from both the point and the patient, will complete the circuit. The form of this "effluve" resembles that of the lines of force between two opposite magnetic poles. If the point be brought too near sparks will pass, and these are unpleasant to the patient. A variety of this method (Fig. 27) is to enclose the pointed conductor in an insulator such as ebonite or glass (condensing electodes); such an electrode may be brought into actual contact with the skin, and used either in a labile or stabile method; it will cause slight burning and tingling sensations. Or a vacuum tube or a tube containing acidu-

lated water may be attached to the resonator and used as an electrode; by these electrodes the strength of the applications is diminished.

Lastly, the patient may be directly intercalated in the circuit by the firm application of two large-surfaced electrodes attached respectively either to the two ends of the solenoid (in parallel), or one to the solenoid and one to the resonator.

With the exception of the effluve, none of these methods cause the patient any appreciable sensation. The former occasions slight smarting, tingling, and burning, and it is followed by a vivid hyperæmia, which may take from some hours to two or three days to disappear. The strength of the applications can be graduated in various ways, thus:—

a. The primary current supplying the induction coil can be altered in strength or voltage.

b. The sparking distance can be increased or diminished; the greater the length of the spark the more powerful are the oscillations.

c. The resonator may be more or less accurately tuned to the solenoid, etc.

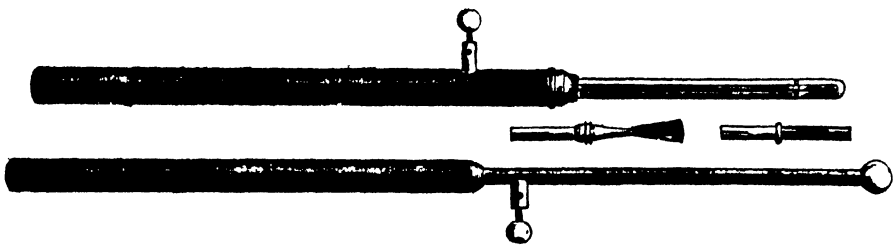


FIG. 27.—High frequency electrodes. Schall.

The physiological effects of the high frequency currents have been investigated by d'Arsonval, Oudin, Bruchard, and others.

Except in the case of the "effluve" and of spark discharges, no sensation is experienced and no stimulation of muscle occurs, even though a current of three ampères be traversing the body (*Les courants de haute fréquence*, Denoyés, p. 64). M. Cornu allowed electrical energy at the rate of nearly one horse-power to pass through his body without experiencing any sensation whatever; six incandescent electric lamps (125 volts 0·8 ampère) were illuminated by the current. The most probable explanation of this immunity is that of d'Arsonval, who (Turner's *Medical Electricity*, p. 187) suggests that the tissues cannot respond to such rapid alternations. The retina, for instance, only responds to about one octave of ethereal vibrations—those that lie between 497 (red) and of 640 (violet) billions per second; all others, the dark heat rays and the ultra violet actinic rays, are without effect upon it. So too with the auditory nerve, which has a range from seven to eight octaves. D'Arsonval, using alternating currents for stimulating muscles by their motor nerves, found that if the alternations exceeded some 5000 a second, the effect produced steadily diminished as the frequency rose.

It has, however, been established by various authorities—d'Arsonval, Denoyés, etc.—that certain modifications in nutrition are brought about. Denoyés (*Courants de haute fréquence*, p. 103) states "that the influence in nutrition is shown, as regards the circulatory system, by vascular dilatation and lowering of the arterial pressure, afterwards followed by a continued elevation; as regards the respiratory system by a greater elimination of carbon dioxide and a more active absorption of oxygen by the blood; as regards the urinary secretion by a notable increase in the elimination of waste products; and, lastly, as concerns the heat-regulating mechanism by an increased production of heat." In addition to the vascular dilatation and hyperæmia a degree of anæsthesia is produced by local applications.

The high frequency currents can affect certain toxines; thus d'Arsonval found that the diphtheria toxine suffered an attenuation, and Bonome rendered the streptococcus toxine ten times less virulent. The effect of the current is to convert the toxins into toxoids. Since toxoids are not only capable of producing immunity, but are also very much less poisonous, there may be hope for the clinical treatment of diphtheria and tetanus by the conversion of toxin into toxoid within the living body (A. S. Grunbaum, "Goulstonian Lectures," *B.M.J.*, March 21, 1903).

Therapeutic Applications

The currents have already been applied over a wide field of disease, and a considerable time must elapse before their proper rôle in treatment can be appreciated. The following are some of the diseases for which they have been used:—

Gout, rheumatism, diabetes, obesity, tuberculosis.

Affections of the stomach, dilatation, hæmorrhoids, and fissure.

Progressive muscular atrophy, neuritis, spinal diseases.

Trachoma, tinnitus.

Malignant disease.

Skin diseases.

It is impossible in the space of this article to do more than to refer to one or two of the diseases in which there is the greatest consensus of opinion as to the beneficial effect of the treatment; for details as to the other diseases the following works and journals may be consulted —*Courants de haute fréquence*, Denoyés; *High Frequency Treatment*, Chisholm Williams; various papers in the *Archives de physiologie*, by d'Arsonval; *Compte rendu de l'Académie des*

Sciences (Dec. 30, 1901), by Bordier; papers by Apostoli and Berlioz, and Laquerrière in the *Archives d'électricité Médicale* and the *Annales d'électrobiologie*, etc., etc.

A good deal both of experimental and of clinical work has been done in "tuberculosis." Denoyés inoculated a number of guinea-pigs with tubercle and studied the action of the various modes of employment of these currents upon them; other guinea-pigs inoculated at the same time served as controls. The net result of these experiments, which are narrated with great care and elaboration in Denoyés' book, serve to show that the high frequency currents, particularly under the form of a moderate effluve, have a beneficial effect upon experimental tuberculosis. Rivière states (*B.M.J.*, Jan. 11, 1902): "High frequency currents exercise a certain curative action upon tuberculosis. The microbe cannot resist the repeated application of these currents; its reproductive power and the virulence of its toxins become attenuated, a fact which had already been proved by the laboratory experiments of d'Arsonval. It appears that at the same time the human organism under the influence of these currents gets stronger, its enemy, the microbe, grows weaker. The acceleration that these currents bring to the circulation of the blood and lymph favours osmotic action. This allows the organism to eliminate the refuse and the toxins that invade and infect it. It is also probable that under the influence of this form of electrisation the beneficial action of the phagocytes increases in activity. It thus appears evident that in these conditions the attacked organism recovers its strength, and, aided by the phagocytes, finally prevails against the bacillus. It is therefore sufficient to place the patient in the best conditions of food and hygiene to avoid the return of the disease."

Doumer, Oudin, Rivière, Denoyés, and others have recorded many cases of chronic pulmonary consumption which derived great benefit from the application of the effluve to the affected apices. Chisholm Williams records 43 cases, of which 42 put on weight, and lost all symptoms except in a few where a slight cough and a few bacilli may be found occasionally.

Where there is any grave affection of the heart the currents should be used with caution. Good results have been obtained in "chronic dyspepsia" and in "dilatation of the stomach," but the most striking results have been achieved in "hæmorrhoids and in fissure." Dr. Walker mentions an acute case which was apparently cured by two applications; another case of long standing with bunches of piles protruding, bleeding frequently and painful anal fissure has, after daily applications for four weeks, shown almost complete recovery. Doumer records permanent cure in nine cases, each one of which was associated with a fissure. Five or six local applications were sufficient to cure the bulk of his series. The author, using a condenser electrode, can quite confirm these results; one application is usually followed by marked relief. There can be no doubt about the great value of this treatment in hæmorrhoids. The application, lasting from five to ten minutes, should be made by a rectal condenser electrode brought into actual contact with the sphincter and gradually passed into the rectum; owing to the analgesic action of the current but little discomfort will be felt.

Denoyés publishes a case of "progressive muscular atrophy" in which he obtained a remarkable result (*Courants de haute fréquence*, p. 289); similar cases are also on record; secondary muscular atrophies are also benefited.

Condensations or local applications may be tried in "tabes dorsalis." A case treated by the author in this way undoubtedly benefited. In "trachoma" Dr. Walsh found "that twenty-three painless applications of the effluve caused the disappearance of a disease that under ordinary treatment would have lingered on for two or three years. The only altered conditions of environment was the application of the effluve, and the patient's other eye remains as bad as ever, so that the brush discharge may reasonably be regarded as the curative agency" (*Archives of the Röntgen Ray*, Jan. 1903).

The effluve has been used with varying degrees of success in eczema, psoriasis, pruritus, rodent ulcer, and lupus.

Four cases of "diabetes" have been recorded by Vietti, and two by d'Arsonval, in which the sugar either disappeared or was much reduced; others have found that though the general condition was benefited, no diminution in the glycosuria followed. Auto-conduction is the method usually applied in diabetes.

More recently these currents have been found of distinct value in certain cases of "malignant disease," such as cancer of the bladder, uterus, rectum, throat, and nasal cavities. It is possible that they may partly supplant the Röntgen rays in the treatment of malignant disease, for they can be used not only for the cavities above mentioned, in which much success with the Röntgen rays can scarcely be expected owing to the tissue which has to be penetrated, but also for the external cases; and they have this great advantage over the rays, namely, that they do not produce a dermatitis. In properly selected cases the author has seen the disappearance of pain and of pressure symptoms, the complete healing of ulcers, the diminution or disappearance of the growth, the restoration of sleep and appetite, and the gaining in weight, but in no case yet has he observed a complete cure. A very instructive case was the following:—

The patient had been previously operated on, and the right mamma and pectoralis muscle

removed for a scirrhus of a very malignant type; owing to a rapid recurrence both in the scar and to the appearance of two lumps in the left mamma, accompanied by many skin nodules and the enlargement of the superficial and deep lymphatics, the electrical treatment was tried. After consultation with a surgeon it was decided to treat the right side with the Röntgen rays and the left side with the effluve on alternate days. The rays had to be given up after twelve five-minute applications owing to a commencing dermatitis; the high frequency treatment on the left side could be continued. On a re-examination in consultation with the surgeon, after a total of twenty-five applications—twelve of the rays and thirteen of the effluve—both sides showed much improvement, the superficial nodules had disappeared, the lymphatics were smaller, the right arm less swollen and more movable, the lumps in the left mamma were more diffuse and smaller, and it appeared to us that both methods of treatment had been of equal efficiency. All treatment had, however, to be suspended owing to internal secondary growths causing œdema of the lower extremities. In a case of Dr. Macmillan's of recurrent epithelioma after removal of the cervix, the Röntgen rays, which had been used with benefit in the electrical department of the Edinburgh Royal Infirmary, had to be given up owing to a dermatitis; this case being now treated by a condenser vaginal electrode with the effluve, showed the same temporary improvement which had attended the use of the rays, each application being followed by a lessening in the amount and odour of the discharge, and by a feeling of *bien-être*. Two applications of seven minutes each a week seemed to be sufficient to keep the disease in check.

Where possible the author prefers to treat such cases by the effluve locally on one day and by condensation on the following day, so as to obtain both a local destructive effect on the growth and a general nutritional improvement. In the *West London Medical Journal* for July 1902, Dr. James Allan records four cases treated by the effluve. The following are brief notes of these cases:—

Case I.—Miss H., aged 48, operated on for sarcoma of the antrum twice. On a further recurrence electric treatment was begun. The growth was easily seen in the nasal passages; the patient looked very ill and complained of much pain. After the second application by means of a small condenser electrode improvement began, the pain subsided, the patient slept well, and after fifteen sittings the visible growth had disappeared. The pathological report was a spindle-celled sarcoma.

Cases II and III.—Two cases of recurrent scirrhus of the breast also yielded to the treatment.

Case IV.—A fourth case was one of epithelioma of the cervix which had been given up as hopeless by two doctors. The cervix was completely involved, the patient weighed 6 st. 5 lb., and looked and felt very ill. The case being a desperate one, daily applications of from forty to sixty minutes were given by means of a condenser electrode passed up to the cervix. After about six weeks' treatment a very severe reaction set in accompanied by rigors. Three weeks later treatment was resumed with signs of improvement. The local condition did not alter much, but the pain and discharge decreased, sleep and appetite returned; the growth seems to be entirely arrested; present weight 7 st. 10 lb.

Dr. Allan adds: "We must not hastily jump to conclusions about this new treatment, but we seem to be on the threshold of a very important therapeutical discovery which will bring relief and health to those suffering from a malignant disease which up till now has baffled the medical world."

Xylene.—A carbide of hydrogen (dimethylbenzene, $C_{16}H_{10}$), which resembles benzol in its properties. *See* Benzol.

Xylidine.—A methylated homologue of aniline ($C_8H_9.NH_2$), obtained from dimethylaniline; xylidine-red is a staining reagent resembling rosaniline.

Xylo-.—In compound words xylo- (Gr. ξυλον, wood) means belonging to wood or woody, *e.g.* *xylophagous* (wood-eating).

Xyloidin.—A powder produced by the action of nitric acid on starch and woody fibre ($C_6H_9(NO_2)O_5$); it is inflammable.

Xylol. *See* Xylene; xylol balsam is a solution of Canada balsam in xylol, used as a mounting agent in microscopy.

Xylonite. *See* Celluloid.

Xylose.—A sugar ($C_5H_{10}O_5$) isomeric with arabinose; it is prepared from wood-gum, hence the name (*xylose*).

Xylotherapy.—The treatment of disease by various woods, such as cinchona bark, pine-wood, etc.

Y-shaped.—Having the shape of a capital Y or forked stick; *e.g.* the Y-shaped cartilage of the acetabulum in childhood, the Y-shaped ligament (ilio-femoral ligament), and the Y-shaped double monster (hypsiloid fused twins).

Yawning. *See also* Hysteria (*Disorders of Respiratory Organs*); Physiology Respiration (*Special Respiratory Movements*); Respiration (*Hyperpnœa*).—Yawning is a physiological expression of fatigue. It consists of the following phenomena:—

1. A deep inspiration, with elevations of the vomer and palate.

2. A forcible spasmodic depression of the lower jaw.

3. A flow of tears.

4. A clicking sound in both ears.

5. A tendency to stretch the limbs, especially the arms.

6. An expiration, often accompanied by a sound, the character of which has probably given the name to the act (Poore).

We know little or nothing of the cause of yawning. It is a passing phenomenon, which is removed by rest.

Yaws.

Definition	585
Clinical Features	585
Etiology	586
Diagnosis	587
Treatment	588

See also Skin, Bacteriology of (*Yaws*); Skin Diseases of the Tropics (*Constitutional Infective Diseases with Marked Skin Lesions*).

Synonyms.—*Frambœsia*, *Pian* (West Indies); *Buba* (Brazil); *Coko* (Fiji); *Tonga* (New Caledonia); one of the varieties of *Paranghi* (Ceylon); *Bouton d'Amboine* (Moluccas); *Patek* (Sumatra); *Dubea*, *Ajortor*, or *Tongara* (Gold Coast); *Ogodo* (Sierra Leone); *Effier* (Benin); *Soombah*, *Gattoo*, *Framosi*, *Tetia*, *Momba* (various West African names); *Kwena* (Burmah); *Lupani* (Samoa); *Purru* (Malay Peninsula).

Definition. — A specific constitutional and communicable disease, occurring almost entirely in the tropics, and chiefly manifesting itself by an eruption of reddish tubercles on the skin.

History and Distribution. — Although the disease probably existed much earlier than 1648, the first definite account of it was then given by Pison. It is generally supposed that West Africa was the original home of yaws, and that by means of the slave-trade it spread to the West Indies and South America, but this would not altogether explain its occurrence in Oceania. However this may be, its headquarters at the present day are unquestionably tropical Africa, and more particularly the West Coast, the West Indies, Samoa, Fiji, New Caledonia, and other Pacific islands. It is also common in Ceylon, Java, Sumatra, the Moluccas, and in Brazil, Guiana, and Venezuela. It occurs, too, in Assam and Borneo, and occasionally in Northern Africa and in India. By the adoption of careful measures of segregation it has been greatly lessened in some West Indian islands (Dominica) in which it was formerly frequent. In Barbados it has practically disappeared.

Clinical Features.—The incubation period lasts from two to ten weeks, and is usually unattended by symptoms. Although, in the inoculation experiments of Charlouis in 1880, the average period was about fourteen days, and in those of Paulet twelve to twenty, there is a general consensus of opinion that in naturally acquired yaws it is longer. The disease is frequently ushered in by a febrile attack lasting from a day to a week or more. This is occasionally severe and attended by marked pains, anorexia, anæmia, and so on; but, on the other hand, is sometimes so slight as to be unnoticed. Deep-seated rheumatic pains are, however, usually noted, and these, together with the fever, may recur throughout the illness, preceding the successive crops of yaws. Otherwise the general health is unaffected, unless from such complications as severe ulceration a cachexia is induced. Following these symptoms a papule occasionally appears at the site of inoculation, or if the latter be an already existing sore this becomes unhealthy, or a small ulcer forms. When the primary papule occurs it does not differ from the ordinary eruption, and is not comparable to the initial hard chancre of syphilis.

The eruption gradually develops, and may present itself in the form of squamæ, papules, or tubercles. Owing to its occurrence in crops all these varieties may be present together, but are really stages of the same eruption. During its development the glands, locally or generally, often become enlarged and tender. The first cutaneous symptom is a dryness and harshness of the skin, followed shortly by the development of irregularly scattered desquamating patches, varying in shape.

This squamous stage (pian dartres, yaws cacca) varies in extent, and may be extremely slight, but occasionally involves the entire body. Sometimes it is persistent or appears at a late stage. On the dark skin of the negro well-marked patches are very prominent.

In these patches, after some days, minute papules appear and gradually push their way through the epidermis, which splits over them and curls away from their summit. Later, these (pian gratelle) become capped by a spot of a characteristic cheesy, yellowish discharge, and may then gradually disappear or enlarge into the typical granulomata. Like the squamous form, however, they sometimes persist as papules, and constitute a most obstinate and long-continued type of eruption.

The fully-developed tubercles vary in size from a split-pea to a golf ball, are rounded and hemispherical, or where very large or pressed upon, more flattened, and are covered by a cap of the same yellowish discharge. They may coalesce and form a large irregular patch, and are particularly apt to form clusters about the corners of the mouth and nostrils, where too, from the moisture, they become like condylomata, bare and sodden. During the development of the eruption itching is troublesome, and a peculiar musty smell often accompanies it. The crust, although firmly adherent, can be

removed painlessly, and the surface underneath is insensitive, a somewhat important diagnostic point which is used as a rough test by West Indian negroes, who squeeze the juice of a lime over it. After removal the growth is seen to be grey or red in colour. Some oozing of blood may occur from it, and it at once begins by pouring out fresh cheesy secretion to form a new cap. The yaws vary greatly in number, size, and distribution; they may be localised or general, but are apt to involve the exposed parts, face and limbs rather than the trunk, and the scalp is usually unaffected.

They do not involve the mucous membranes, except around the mouth, nose, and anus; and this, according to Nicholls, occurs in about 2·8 per cent. Near a nail they may cause a troublesome onychia. Several surrounding an area of healthy skin constitute "ringworm yaws." When, as not infrequently happens, one yaw grows much more rapidly and to a larger size than the others, it is often particularly persistent and obstinate, and constitutes the so-called mother-yaw or mamma-pian. Situated on the sole of the foot (tubboes), from the pressure of the thickened skin the yaw is forced out in a coral-like formation, undermining the latter, and from tension and the irritation of walking this form is extremely painful. Finally, it undermines the epidermis with pus, ruptures it, and appears as a fungoid, often fœtid, tumour (crab-yaw).

Although ulceration is not a normal part of the history of a yaw, it occurs in a certain proportion, and may spread beyond them, producing extensive sores with resulting deformity from cicatrisation. It is due to the entry of pyogenic organisms, and probably varies with constitutional condition and care in treatment. Nicholls' estimate of 8 per cent seems somewhat low in the writer's experience in Africa.

This stage of eruption may last from weeks to years. Occasionally many recurrences may take place after apparent convalescence (memba yaws), and extend the duration to years. The tumours mature in about a fortnight, remain stationary perhaps for a time, then the crust shrinks, darkens, dries, and finally falls off, leaving a pigmented spot.

Any acute intercurrent disease may result in the disappearance of the eruption, although this is, as a rule, only temporary. It has been seen after remittent fever, dysentery, and so on. One attack usually confers protection, but true second attacks have been recorded.

Complications and Sequelæ.—In addition to the not infrequent ulceration attacking the growth itself, a form of serpiginous ulceration of the limbs has also been described. Powell has recently reported swelling of the phalanges as occurring in a mother and child during the third year of illness. Both conditions are decidedly rare. In Fiji, where syphilis is unknown, lupoid ulceration of the soft palate or nose and gummatous nodes or thickenings have also been reported by Daniels, Corney, Finucane, and others; and very similar symptoms were recorded by Rat in the West Indies. This, however, is not the general experience, and there seem to be some differences between yaws in Fiji and that usually seen in Africa and elsewhere.

Etiology.—That yaws is communicable may be regarded as proved by numerous accidental inoculations, and by the experiments of Charlouis and Paulet, who successfully inoculated cases artificially. The teaching of experience would also seem to show that it can be propagated by contact, but that some solution of continuity of the skin is necessary. Thus, vaccine vesicles, ulcers, including those of syphilis and leprosy. the bites of leeches, ticks, mosquitoes, or other insects, scratches, or other wounds, may all act as portals to the virus of the disease. Overcrowding, filthy habits, and defective diet, naturally predispose to and aggravate its attacks. Sex has no special influence, but the age of greatest liability is that from five to fifteen, although no age is exempt.

Its essentially tropical distribution and the far greater liability of negroes as compared to white men are also well-known facts. The comparative immunity of Europeans is doubtless largely due to the protective influence of clothing, their greater attention to hygiene and avoidance of yaws patients, and of overcrowding. Second attacks occur and have been produced by inoculation (Charlouis), but are not common, and natives in Fiji expose their children to infection with the idea that their health is subsequently improved. A very similar idea prevails in some native villages on the Niger. A form of yaws occurs in fowls, but animals are not subject to it as they are to verruga.

Infection appears to cling to huts, and after heavy rains outbreaks occasionally occur, due probably to the inhabitants being more confined to their huts on these occasions. The common house-fly and the clothes and mats of infected persons are, with considerable reason, regarded as frequent vehicles of infection.

Relation to Syphilis. — By Hutchison and others yaws is looked upon as possibly the parent form of syphilis, and as merely that disease modified by race, climate, and erratic modes of infection. By the majority of those, on the other hand, who have studied it in its own haunts, it is regarded as a distinct disease, and the writer's experience in Africa leads him to a similar opinion. Finucane's recent description, however, of Fijian yaws would seem to indicate a far closer resemblance to syphilis than is usual elsewhere, although Daniels considers the type in Fiji and in the West Indies to be identical.

The chief argument (and one apparently un-

answerable) against the identity of syphilis and yaws is the occurrence of authentic cases of their co-existence. In some of these yaws patients contracted typical hard sores, and were cured by antisyphilitic treatment without any corresponding effect on the yaws.

The latter differs, too, from syphilis in its localised distribution, its longer incubation, its different mode of infection, and age of incidence.

It also occasionally occurs in outbreaks, and it resists antisyphilitic treatment. In addition, the primary lesion (if present) is not indurated; the effect on general health is usually slight, and febrile attacks may produce improvement; the type of eruption, unlike that in syphilis, is constant; the mucous surfaces, eye, and testicles are practically immune; and unless from entry of pyogenic organisms the lesions are not destructive.

Then the disease is auto-inoculable (Charlouis), no undoubted hereditary or congenital cases have been recorded, and mothers suffering from it have frequently been delivered of healthy children. In some Niger villages, where a large proportion of the children were affected by yaws, the writer failed to find evidence of congenital syphilis. Nor has he found the acquired disease in the tropics to differ in any degree from the ordinary type among Kroomen, in whom it is extremely common. Lastly, the morbid anatomy of yaws shows points of divergence from the syphilitic lesion. The granulation cells are not definitely grouped, and are unaffected by hyaline degeneration; there are no giant-cells, the vessel walls are unthickened, and the epithelium is more markedly altered, while the stroma is less evident (M'Leod).

Histology.—With the exception of some proliferation of the fibrous structures (Nicholls), no marked change is found internally, unless from concomitant disease. In the skin the first change noted is slight dilatation of vessels in the papillary layer, but without alteration in their walls, together with some leucocytic extravasation. A few scattered infiltrations of granulation cells are seen near the vessels, and also slightly around the follicles and coil-glands, although these are themselves unchanged. These granulation cells are oval and polygonal, and have large eccentrically placed nuclei (plasma cells).

Simultaneously the inter-papillary epidermis grows down, and its cells become enlarged and œdematous. The superjacent stratum granulosum is involved, cornification is interfered with, the nuclei of the cells persist, and lamellæ tend to form in the stratum corneum. This vascular dilatation, epithelial proliferation, and œdema and imperfect cornification, produce the scaly lesions. Later the infiltration with granulation cells becomes more general and denser around the vessels, follicles, and glands. Here and there are large connective tissue cells, but no attempt at organisation. An attenuated fibrous stroma supports the cells. The inter-papillary processes of epidermis grow further down, and the greatly lengthened papillæ approach the horny layer, while many leucocytes are seen between the prickle cells. Although cornification is imperfect it proceeds actively, and the horny layer becomes greatly thickened, but composed of still-nucleated cells, and separated into lamellæ divided by layers of leucocytic débris and fibrin. Pigmentation diminishes or disappears at the centre of the lesion, but returns after healing, chiefly in the lower epidermic layers. Most of the melanin granules are contained in the cells of the basal, and to some extent of the granular layer, but are occasionally intercellular, and when they surround a connective tissue simulate a branching pigment cell (M'Leod).

Bacteriology.—The numerous organisms present in the skin increases the difficulty of isolating the one responsible, and although several have been described, in no case yet has the evidence been completed by successful inoculation.

Pierez found a micrococcus on the surface of the skin and in the horny layer. He also obtained it in the blood, and from the latter made cultivations. He found a similar organism in fowl yaws. Nicholls also invariably obtained in the growths a micrococcus from ·5 μ to 1·5 μ in diameter, which occurred either singly or in twos, threes, fours, short chains, or small colonies, and multiplied by transverse fission. It was never present in the blood, but in one case was seen in the fibrous structure of lungs, liver, kidneys, and spleen, a fact which might possibly explain the body pains. Pure cultures to the third generation showed a white, mucoid, oval, or rounded mass, not so well displayed in fluid media. Cultures were also obtained from the dust of yaws hospitals, but inoculations on animals were again negative. Powell also, after careful preparation of the skin, made successful cultivations of a staphylococcus in 24 cases, and in two he found a yeast, the presence of which might, he thought, explain the acid reaction of the discharge. Breda described a bacillus (Frambœsia bacillus) as invariably present in the epidermis and corium, and Boyden and Hamilton obtained cultures in the case of the former of a bacillus not unlike bacillus subtilis. In none of these cases, however, was the evidence completed by successful inoculations.

Diagnosis.—A solitary initial papule might, perhaps, be mistaken for oriental sore, but the progress of the case would soon decide the diagnosis, and syphilis is practically the only disease that can be confused. Geographical considerations and other differences distinguish verruga.

Prognosis.—Nicholls gives the mortality in

the West Indies as 25·8 per 1000, chiefly from intercurrent disease. By cachexia or debility it may predispose to or increase the severity of subsequent disease, and the multiple skin lesions give opportunities for entrance of the germs of phagedæna, tetanus, and so on, but otherwise the general health is rarely much affected.

Prophylaxis.—The comparative immunity of Europeans and the effect of segregation indicate that the most efficient means of prevention consists in avoiding of overcrowding and dirty clothing, care about abrasions, and isolation of the sick. Thorough purification or destruction of infected huts and clothes, the local treatment of the tubercles, and their protection from insect carriers of infection, are similar useful measures. All precautions should be taken in vaccinating in yaws districts, more especially during outbreaks.

Treatment.—General: Anæmia and debility should be counteracted by plenty of nourishing, easily digested diet, and tonics. Chill should be avoided, as tending to repress eruption, and the patient's hut, therefore, should be warm and dry. Diaphoretics and aperients are useful in favouring the eruption, and sulphur and bitartrate of potash are frequently prescribed during the early stage, and are also sometimes useful later. When the eruption is out—mercury, iodides, and sarsaparilla are extensively given, but the former, from its tendency to produce anæmia, should be cautiously prescribed. Arsenic is particularly valuable in badly developed cases, and may be given as Donovan's solution. Powell, with the object of producing a curative febrile condition, has inoculated cases with Haffkine's comma bacillus, and suggests a similar trial with the erysipelas germ. It seems possible that thyroid extract would produce the effects of the febrile state without the obvious disadvantages of this treatment, and it has been tried in Trinidad with favourable results (De Verteuil). At a later stage various tonics, such as iron, quinine, and arsenic, are sometimes indicated.

Local: Various applications have been recommended. Carbolic, chromic, picric, and salicylic acids, nitrate of silver, and sulphate of copper, and many forms of mercurial ointments, may be used. Ulcerating tubercles should be touched with carbolic, cleansed with lotion, and dressed with iodoform or other antiseptic. Crab yaws are best treated by prolonged soaking in hot lotion and removal of the skin covering them, and subsequent application of copper sulphate or calomel ointment. Cleanliness of the skin is most essential, and sea-bathing is strongly recommended.

Yeast.—Saccharomyces, a genus of Fungi. *See* Enteroptosis (*Treatment, Fresh Yeast*), Micro-Organisms (*Blastomycetes*); Teeth (*Bacteriology of the Mouth*).

Yelk.—Yolk (*q.v.*).

Yellow Atrophy of Liver. *See* Jaundice; Liver, Diseases of (*Special Conditions Characterised by Jaundice, Acute Yellow Atrophy*); Pregnancy, Affections and Complications (*Digestive, Jaundice*).

Yellow Body.—Corpus luteum. *See* Generation, Female Organs of (*Ovaries*).

Yellow Chill. *See* Blackwater Fever (*Nomenclature*).

Yellow Colour. *See* Colour Vision (*Theories of Colour Perception*).

Yellow Fever.

Synonyms 588
Definition 588
Virus of Yellow Fever . . . 589
Etiology—
(*a*) *Distinguishing Characters* . . 590
(*b*) *Endemic Centres* . . . 590
(*c*) *Epidemic Extensions* . . . 591
(*d*) *Epidemic Characters* . . . 591
(*e*) *Yellow Fever as a Ship Disease* . 592
(*f*) *Topographical and Altitudinal Relations* 593
(*g*) *Meteorological Relations* . . 593
(*h*) *Conditions affecting Susceptibility—Race, Acclimatisation, Age, Sex, Profession* . . . 593
(*i*) *Predisposing Causes* . . . 594
(*j*) *Sources of the Virus* . . . 594
(*k*) *Modes of Transport and Transmission* 594
Clinical History 595
Morbid Anatomy 596
Diagnosis 597
Mortality and Prognosis . . 597
Prophylaxis 598
Treatment 598

See also Blackwater Fever (*Diagnosis from Yellow Fever*); Dengue (*Diagnosis*); Hæmatemesis (*Causes*); Malaria (*Diagnosis*); Myiasis (*Flies as Carriers of Infection*); Pulse (*Frequency in Yellow Fever*); Purpura (*Symptomatic, Infections*).

Synonyms.—Typhus icterodes (Cullen): *Fr.* La fièvre jaune; *It.* Febbre gialla; *Span.* Fiebre amarilla; *Ger.* Das Gelbfieber.

Definition.—*Clinically*, yellow fever is characterised by a single paroxysm, lasting usually from three to five days, accompanied by frontal and supraorbital headache, injection of the conjunctivæ, flushing and puffiness of the face, severe pain in the loins and lower extremities, gastric distress, retching or vomiting, with a diminished secretion of urine. A fall in the temperature may now end in convalescence, or the remission be followed by yellowness of the

skin, scanty albuminous urine, increased gastric irritability, hæmatemesis (black vomit), and other hæmorrhages. *Pathologically*, yellow fever gives rise to extensive fatty degeneration most marked in the liver and kidneys.

Virus of Yellow Fever.—It is needless to mention the various organisms that have from time to time been announced as the cause of yellow fever. Their claims have been finally disposed of mainly through the laborious researches of Sternberg. The *Bacillus icteroides* discovered by Sanarelli in 1897 appeared to answer more or less fully the requirements which determine pathogenicity, and was accepted by some of the best authorities as the cause of the disease. The American Commission appointed to investigate the nature of yellow fever, consisting of Wasdin and Geddings, as the result of a long series of observations and experiments, reported in 1899, "that the micro-organism discovered by Guiseppe Sanarelli, and named by him *Bacillus icteroides*, is the cause of yellow fever." They base this conclusion on the following grounds: 1. They succeeded in isolating the bacillus from the blood in 13 out of 14 cases of undoubted yellow fever, and in 85·7 per cent of their necropsies. They thus assume that the organism is present in *all* cases of the disease. 2. They failed to find it in the blood of those suffering from, or in the bodies of those that had died of, other maladies. It is not therefore, they urge, a microbe of secondary infection. 3. They instituted numerous experiments on animals with the bacillus in pure culture, and found it to produce a fever running somewhat the same course, accompanied by many of the symptoms, and, in fatal cases, presenting lesions analogous to those of yellow fever.

A more critical examination of the facts has raised doubts as to the pathogenicity of this organism. It has not been proved that the bacillus is present in every case of yellow fever. Some competent observers have failed to detect it in a considerable proportion of cases. Sanarelli himself was able to isolate it in 58 per cent only of his necropsies. Nor has it been satisfactorily established that it is peculiar to yellow fever. Other organisms, again, are capable of producing symptoms and lesions somewhat similar to those caused by the *Bacillus icteroides*. Nothing is really known of the effects of living cultures on man. Those engaged in culture experiments on the bacilli of plague and cholera have accidentally contracted these diseases. Nothing of the kind has happened to those experimenting with this bacillus, even when it has been diffused through the air of the laboratory. The serum of animals rendered immune by injections of the *Bacillus icteroides* has not, so far as it has been tested, proved prophylactic or curative in yellow fever. Finally, the serum of yellow fever fails in many cases to agglutinate the *Bacillus icteroides*. It may thus be safely affirmed that its causal relation to yellow fever has not been established.

Nothing need here be said of the bacillus discovered by Durham and Myers in 1900, as no evidence of its pathogenicity has hitherto been adduced.

The Rôle of the Mosquito in the Transmission of Yellow Fever.—It will be convenient to notice here the evidence bearing on the communicability of yellow fever by infected mosquitoes and by the blood of those suffering from the disease. Finlay of Havana has maintained for nearly twenty years that yellow fever is propagated by the mosquito, and has based a system of prophylaxis on this hypothesis. Mosquitoes fed on a yellow fever patient are applied to a healthy subject, after an interval of two to five days, who by this means is believed to acquire a mild but protective form of the disease. Finlay's statements were never taken seriously by the profession. As to the main point—the communicability of the infection by the *Culex fasciatus* (Fabr.), or more correctly the *Stegomya fasciata*—recent observations confirm his contention, although they go, at the same time, to show that the mosquito is incapable of communicating the infection or of conferring immunity so soon after contamination as his theory postulates. This point, like many others, requires more careful investigation.

The experiments of Reed, Carroll, Agramonte, and others render it practically certain that the infected mosquito is capable of communicating yellow fever. A special sanitary station was fitted up in an isolated, well-drained, and airy spot near to Quemados in Cuba, and every precaution was taken to prevent contamination from without. The results, briefly stated, were, that of ten non-immunes bitten by mosquitoes fed on the blood of yellow fever patients not less than twelve days previously, eight contracted the disease—the incubation period varying from forty-one hours to five days seventeen hours. Other experiments—of which, however, we have not the details—have since been conducted, and entirely confirm the earlier observations. In most instances the disease thus communicated has been of a mild type, but recently three fatal cases of experimental yellow fever have been reported, and it has been found necessary to impose restrictions on these experiments.

The fact that the experiments have been conducted in a yellow fever country detracts somewhat from their demonstrative value. But it is to be remarked that the subjects were placed in quarantine for a period of from nine to twenty-one days before being bitten, and it is specially significant that only those in the station bitten by infected mosquitoes took yellow fever, and that it appeared in them within the usual period of incubation. It would, nevertheless, be de-

sirable that the experiments be repeated in a country free from yellow fever.

That the disease following the bite of the infected mosquitoes was true yellow fever hardly admits of a doubt. As these investigations have been conducted by competent and responsible men, and carried out with reasonable care, it may be accepted as proved that yellow fever is communicable by mosquitoes fed on yellow fever blood. Further, as the mosquito is incapable, according to these observations, of communicating the disease for ten or twelve days after feeding, the insect cannot act as a simple transporter and inoculator of the virus, but as a *host* of the parasite, which must be supposed to undergo either multiplication or development in the culex, in a manner analogous, perhaps, to that which takes place in the case of the parasite of malaria in the anopheles. Hitherto the parasite of yellow fever, whether bacterial or protozoal, has not been discovered either in the mosquito or in the blood of the patient, although the latter has been examined microscopically, times without number, by the most skilled observers furnished with the best appliances. The infective blood does not contain any microbe that can be grown on any ordinary medium.

Carter has shown that when a yellow fever patient is introduced into a place previously free from the disease, an interval of ten to twenty days usually elapses before the occurrence of secondary cases.[1] This fact finds an apparently satisfactory explanation in the circumstance, that about twelve days are necessary for the mosquito to become infectious. An infected house or ship on this hypothesis will mean one in which contaminated mosquitoes are present. We shall have occasion, however, to point out in the sequel that instances are on record in which the interval between the primary and secondary cases has been considerably less than twelve days. It may also be noted in passing, that Kermorgant, in his account of the epidemic at Senegal in 1900, expressly says: "Quand l'épidémie a commencé à Dakar, il n'y avait pas de moustiques." This, perhaps, should not be taken quite literally, for mosquitoes are never altogether absent from these regions.

Admitting, then, that the mosquito transmits yellow fever, many points will have to be worked out before we can hope for much light on the etiology and prophylaxis of the disease. Is the *Stegomya fasciata* the only mosquito capable of communicating yellow fever? Does the insect transmit the infection to its progeny? Can the mosquito become infected in any other way than by feeding on the blood of a yellow fever patient? Can man contract the disease in any other way than by the bite of an infected mosquito? How far does the transmission of the infection by the sole agency of the mosquito harmonise with and explain the limitation of yellow fever to particular zones, to seaport towns, its persistence in ships, and other well-ascertained facts in the epidemiology and etiology of the disease?

Other experiments made at the same station seem to prove that yellow fever is communicable by subcutaneous injections of the blood of the sick, taken from twelve hours to two days after the commencement of the fever. In six out of seven experiments of this kind the result was positive. The amounts injected varied from 2 c.c. to 0·5 c.c. The refractory patient was believed to be naturally immune. Another experiment is reported in which the injection of half a centimètre cube of blood from an experimental case gave rise to yellow fever. Until the converse experiment, however, is made, and mosquitoes fed on the blood of a patient suffering from the fever caused by these injections are proved capable of communicating yellow fever to healthy persons, some doubt must remain whether the fever so induced is caused by the parasite itself or by a toxin present in the blood. It is asserted that yellow fever has been communicated by blood passed through a Berkefeld filter. This requires confirmation. The specific virus is destroyed by subjecting the blood to a temperature of 55° C. The chief significance of these experiments lies in the promise they afford of putting us on the track of a parasite that has hitherto evaded detection. A careful and critical investigation of the facts bearing on the etiology of the disease is now more than ever necessary.

Etiology. — *Distinguishing Characters of Yellow Fever.*—Yellow fever is distinguished from other pestilences: (*a*) By its tendency to invade and cling to ships, and by the extent to which its spread is dependent on maritime commerce; (*b*) By its predilection for towns, especially those situated on the sea-coast and on the borders of navigable rivers, and by the rarity with which it invades rural districts; (*c*) By the tenacity with which it clings to particular houses and localities; (*d*) By its epidemic range being confined to warm climates, and its prevalence in these restricted to the warm season; (*e*) By the extreme variations it exhibits in its spreading power, and in the mortality it causes in different localities and outbreaks; (*f*) By the marked influence of race, nationality, and acclimatisation on susceptibility to infection.

Endemic Centres.—Yellow fever is endemic in the West Indies, whence come the first quite reliable accounts of the disease, reaching no farther back than the year 1647. A second

[1] This is by no means a new observation. Griesenger, half a century ago, observed: "On remarque encore que très souvent il y a un intervalle de huit jours à trois semaines entre un première cas qui se développe et l'invasion consécutive de la maladie chez un autre."

centre is found on the West Coast of Africa, its limits here including Sierra Leone, the Gambia, and Senegal. The first historical outbreak on the West Coast occurred in 1759. Rio de Janeiro and the neighbouring coasts of Brazil form a third centre, where the disease has only become acclimatised since 1849.

In none of these centres, however, has the disease been continually present from the time when its presence was first noted. It has been absent, at least in a recognisable form, for years from every island in the West Indies, and from every locality on the West Coast of Africa and Brazil. Havana, until recently a hotbed of yellow fever, is believed to have been free from it from 1655 to 1761, a period of more than a hundred years, during which the town was reputed for its salubrity. The history of yellow fever points to it being carried by shipping from place to place within a certain zone, and from one zone to another, and persisting for a longer or shorter time in a given place according as conditions are more or less favourable, and then disappearing, until it is again introduced from without. It is thus endemic in zones rather than in places, and is probably maintained by the native infant population who have it in a mild form.

Epidemic Extensions. — Yellow fever was scarcely absent from one or other of the coast or river towns of the Southern States of the Union up to the last quarter of the nineteenth century. New Orleans, Charleston, and Mobile, from their proximity to the West Indies and their frequent intercourse with infected localities, suffered most. Baltimore, Philadelphia, and New York, on the middle Atlantic coast, were also frequently visited by the disease. Yellow fever appeared so far north as Quebec in 1805 and Halifax (Nova Scotia) in 1862. From the West Indies it has from time to time invaded Central America, Venezuela, Guiana, and Bermudas. With the exception of Peru, where the disease has shown a tendency to become acclimatised, the Pacific Coast of America has suffered little.

In Europe, Spain and Portugal have been the chief scenes of its ravages. The close commercial and political relations subsisting between these countries and the West Indies and Brazil, as well as their semi-tropical climate, account for the frequency and severity of the outbreaks of yellow fever from which they have suffered. Indeed, some of the epidemics in Spain have been incomparably more fatal than any recorded in the countries where yellow fever is endemic, falling as they have done on a susceptible population. The disease has always declared itself in one or other of the seaports—Cadiz, Malaga, Barcelona, Gibraltar, or Lisbon, and has generally been confined to these. In 1800-1804, and again in 1819-21, yellow fever penetrated for a considerable distance inland, and maintained itself for some time in a quasi-endemic manner. Leghorn experienced a fatal outbreak in 1804. It has frequently been introduced into the harbours of France and England, but has rarely spread on shore, and never to any extent. Yellow fever was brought into Swansea from Cuba in 1864, and caused sixteen deaths on shore. The East Coast of Africa, the northern parts of Europe, the whole continent of Asia, Australia, and Polynesia have hitherto remained exempt. Its latitudinal limits have been Monte Video in 34° 54′ S., and Swansea, 51° 37′ N.

Epidemic Characters.—Yellow fever never appears in a seaport outside its endemic limits excepting after the arrival of a vessel from an infected port. An epidemy begins by one or two cases, which generally declare themselves near the shore—in the custom-house, or in the quarters frequented by sailors or those employed in discharging vessels. After an interval, not uniform, and often not very accurately recorded, but which may be stated as from ten to thirty days, secondary cases appear in the house or vicinity of the primary case, or among those who have visited the infected house. As new foci are established the circle of infection widens, and the disease first assumes epidemic proportions after the lapse of six weeks to two months. It is important to bear in mind that as the disease extends the interval between primary and secondary cases appearing in houses situated in districts and suburbs previously exempt often does not exceed a few days. A patient, for example, residing in a hitherto healthy suburb contracts the disease. Other cases immediately follow in the same house. The patient is removed to another house for treatment, and the inmates of his new quarters are in turn seized with fever in three or four days after his arrival. Several instances of this kind will be found in the report of the Lisbon epidemy of 1857 (see also *Bérenger-Féraud*, pp. 546 and 598). Instances such as these, and they are somewhat numerous, should not be ignored merely because they do not seem to harmonise with any of the current theories of infection.

In most instances yellow fever is restricted to particular localities, especially the insanitary quarters of the town, where the streets and lanes are narrow, overcrowded, badly ventilated, and imperfectly exposed to the sun's rays. The progress, extent, and duration of an epidemy are largely dependent on season, temperature, local conditions, and the susceptibility of the population. When the whole community is susceptible, and climatic and other conditions are favourable to its spread, it may invade a whole city, as happened to Barcelona in 1821, where 70,000 attacks and 20,000 deaths took place in a population of about 80,000.

The first cases are, as a rule, most fatal.

At St. Louis in 1900 the case-mortality at the beginning was 72·2 per cent. When the outbreak was at its height it fell to 49·2, and in the period of decline to 40 per cent. It occasionally happens, however, that the disease is mild at the commencement, and increases in virulence as the epidemy declines. In most outbreaks abortive cases occur, marked by gastric troubles, headache, loin pains, and slight icterus. It has been observed that these mild attacks do not always confer an immunity. It has often been asserted that dogs, birds, and other animals suffer from symptoms analogous to those of yellow fever, when that disease is epidemic. This requires confirmation.

Yellow Fever as a Ship Disease. — Special interest attaches to ship outbreaks, both from the part which maritime commerce plays in the diffusion of yellow fever, as well as from the fact that the manner in which it is propagated often admits of being more closely followed on board ship than on land.

1. Ships are particularly liable to become infected. This is partly explained by the susceptibility of those on board, and partly by the habits of sailors, which lead them to frequent the most insanitary localities on shore. Still, we should hardly have expected that a disease propagated by mosquitoes should have shown that special predilection for shipping. Old wooden vessels often fall into an extremely insanitary condition, especially those engaged in the sugar, coal, and timber trades, and these are just the vessels most liable to become infected. Sailing vessels are often detained in the tropics by calms and contrary winds for weeks after leaving a yellow fever port, and this, no doubt, favours the outbreak of the disease. 2. Ship outbreaks often prove extremely fatal, especially in the case of crowded ships of war, where isolation and disinfection cannot effectively be carried out. Not to mention exceptional instances, it has been no uncommon experience of ships of war in comparatively recent times to lose during a short voyage one-half or one-third of their men by yellow fever. The *Eden* in 1829 lost 110 out of 160; the *Bann* in 1823 had 99 attacks and 33 deaths among 107 whites, while none of the 27 negroes on board suffered. The *African* in 1881 lost 18 out of 28, and *La Fortuna* in 1856 had 118 seizures and 56 deaths out of an effective of 212 men. In some few instances where the crew has numbered from 5 to 10 all on board have perished. 3. The virus of yellow fever often clings to a vessel with extreme tenacity. The *Eclair* ship of war was infected in July 1845, and notwithstanding efforts at disinfection she carried the disease with her to England in September, and communicated it to the pilot and several others who had to board her. She lost during the voyage about 60 out of 140 men. After wintering and undergoing repairs and cleaning in England, yellow fever once more broke out on board in the following year when the vessel entered the tropics. Still more remarkable is the case of the United States cruiser *Plymouth*, in which yellow fever reappeared after she had been disarmed, discharged, exposed during the winter to a mean temperature of 28° F., scraped, twice fumigated with sulphurous acid, and washed with chloride of lime. On dismantling the vessel a second time, flies were found between the boardings which had survived the cold and fumigations to which the vessel had been exposed. But it is somewhat difficult to believe that mosquitoes, so sensitive to cold, and whose term of life does not appear to exceed seventy-five days, could have survived the cold for so long a period. 4. A vessel on which the disease has once appeared often proves highly infectious as soon as the holds are opened, and although fresh cases may have ceased to occur among the passengers for a considerable time. The history of the *Anne-Marie* is of peculiar interest in its bearings on many questions connected with the etiology of yellow fever, as the facts were investigated with a thoroughness and recorded with a precision that give them the value of a scientific experiment. The following account is condensed from the original report:—

The *Anne-Marie*, an old wooden vessel, laden with sugar, left Havana on the 13th of June with a crew of sixteen, and arrived at St. Nazaire (France) on the 25th of July 1861, having had nine cases and two deaths from yellow fever during the voyage. The first case appeared on the eighteenth and the last on the twenty-ninth day after leaving Havana, where yellow fever then prevailed. As no fresh case had occurred for thirteen days the vessel was admitted to pratique, the crew discharged, and seventeen labourers belonging to the port engaged to unload her. None of the crew of the infected vessel took ill after landing, and there is no very clear evidence of their having communicated the disease on shore. The work of unloading began on the 27th July, and was finished on the 3rd of August. All the labourers engaged in this work were infected, and nine died; the first case occurring on the sixth and the last on the twelfth day after the work of discharging began. Four of the sick labourers returned to their homes in the country, six to ten kilometres distant from St. Nazaire. They were attended by a local practitioner, Dr. Chaillon, who had never been near the infected vessel. He continued to visit them for eight days. Two days after his last visit (that is, ten days after his first contact with the patients) he was seized with yellow fever and died. A stonecutter working on the quay about 600 feet to the leeward of the vessel, but who had neither visited the ship nor had any intercourse with those on board, was attacked on the 4th of August and died. The

Chastang, a tug vessel with five hands, lying close to the leeward of the *Anne-Marie*, was infected, the first case appearing on the 1st of August. All five were attacked in succession and all died. Four other vessels to the leeward, two of which were not in direct contact with the *Anne-Marie*, also furnished fatal cases. Vessels to the windward, although equally close to the focus of infection, escaped. A shoemaker, who is said to have shared his bed with one of the labourers, was attacked and died; and two women who, it was believed, had been in the company of the sailors, and had obtained some articles of dress from the vessel, were likewise seized with yellow fever, but the precise mode in which they were infected was not satisfactorily ascertained. There were, in all, 44 cases and 26 deaths.

The cessation of the disease on board during the latter part of the voyage while the vessel, as was afterwards proved, was so severely infected, is no less remarkable because it is what is observed in the majority of these instances. The fact that the second officer, who had escaped during the voyage, fell a victim while superintending the discharge of the cargo, proves that the arrest of the disease on board was not the result of all the susceptible having been attacked. The two incidents, however, of greatest etiological significance in this history are the infection of the stonecutter and that of Dr. Chaillon. The case of the stonecutter proves conclusively that the virus or its bearer is transportable for a distance of at least 600 feet by the wind, and it is certainly as conceivable that a mosquito from the vessel communicated the disease in this case as that an infective dose of a bacillary virus should have been diffused through the atmosphere for such a distance. Dr. Chaillon must have contracted the infection in one of three ways: (*a*) By the agency of a mosquito contaminated on the spot by one of the patients, but for this the time was insufficient if reliance can be placed on recent observations; (*b*) By an infected insect or a virus transported from the vessel in the clothing or effects of the sick, and thus the case would be one of transmission by clothing; (*c*) Directly from one of the patients whom he was attending. This last mode is that which naturally suggested itself to those who made the inquiry, and they looked upon it as all the more probable from the circumstance that the doctor was short-sighted, and had to come into very close proximity to the patient he was examining. He was also very assiduous in his attendance on the sick, and personally and perseveringly applied frictions to one of his patients. There is no great difficulty in admitting the possibility of an infected mosquito, fragile as it is, being carried from the ship in the clothing of a patient; but this would be, after all, of the nature of an accident, and it would be somewhat surprising if this mode of transport should have happened at the same time in the case of the shoemaker and the two women, all of whom contracted the disease without having had any direct communication with the infected ship.

Topographical and Altitudinal Relations.— Yellow fever, as we have seen, is distinctly a disease of river and seaports—a peculiarity, as Hirsch observes, "without analogy in the history of other infective diseases." Instances of yellow fever breaking out in inland towns have become more numerous since railways have rendered communication with infected ports more rapid. These inland outbreaks have never, however, exhibited great spreading power, nor has the disease become endemic in localities remote from the sea. It has never become diffused in rural districts, which are those most liable to malaria. Although generally restricted to localities near the sea-level, yellow fever has occasionally broken out at higher elevations—Madrid (2000 feet), Cordova in Mexico and São Paulo in Brazil (2500 feet), and at Newcastle in Jamaica (4000 feet), above the sea-level.

The physical and geological characters of the soil have no influence on the prevalence of yellow fever. Unlike malaria it has no preference for marshy localities. When the French army was encamped near the marsh of Téjeria (four leagues from Vera Cruz, where the disease was decimating the troops) the soldiers suffered terribly from malaria, but were almost exempt from yellow fever. It has often been observed to prevail with special severity where extensive excavations have been in progress.

Meteorological Relations. — In the regions where yellow fever is endemic its wide diffusion is commonly limited to the warm season. In higher latitudes, epidemics uniformly begin in summer or autumn, decline as the temperature falls, and are, in most instances, extinguished when the thermometer reaches freezing-point. The disease, however, will maintain itself for a time at a much lower temperature than that necessary to set it agoing. The influence of rainfall and humidity is less evident, but it is to be remarked that in all its endemic centres yellow fever attains its maximum in the rainy season.

Conditions affecting Susceptibility; Race.— Apart from acclimatisation the negro race exhibits a marked resistance to the infection. The Soudanese troops, 400 in number, forming part of the French expedition to Mexico, and stationed in towns where yellow fever was working havoc among the French and Mexicans, remained completely exempt. This resistance to the infection could not be ascribed to an immunity acquired by their having already had the disease, as these negroes came from a region where yellow fever is unknown. In some epidemies the negro population, acclimatised for

generations to the conditions met with in the States, has paid a considerable tribute to the malady, but, as a rule, when the negro does take the disease it is in a comparatively mild form. In the Decatur outbreak of 1878, for example, the case-mortality of the whites was 44, that of the coloured population 13 per cent. Natives of the north of Europe are more susceptible to yellow fever and suffer from a more severe type of the disease than those from the south. Blair found the case-mortality of the Italians and French to be 17·1, that of the Scandinavians and Russians 27 per cent.

Acclimatisation.—Natives of, and those who have been long resident in, yellow fever countries are seldom attacked when the disease is epidemic. That the immunity of the native is, in part at least, the result of his having already passed through the disease is shown by the fact that when yellow fever was introduced for the first time into Rio the natives suffered as much as strangers. We can give no explanation of the remarkable difference in susceptibility between the natives of the north and south of Europe. One well-developed attack usually confers an immunity for life.

Age; Sex; Profession.—Yellow fever is most fatal to adults. In infants and young children the disease is often so mild that it is with difficulty to be distinguished from other febrile diseases common to that age. Yellow fever, for some reason, is nearly twice as fatal to males as females. Physicians and nurses in yellow fever hospitals have often enjoyed a remarkable exemption when the disease has been epidemic in a town, and this has been urged as a proof that yellow fever is not infective. There are numerous instances, however, in which those in attendance on the sick have suffered out of all proportion to the community generally. Thus, among the French soldiers at Vera Cruz in 1862, of forty-six attendants detailed for the service of the yellow fever wards, forty-three (93 per cent) were attacked and eighteen died, while the rest of the troops stationed in the same town had rather less than 20 per cent of attacks. So perilous, indeed, did the service prove that the authorities had to employ Soudanese negroes to nurse the sick. These, as we have seen, exhibited an inexplicable refractoriness to the virus. Medical men, if unacclimatised, often pay a heavy tribute to the infection. Thus, in Senegal in 1830, eight out of twelve; and in 1878, twenty-two out of twenty-seven medical officers perished. Those who visit the sick in their homes are more frequently seized than those who are on hospital duty. It is difficult to understand why in one epidemy all the attendants should escape, and in another almost all should be attacked, except on the assumption that some intermediary, not always present, is necessary for the transmission of the infection.

Labourers engaged in discharging infected vessels, custom-house officers having to deal with baggage from infected ports, washerwomen employed in washing infected linen, and bakers, are especially liable to be attacked.

Predisposing Causes.—Every kind of excess has been alleged to predispose to the disease, but it is impossible to determine what influence, if any, is to be ascribed to these causes. On one point there is practical unanimity—that the risk of contracting yellow fever is much greater by night than by day.

Sources of the Virus.—There can be no reasonable doubt that the virus, whatsoever it may be, multiplies in the body of the patient, and that the patient himself is the primary source of infection. The doctrine of Hirsch, Scheube, and others, that it is not the yellow fever patient, but the yellow fever *place* that infects, is wrong, in so far as it ignores the fact that the place is directly or indirectly contaminated by the patient.

Modes of Transport and Transmission.—We have already seen the important part played by shipping in the diffusion of yellow fever. The instance of the *Anne-Marie*, and we might have adduced many others, is conclusive that the germ of the disease or its bearer is transportable for short distances by the wind. It is equally well established that the virus is transported from place to place by persons actually sick, or in the incubatory stage of the disease. It is probably spread largely by those suffering from mild or abortive forms not recognised, which may explain a considerable number of what have been looked upon as spontaneous outbreaks. How long the patient remains infective is unknown. The important question remains, whether the virus can be transported in contaminated clothing. Too much value is not to be attached to the negative experiments recently made in Cuba. They certainly do not justify the inference of the Commission "that the disinfection of clothing, bedding, or merchandise supposed to be contaminated is unnecessary." If any amount of negative evidence could prove the non-communicability of the infection by contaminated clothing, the immunity of those employed in handling and washing the linen in the Lazaretto of Philadelphia for a long series of years, that of those similarly employed at Bermuda in 1843, and in the colonial hospital at Demerara in 1841-45, would suffice. But it is impossible to ignore well-authenticated instances which seem to admit of no other explanation than that the virus of yellow fever is, in certain circumstances, transmissible by clothing and other effects. The Lisbon outbreak of 1857 began in the custom-house. It was custom-house officers, soldiers, and guards, whose duty it was to inspect and unpack the baggage coming from Brazil, or to be present in the low, unventilated rooms where this baggage was stored, it was these who were first attacked, it was

by these exclusively that the infection was first spread, and it was these who suffered most. Eight out of nine custom-house officers so engaged died of this disease, and scarcely less was the morality of those on guard at this store. The baggage store was clearly the primary focus of the infection, and it was equally evident that this focus was not established by the presence in it of a person sick of yellow fever. In what way did it become infected if not from contaminated baggage? How are we to explain the Madrid outbreak of 1878 if yellow fever cannot be transmitted by fomites? A body of troops returning from Cuba to Spain remained in perfect health during the voyage. On arriving at Madrid their baggage, which had not been opened from the time they left Cuba, was unpacked. A small outbreak of yellow fever followed, limited to the inhabitants of the district where the returned soldiers were quartered; they themselves, being acclimatised, escaped. It was observed here, as at Gibraltar in 1829, and at Ascension in 1838, that the washerwomen who were employed to wash the soldiers' effects were amongst the first to be seized. To mention one other instance scarcely less free from ambiguity: a medical officer, M. Massola, who had been on duty at the yellow fever hospital and post-mortem rooms at Gorée in 1878, was ordered to proceed to Bakel, a post on the Upper Senegal, 470 miles from Gorée. He left on the 15th, and reached his destination on the 29th of July, and was assisted by a soldier attached to his service, belonging to the post, in unpacking his luggage, which contained the non-disinfected clothing he had been using in the hospital. On the 16th of August this soldier was seized with yellow fever, and in succession seven out of the eight Europeans at that remote post were attacked and died of the disease. Strain (*Journal of Tropical Med.*, April 1899) relates several instances of yellow fever at São Paulo in the interior of Brazil, following the unpacking of boxes and cases from the infected port of Santos. If infected mosquitoes can be stowed away for months in the hold of a ship, it may be contended that there is no improbability in the supposition that they may be conveyed in luggage, but this must be impossible for long distances, if it be the case, as Reed asserts, that the mosquito deprived of water dies within a few days. Some doubts must be entertained of the accounts of the germs lurking in clothing, bedding, sails, and other articles for periods of two or three years, or even longer; but whatever explanation may ultimately prove correct, the transmissibility of the infection of yellow fever by fomites appears to be conclusively established.

Clinical History.—The attack is occasionally preceded, for a few hours or even days, by headache, giddiness, lassitude, loss of appetite, and gastric discomfort, but the cases in which premonitory symptoms are observed are comparatively few. The invasion, which often occurs at night, is announced by a chill or rigor, violent in proportion to the impending attack, although it is well to bear in mind that in malignant cases the chill is more frequently absent. The temperature, which has already begun to rise before the chill, reaches from 102° to 104° F. within the first twenty-four hours. The pulse at this stage is fast, strong, and full, but even as early as the second day it begins to diminish in frequency and force, although the temperature persists or continues to rise. This want of correspondence between the pulse and temperature is of considerable diagnostic value. The face is red or livid and swollen, the eyes brilliant and injected. Photophobia, pain in the eyeballs, distressing frontal or orbital headache, intense lumbar pains radiating towards the pelvis, and articular and muscular pains in the lower extremities, are the symptoms most complained of by the patient at this stage. From the onset there is a feeling of weight and pain in the epigastrium, with anorexia, nausea, a varying degree of thirst; and after a time, in all but the mildest cases, retching or vomiting. Vomiting usually makes its appearance on the second day, first of the contents of the stomach, then of a clear or bile-tinged acid fluid mixed with mucosities. The tongue is moist, contracted, coated with a white fur, and red at the tip and edges. The gums are often swollen and bleed readily. The urine is generally reduced in quantity, is high-coloured, often turbid, acid in reaction, and from an early stage contains traces of albumin. The bowels are constipated.

This state of things lasts for two, three, or four days, according to the gravity of the case. The fever, which is of the sub-continued type, now begins to fall. The pulse tends to regain its normal characters, and the conjunctivæ often, at this stage, begin to assume a yellow tinge. A more or less marked amelioration takes place, which may end in recovery or be followed by grave symptoms.

When recovery is about to ensue the nausea and vomiting abate, the appetite returns, the temperature falls, the urine becomes more abundant, and the albumin disappears.

But the lull, instead of heralding recovery, may be but the prelude to the evolution of a new train of phenomena characteristic of the malady and of grave significance. This issue is to be feared if the gastric distress persists during the lull. There may now be a secondary rise of one or two degrees, or the temperature may persist about the point reached after the remission, or in the worst cases a rapid fall below the normal takes place with symptoms of collapse. It is during this second stage that jaundice, hæmorrhages, and algidity develop. The pulse sometimes becomes abnormally slow,

occasionally sinks to forty a minute, less frequently it is weak and accelerated. The conjunctivæ, skin of face, neck, and upper part of chest assume a tint varying from a straw colour, easily overlooked, to a deep saffron or even bronze hue. In a considerable number of even severe cases distinct jaundice only makes its appearance after death. Gastric irritability, which had temporarily subsided, recurs. The vomited matters begin to show dark grains or flakes—the precursors of the black vomit. Quantities of a fluid resembling coffee-grounds are now ejected. This black vomit, so characteristic of yellow fever, is only one sign of a hæmorrhagic state which frequently manifests itself in epistaxis, oozing from the lips, gums, tongue, ears, or eyes, by hæmorrhages from the bowels, bladder, and uterus, and by ecchymosis into the skin. In malignant cases slight pressure will cause blood to ooze from the tongue as from a sponge. During this stage the urine is scanty, always highly albuminous, and may or may not give the reactions of bile. Sometimes it is suppressed. The excretion of urea is notably decreased, and its accumulation in the blood gives rise to the symptoms of uræmia. The bowels are relaxed, the motions black and tarry or dark and watery.

Recovery not infrequently takes place after all of these symptoms in a minor degree have developed. An increase in the urinary secretion and a diminution in the albumin are the most hopeful signs. This is generally followed by an abatement of the vomiting. When the disease has reached this stage, if the patient escapes with his life, convalescence is protracted.

Death usually occurs from a combination of causes—enfeebled circulation and impeded respiration, with exhaustion from fever, vomiting, and hæmorrhages, and the depression of the nervous system resulting from the presence of urea and other toxic substances in the blood. A fatal termination takes place, as a rule, from the fourth to the eighth day. Relapses are most frequent after abortive cases.

In most outbreaks a considerable proportion of the cases terminate favourably in the first stage. The symptoms, in fact, may be so mild that the true character of the disease is overlooked. At the other extreme are cases of great severity running a rapid course. In these, as we have already said, rigors are generally absent. There is extreme prostration from the very commencement, with coldness of the extremities, vertigo, dilated pupils, profuse multiple hæmorrhages, stupor, delirium, coma, or convulsions, death taking place within thirty-six or forty-eight hours. Each epidemy has its own character. In the Lisbon outbreak of 1857 algidity was the distinguishing symptom. The external temperature fell below the normal; the pulse was small, feeble, and sometimes fast, the features shrunken and livid, the skin generally cyanosed and covered with purpuric spots, the extremities deeply livid and cold, with oozing of blood from nose and gums, black vomit, and almost total suppression of urine. In some epidemies, again, vomiting, purging, cold sweats, and prostration have replaced the ordinary symptoms of the first stage.

Complications.—The more common complications are parotitis of one or both sides, boils, carbuncles, abscesses (if large a good sign), and erythema of the scrotum. In Senegal two cases of gangrene of the extremity of the penis were observed.

The following charts (pp. 597 and 598) represent the more common types of temperature curves—the last three fatal.

Morbid Anatomy.—Rigor mortis appears early, and is extremely marked and persistent. The skin and tissues are almost invariably jaundiced, and hypostatic suffusions particularly pronounced and diffused. The axillary, femoral, or mesenteric glands are often enlarged (Durham).

The membranes and substance of the brain and spinal cord are often highly congested. The lungs and bronchial tubes are more or less congested, but present no characteristic lesions. The pericardium is occasionally ecchymosed, and may contain a moderate quantity of yellow serous or sanguineous effusion. The heart may be either normal in appearance or pale and flabby. Small ecchymotic points are often found in the substance of the organ. The muscular fibres may be normal, or present distinct evidence of fatty degeneration.

The liver is normal in size, and varies in colour from a buff to deep lemon colour. The specific gravity of the hepatic tissue is reduced in proportion as the yellowness is more developed. The hepatic cells are in a state of fatty degeneration, with partial or complete necrosis of the cell. The fatty changes are more marked in some portions of the liver than in others.

The spleen is normal in size, colour, and consistence, unless the patient has suffered from malaria.

The stomach often contains more or less of a fluid similar to black vomit, and presents patches of congestion, hæmorrhagic suffusions, and catarrhal thickening of the mucosa. The small intestine frequently contains a fluid similar to that found in the stomach. The mucous membrane is arborescent in patches. The large intestine is normal.

The kidneys are frequently congested and swollen when death has taken place very early, otherwise they are normal in size and colour. They are always the seat of diffuse parenchymatous nephritis. The epithelium of the convoluted tubules are specially involved and undergo fatty degeneration, necrosis, and desquamation.

DIAGNOSIS.—Attention to the state of the spleen, and a careful examination of the blood and urine, taken along with the circumstances of place and the history of the case, will suffice to distinguish yellow fever from the bilious hæmorrhagic form of malaria and from hæmoglobinuric

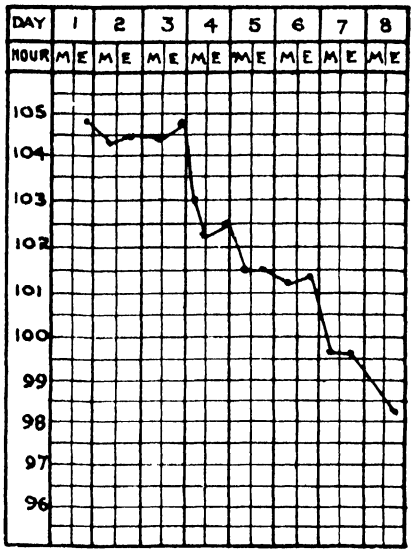


CHART 1.

fever, the only diseases with which it is likely to be confounded.

MORTALITY AND PROGNOSIS.—The proportion of a susceptible population attacked varies greatly in different epidemies. The civil population of Gibraltar in 1804 numbered 14,000. Of these only 25 escaped, 12 of whom had already

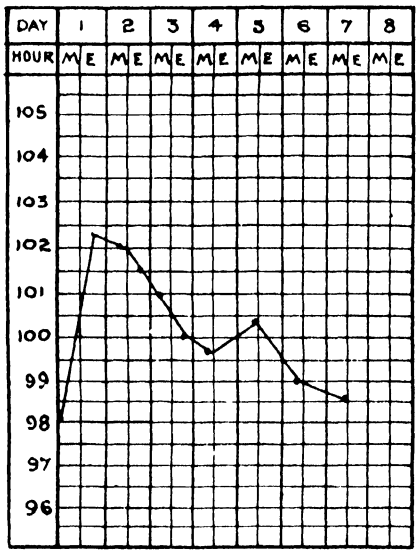


CHART 2.

suffered from the disease. More commonly the diffusion of the infection is limited to the poorest and filthiest parts of a town. The case-mortality exhibits extraordinary extremes, varying from ½ per cent in New Orleans in 1897 to 87 per cent in the same city in 1853.

The prognosis must thus have regard, in the first place, to the type of the prevailing epidemy. The age and nationality of the patient has also to be taken into consideration. A severe, long-continued chill, a high temperature, extreme irritability of the stomach, and, above all, scanty and albuminous urine, indicate a severe attack. A fall of the temperature below normal with signs of collapse, generalised hæmorrhages,

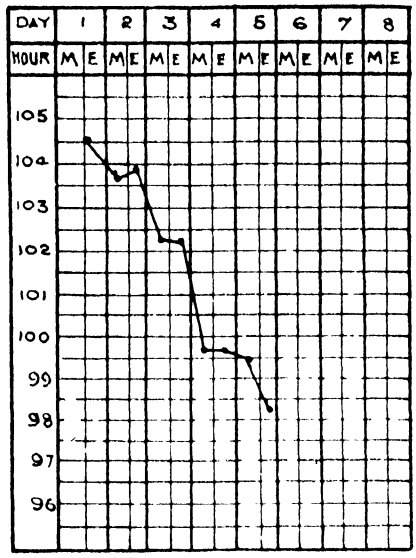


CHART 3.

suppression of urine in the second stage, are separately of the gravest import, and combined prognosticate a fatal issue.

PROPHYLAXIS. — The stamping out of the disease in its endemic foci is obviously an object of primary importance which has never been seriously taken in hand. For the accomplish-

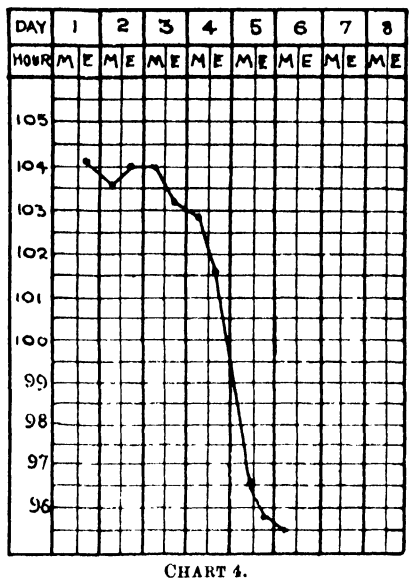


CHART 4.

ment of this object the extermination of mosquitoes should be attempted by all the methods adopted in the case of malaria. The low-lying districts of seaports within the yellow fever zone should be drained, surface pools and other collections of water dried, water-butts and cisterns covered, and every breeding-place of the *culicidæ* abolished. Scarcely less important is the widening and opening up of narrow streets and lanes

to sun and air, and the prevention of overcrowding. As there is reason to believe that native children maintain the infection, their febrile complaints should be attended to, and the contamination of mosquitoes prevented by surrounding their beds with mosquito netting. Were these measures vigorously carried out there is

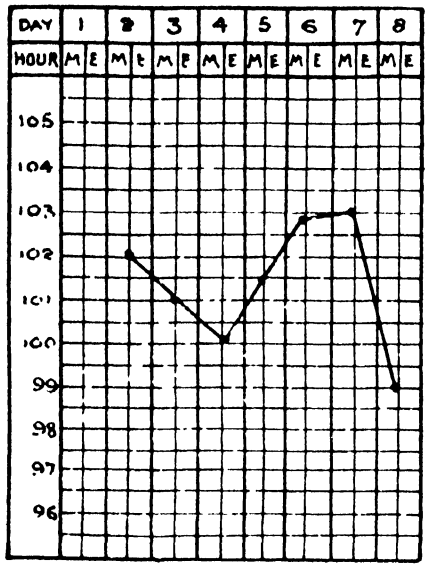


CHART 5.

good reason to hope that this scourge might be greatly mitigated.

To prevent the infection of ships in ports where the disease is present, they should be moored at some distance from the shore or an infected vessel. As mosquitoes are conveyed from shore in lighters this mode of infection

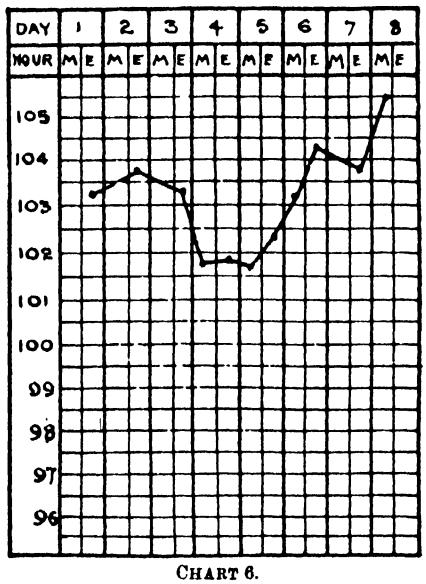


CHART 6.

should be guarded against. It has been suggested to fumigate them with sulphur before they are brought alongside the ship. Unacclimatised sailors should be prevented from landing especially by night.

When the disease makes its appearance on board a vessel while in harbour the sick should be removed to hospital, the crew and passengers transferred to a quarantine station or camp, the cargo disembarked, and the vessel disinfected by sulphurous acid or formaldehyde, clothing and bedding by steam, and the furniture and decks by solution of corrosive sublimate.

When yellow fever declares itself at sea the sick are to be isolated, and the infected quarters evacuated and disinfected. When possible, a course should be steered for colder latitudes. For preventing the introduction of the disease into healthy towns reliance is to be placed on the inspection of vessels from infected ports, the isolation of the sick, and the supervision of the healthy. Disinfection is as necessary in the case of vessels having had the disease on board during the voyage as in the case of those on which there are sick persons on arrival. Detention, however long, is no safeguard.

The principal means of combating the disease when it appears on shore are prompt isolation of sick and contacts, and thorough disinfection of the houses and belongings of the sick. When these means fail the evacuation of the infected district has been carried out with the best results. When yellow fever breaks out in barracks the men should be camped out, all contaminated baggage, bedding, and clothing being left behind for disinfection, and the camp changed if fresh cases should occur. Susceptible persons should remove from an infected locality.

TREATMENT.—It is of primary importance to remove the patient, when possible, from the focus of infection to a healthy locality. A marked amelioration of the symptoms is often found to follow the removal of a patient to an airy room in a healthy part of the town.

In the milder cases the treatment should be mainly hygienic. Constipation is to be obviated by purgatives, the action of the kidneys promoted by alkaline drinks, and the temperature moderated by tepid spongings. Constipation is generally present, and in the more severe cases, at least, it is well to administer at once 5 to 10 grains of calomel, followed next day by sulphate of sodium. The calomel may sometimes be advantageously repeated, as, apart from its purgative and bactericidal properties, it has often a marked effect in diminishing gastric irritability. Castor oil is a favourite purgative with some, but its nauseous taste not infrequently brings on troublesome or even dangerous vomiting. Experience sanctions the use of a warm mustard foot-bath at the beginning of the illness. The feet and legs are placed in water as warm as can be borne, to which mustard has been liberally added, and the patient is enveloped in a blanket. This promotes perspiration, and relieves, temporarily, the headache and pains, and, if the perspiration be free, the fever is lessened.

When the temperature is high one or two doses of antipyrine, given at the early stage when the pulse is strong, are occasionally of service.

In all but the mildest cases resort should be had to bichloride of mercury, in combination with bicarbonate of soda, as suggested by Sternberg. His formula is: bichloride of mercury, ½ of a grain; bicarbonate of soda, 150 grains; water, 1 quart. Three table-spoonfuls are to be taken every hour ice-cold. This remedy has been found to allay gastric irritability and increase the urinary secretion. Under this treatment 374 cases treated by different physicians in the United States, Cuba, and Brazil, the mortality was 7·3 per cent. These results justify its use in all serious forms of the disease.

While this treatment is being employed two symptomatic indications must be steadily carried out, viz. to favour the elimination of toxins by the kidney and to moderate the fever. The issue of a case, so far as it is determined by treatment, will depend upon our success in meeting these indications.

The patient should be encouraged to drink freely of some mildly alkaline water. To those who can afford and procure it, from two to four litres of Vichy water should be administered daily. Aerated water, with the addition of 60 grains of bicarbonate of soda per litre, will answer if Vichy water cannot be had. The water should be given in small doses, say half an ounce to an ounce every quarter of an hour, either at the ordinary temperature of the room or ice-cold, as may be best supported by the patient. Should the water be rejected, cold diluent enemata may be administered. The amount injected at a time should not exceed what can be retained and absorbed, and the injections should be repeated every two or three hours. As soon as the patient's stomach can bear it, the alkaline drinks are to be resumed. The object is to promote the action of the kidneys and prevent the accumulation of toxins in the system. In severe cases, where the stomach cannot tolerate liquids, the subcutaneous injection of 200 to 300 c.c. of a sterilised solution of common salt (2½ drs. in 40 oz. water) may be of service.

When the temperature is under 102° F. the whole body—the back as well as the front of the trunk—and the limbs should be successively washed every two or three hours with a large towel wrung out of water at a temperature 80° to 85° F., a little higher or lower according to the sensations of the patient. When the temperature reaches 103° and the skin is dry, ice should be added to the water to bring it to 60° or 70° F., and the washings repeated every hour. The higher the temperature the more ice is to be added to the water. That part of the body only which is being washed should be exposed. It should then be thoroughly dried and covered, and another part treated in the same way. When the skin is perspiring, cold sponging should be avoided, and, indeed, in this case is not needed. When there is much heat and congestion of the head an ice-bag should be applied. These cold washings of the skin are agreeable to the patient; they moderate the temperature, increase the urine, and allay gastric irritability. When the temperature exceeds 104° F., and the measures recommended fail to control it, resort is to be had to the cold bath. The patient is placed in a bath at 85° or 90° F., which is to be reduced to 80° or 70° F., or lower, so as to reduce the temperature of the body by two degrees at least. Fifteen minutes will usually be sufficient to effect this, and the ordinary precautions to prevent collapse taken. In adynamic cases the warm bath or hot packs should be used. In cases marked by collapse, the application of heat and friction to the extremities and subcutaneous injections of ether are called for. If the heart's action is feeble and irregular, digitalis in small doses along with stimulants should be tried.

Vomiting is one of the most distressing symptoms of the disease. The treatment already recommended, if systematically carried out, will do much to control it. Sinapisms over the epigastrium, a few drops of chloroform given at frequent intervals, drop doses of creasote, an ice-bag over the pit of the stomach, are the remedies most commonly employed. They are all occasionally useful; more frequently they all fail.

In hæmorrhagic cases little good can be expected from medicines. It was thought that the perchloride of iron in full doses was of some service in the Lisbon epidemy. Some recommend ergot hypodermically in these cases, but it is seldom of any service. It was in this form that King and Laird found advantage from the use of oil of turpentine in doses of 20 minims, in camphorated water, three times a day, a little spirits of nitrous ether being added if strangury occurred.

Diet.—During the first two or three days no food whatever, liquid or solid, should be given. When the fever has subsided and gastric irritability has disappeared, milk in small quantities, alone or mixed with lime water, should be given cautiously. As convalescence progresses, chicken broth, or beef tea, and light farinaceous foods should be allowed. For some weeks after recovery the strictest regulation of the diet and habits of life should be insisted on to obviate the risk of relapse.

Yellow Jack. *See* YELLOW FEVER.

Yellow Lotion. *See* YELLOW WASH.

"Yellows."—Jaundice, especially in veterinary medicine.

Yellow Spot.—The macula lutea. *See* RETINA AND OPTIC NERVE (*Retina, Ophthalmoscopic Appearances*).

Yellow Vision.—Xanthopsia. *See* Colour Vision (*Colour-Blindness, Varieties*); Jaundice (*Obstructive, Symptoms*); Santonin; Toxicology (*Vegetable Poisons, Santonin*).

Yellow Wash.—Lotio hydrargyri flava. *See* Mercury.

Yemen Ulcer.—Tropical Phagedæna. *See* Skin Diseases of the Tropics (*Tropical Phagedæna, Synonyms*).

Yerba Santa. — Mountain balm or Eriodictyon (literally, *sacred herb*), the leaves of a Californian shrub (*E. Californicum*), containing a large quantity of gum resin; it is used as an expectorant in acute bronchitis; also, as an excipient, for hiding the taste of quinine; the dose is 15 grains, and of the fluid extract (official in the United States Pharmacopœia) 15 minims to 1 fl. dr.

Yerbine.—A variety of caffeine; it is obtained from the *Ilex paraguayensis* (*Yerba maté*).

Yersin's Serum. *See* Plague (*Treatment, Preventive Sera*).

Yew. *See* Toxicology (*Abortifacients*).

Ymshi. *See* Morphinomania (*Pipe Residue in Opium Smoking*).

Yohimbin.—An alkaloid obtained from a West African tree (the Yohimbehe tree), forming a white amorphous powder slightly soluble in water, readily so in alcohol, ether, and chloroform; it has a local anæsthetic effect when applied to the conjunctiva and when injected hypodermically (1 per cent solution) into the tissues; there is a good deal of evidence that Yohimbin acts as an aphrodisiac in cases of sterility due to impotence, when its action is due to increased excitability of the sacral part of the spinal cord.

Yolk. *See* Diet (*Animal Foods, Eggs*); Fœtus and Ovum, Development of (*Ovum, Parts*).

Young-Helmholtz Theory. *See* Colour Vision (*Physiology, Theories of Colour Perception*).

Young's Rule.—The rule for determining the doses of medicines to be given to children; twelve is added to the age, and the result is divided by the age, the quotient becoming the denominator of the fraction of which 1 is the numerator, thus $\frac{6}{6+12}=\frac{1}{3}$; *i.e.* one-third of the adult dose is the proper quantity for a child of six years. This rule does not apply to opium. *See* Prescribing (*Dosage, Age*).

Ypsiliform.—Having the shape of the Greek upsilon (Y); *e.g.* ypsiliform, hypsiloid, or Y-shaped fused twins.

Yumoto. *See* Balneology (*Japan, Sulphurous Waters*).

Yverdon. *See* Balneology (*Switzerland, Sulphur Waters*).

Zaglas' Ligament. — Part of the posterior ligament of the sacro-iliac joint.

Zahnleiste. — The dental lamina or (literally) tooth-band, being an epithelial downgrowth from the thickening of the epithelium of the mouth from which the gum is developed; the teeth appear along the under surface of the dental lamina. *See* Teeth (*Development*).

Zanaloin.—The aloin obtained from Zanzibar aloes; socaloin. *See* Aloes.

Zang's Space.—In descriptive anatomy Zang's space is that found in the supra-clavicular region of the neck between the two tendons of origin of the sternocleido-mastoid muscle.

Zappert's Counting Chamber.—A modification of the Thoma-Zeiss counting chamber for the estimation of the number of blood corpuscles.

Zea Mays.—Indian corn or maize. *See* Pellagra (*Causes*).

Zeismus.—The morbid condition due to eating diseased maize; pellagra (*q.v.*).

Zeiss' Glands.—The sebaceous glands of the eyelids, connected with the eyelashes; suppuration in one of them causes a Zeissian stye (*hordeolum zeissianum*).

Zeller's Operation. *See* Deformities (*Syndactyly, Treatment, Operative*).

Zelotypia.—An abnormally great degree of zeal in bodily or mental effort (Gr. ζῆλος, rivalry or emulation; τύπος, a blow or type).

Zenker's Degeneration. — A degenerative change in muscular tissue (waxy or hyaline in character), occurring in typhoid and other long-continued fevers.

Zenker's Paralysis. — A form of paresis of the lower limbs due to long-continued kneeling or squatting as in certain occupations; the external popliteal nerve is that most affected.

Zermatt. *See* Therapeutics, Health Resorts (*Switzerland*).

Zestocausis or Zestokausis.—The vaporisation or cauterisation of the uterine cavity by means of a two-way metal catheter

with its outer tube closed at the end (zestocautery) and heated by steam. *See* ATMOKAUSIS; CURETTAGE.

Ziehl's Solution.—A staining fluid, containing carbolic acid and fuchsin, for the recognition of the tubercle bacillus. *See* POST-MORTEM METHODS (*Bacteriological Stains, Fuchsin*).

Ziemssen's Points.—The points of entrance of motor nerves into muscles; they are to be chosen for the application of electricity.

Ziemssen's Test.—A means of distinguishing a distended gall-bladder from a renal tumour; it consists in distending the colon with gas. *See* GALL-BLADDER AND BILE-DUCTS, DISEASES OF (*Tumours of Gall-Bladder, Diagnosis*).

Zimmermann's Corpuscles or Particles. — Blood platelets or plaques. *See* BLOOD (*Blood Plates*).

Zinc. *See also* DERMATITIS TRAUMATICA ET VENENATA (*Causal Agents, Chemical*); TOXICOLOGY (*Irritants, Zinc*). The metal itself, Zincum, is not official. The following are the official salts:—1. ZINCI OXIDUM. Insoluble. *Dose*—3-10 grs. *Preparation*—Unguentum Zinci. 2. ZINCI CARBONAS. Insoluble. *Dose*—1-3 grs. 3. ZINCI SULPHAS. Soluble, 10 in 7 of water. *Dose*—1-3 grs.: 10-30 grs. as an emetic. *Preparation*—Unguentum Zinci Oleatis. 4. ZINCI CHLORIDUM. Freely soluble. *Preparation*—Liquor Zinci Chloridi. 5. ZINCI ACETAS. *Dose*—1-2 grs. 6. ZINCI VALERIANAS. 7. ZINCI SULPHOCARBOLAS. There are also a large number of unofficial salts.

The salts of zinc are chiefly used for purposes of external application. The *chloride* is a powerful caustic, and in the form of a stick or paste may be employed for the destruction of warts, etc. In solution it is a strong antiseptic, and was formerly employed to disinfect bed-pans, closets, etc. A solution of 40 grs. to the ounce has been used on gauze or lint for plugging cavities after operations in the oro-nasal region, but it causes considerable pain. Very weak solutions have been recommended for conjunctivitis and gonorrhœa. The *sulphate* is an excellent astringent and antiseptic for application to ulcers and raw surfaces, for conjunctivitis, and as an injection for gonorrhœa, leucorrhœa, and similar conditions. The strength of solution varies according to circumstances, but it is usually employed as Lotio Rubra of the strength of 2 grs. to the ounce and coloured with compound tincture of lavender. The *oxide* and the *carbonate* are widely used in the form of powder or ointment for their mild astringent effect. Purified zinc carbonate, known as Calamine, is an excellent astringent. Internally the sulphate is a prompt emetic, and does not cause much nausea or depression. The administration of zinc salts in various affections of the nervous system was formerly in considerable vogue, but has now been abandoned. The oxide of zinc will sometimes check the night-sweats of phthisis. The sulphate has been prescribed in cases of serous diarrhœa.

Zincalism. — Chronic zinc-poisoning as is met with in zinc-smelters, zinc-grinders, brassfounders, calico-printers, and others. *See* TOXICOLOGY (*Irritants, Zinc*).

Zinc Perhydrol.—A white powder containing 50 per cent of zinc peroxide (ZnO_2); it is used externally in the form of ointment or powder as a disinfectant in wounds, ulcers, and burns, and as an antiseptic in some skin diseases such as *dermatitis traumatica et venenata, acne rosacea*, etc.

Zincum. *See* ZINC.

Zingiber.—Ginger: the dried rhizome (scraped) of *Zingiber officinale*. It contains an aromatic volatile oil, gingerol, and several resins. *Dose*—10-20 grs. *Preparations*—1. Syrupus Zingiberis. *Dose*—½-1 ʒ. 2. Tinctura Zingiberis. *Dose*—30-60 m. It is used as a carminative and stomachic, and also merely as a flavouring agent. Combined with purgative medicines it has the effect of preventing griping. It is somewhat constipating, and is a frequent ingredient of mixtures for checking simple diarrhœa.

Zinn, Artery of.—The central artery of the retina.

Zinn, Ligament or Ring of.—Part of the common tendon of the recti muscles of the eyeball, which forms a circular fibrous sheath for the eye and is not attached to the sheath of the optic nerve.

Zinn, Zonule of.—The suspensory ligament of the crystalline lens of the eyeball. *See* LENS, CRYSTALLINE (*Anatomy*).

Zittman's Decoction. *See* SYPHILIS (*Tertiary, Treatment, Zittman's Regimen*).

Zoamylin.—Glycogen (*q.v.*).

Zoanthropia.—A form of insanity in which the sufferer imagines his body to be inhabited by an animal, or himself to be transformed into an animal.

Zollikofer's Method.—A method for bringing out the iodine reaction in the case of leucocytes; the fresh blood film is placed for a few minutes in a closed bottle containing some crystals of pure iodine.

Zomotherapy or Zoömotherapy.—The treatment of disease (tuberculosis) by a diet of raw meat.

Zona.—Without any qualifying adjective *zona* means herpes zoster or shingles. *See* HERPES (*Herpes Zoster or Zona*). With a qualifying adjective zona is a name given to various parts of the body, etc.; e.g. *zona arcuata* (the internal zone of the basilar membrane of the cochlea), *zona cochleæ* (the cartilaginous part of the lamina spiralis), *zona denticulata* (the lamina denticulata), *zona glomerulosa* (the outer part of the cortical portion of the adrenal body), *zona pellucida s. radiata* (the vitelline membrane of the ovum), *zona vasculosa* (part of the Graafian follicle of the ovary), and *zona virginitatis s. castitatis* (the hymen).

Zonary.—Having the form of a zone, *e.g.* the *zonary placenta* of the Carnivora.

Zone.—A girdle, tract, or belt (Gr. ζώνη, a belt); or, in a special sense, herpes zoster (zona). A *hysterogenous zone* is a part of the body (*e.g.* ovarian region) which, if subjected to pressure in a hysterical woman, leads to the supervention of a hysterical fit; similarly, a *hypnogenous zone* is one pressure upon which causes sleep.

Zonula.—A little zone or girdle, *e.g.* the *zonule of Zinn* (*see* ZINN), the *zonula cartilaginea* (crista spiralis), etc.

Zonular.—Belonging to a little zone or belt, e.g. *zonular cataract* (lamellar cataract).

Zonulitis.—Inflammation of the zonule of Zinn. *See* LENS, CRYSTALLINE.

Zoö-.—In compound words *zoo-* (Gr. ζῷον, an animal) means relating to animals; e.g. *zoo-chemistry* (the chemistry of animal life), *zoo-dynamics* (animal physiology), *zooglœa* (a stage in the life-history of some microbes in which they lie in a secreted, gelatinous matrix), *zooid* (resembling an animal), etc.

Zoster.—A girdle or belt (Gr. ζωστήρ, a warrior's belt), as in such terms as *herpes zoster*, *zoster facialis* (herpes zoster in the region of the fifth cranial nerve), etc.

Zosteriform.—Resembling herpes zoster or zona, *e.g.* zosteriform morphœa. *See* SCLERODERMIA (*Circumscribed*).

Zuckerkandl's Convolution.—The subcallosal convolution of the brain.

Zuckerkandl's Vein.—A small vein (one of the ethmoid veins) by means of which the veins of the lateral wall of the nose communicate with those of the cerebrum.

Zygapophysis.—The articular process of a vertebra.

Zygo-.—In compound words *zygo-* (Gr. ζυγόν, a yoke or jugum) means shaped like a yoke or related to the zygoma of the facial skeleton; e.g. *zygomaticus major* (a small muscle arising from the malar bone), *zygomaxillary* (belonging to the zygoma and superior maxillary bone), *zygospore* (a spore arising from the conjugation or zygosis of two gametes), etc.

Zygoma.—The yoke-shaped bone formed by the junction of the zygomatic process of the temporal bone with the temporal process of the malar bone; from it comes the descriptive term zygomatic; e.g. *zygomatic arch*, *zygomatic fossa*, *zygomatic spine*, etc.

Zymase. *See* ENZYMES (*Monosaccharide-splitting Enzymes*).

Zymine.—A ferment extracted from the pancreas, or simply, an enzyme. *See* ENZYMES; PHYSIOLOGY, TISSUES (*Zymin-secreting Epithelium*).

Zymo-.—In compound words *zymo-* (Gr. ζύμη, leaven) means related to fermentation; e.g. *zymolysis* (decomposition due to the action of organised ferments, *zymosis* (fermentation by organised ferments, or, the morbid state due to a zymotic disease), etc.

Zymogen.—A substance which exists in some secreting glands and which by chemical action produces an enzyme or ferment; literally, a ferment-producer. *See* PANCREAS (*Physiology*); PHYSIOLOGY, TISSUES (*Zymin-secreting Epithelium*).

Zymogenic.—Ferment-producing or fermentative, e.g. *zymogenic bacteria*. *See* MICRO-ORGANISMS (*Composition of Bacteria*).

Zymophore.—Having a fermentative action, e.g. the *zymophore group* of the complement in hæmolysis. *See* IMMUNITY.

Zymotic.—Belonging to the process of zymosis or capable of producing zymosis (fermentation or an infectious disease); e.g. *zymotic disease* (an infectious fever), *zymotic theory* (the theory that in zymotic disease the infecting particles act as ferments on the tissues of the individual attacked), etc. *See* DIPHTHERIA (*Etiology*); EPIDEMIOLOGY; PUERPERIUM, PATHOLOGY (*Fever in the Puerperium*, *Zymotic Causes*).

INDEX

	PAGE
Thiersch's Method	1
Thiersch's Operation	1
Thigh	1
Thio-	1
Thiol	1
Thionin	1
Thioresorcin	1
Thiosinamine	1
Thiothrix	1
Third Nerve	1
Thirst	1
Thiry's Fistula	1
Thlapsis Depressio	1
Thlipsencephalus	1
Thoma-Zeiss Hæmocytometer	2
Thomas's Splint	2
Thompson's fluid	2
Thompson's Line	2
Thomsen's Disease	2
Thoracentesis	2
Thoracic	2
Thoraco-	2
Thoracoplasty	2
Thorax	2
Thornwaldt's Disease	2
Thought-Reading	2
Thread Worms	2
Three Days' Fever	2
Thremmatology	2
Thresh's Disinfector	2
Thresh's Method	2
Thrills	2
Throat	2
Thrombin	2
Thrombo-	2
Thrombokinase	3
Thrombosis	3
Thrombus	3
Thrush	3
Thuja	3
Thumb	3
Thus Americanum	3
Thyme, Oil of	3
Thymectomy	3
Thymic	3
Thymol	3
Thymus Gland	3
"Thymus-Tod"	5
Thynnus	5
Thyro- or Thyreo-	5
Thyroidectomy	5
Thyroid Gland, Medical Section	5
Thyroid Gland, Surgery of	19
Thyroid Gland, Therapeutics	25
Thyroidism	25
Thyroiditis	25
Thyroidotomy	25
Thyroprivus	25
Thyrotomy	26
Tibia	26
Tibio-	26
Tic	26
Tic Douloureux	26
Tickling	26
Ticks	26
Tidal Air	26
Tidy's Process	26
Tight-lacing	26
Tightness	26
Tigretier	26
Tin	26
Tincæ, Os	26
Tinctura or Tincture	26
Tinea	26
Tingling	26
Tinkling, Metallic	26
Tinnitus Aurium	26
Tirol	29
Tisane	29
Tissue	29
Titubation	29
Toast Water	29
Tobacco	29
Tobi Hi	29
Tobin's Tube	29
Tocodynamometer	29
Tocology	29
Toddalia	29
Toes	29
Toilet	29
Toisson's Solution	29
Tokelau	29
Tolu, Balsam of	29
Toluene or Toluol	29
Toluidin-Blue	29
Toluylene-diamine	29
Tolypyrin	30
Tomatoes	30
Tomes' Fibres	30
Tonga	30
Tongue	30
Tongue-Tie	42
Tonic	42
Tonka Bean	42
Tonometer	42
Tonsillitis	42
Tonsillolith	42
Tonsillotome	42
Tonsils, Diseases of	42
Tonus, Muscle	47
Tooth	47
Topæsthesia	47
Topalgia	47
Tophaceous	47
Tophus	47
Topical	47
Topo-	47
Torcular Herophili	48
Tormina	48
Torpor	48
Torquay	48
Torricellian Vacuum	48
Torsion	48
Torticollis	48
Torula	48
Torus	48
Total Aphasia	48
Total Necrosis	48
Touch	48
Touch Corpuscles	48
Tourette's Disease	48

	PAGE
Tourniquet	48
Tow	48
Toxæmia	48
Toxalbumins	48
Toxalbumoses	48
Toxic	48
Toxicity	48
Toxicogenic	48
Toxicology	48
Toxicomania	79
Toxicosis	79
Toxine	79
Toxiphobia	79
Toxoid	79
Toxone	79
Toxophore	80
Toynbee's Experiment	80
Trabecula	80
Trabs Cerebri	80
Trachea, Affections of the	80
Tracheal Tugging	83
Tracheitis	83
Trachelectomy	83
Trachelismus	83
Trachelo-	83
Tracheloparasitus	83
Trachelorrhaphy	84
Tracheloschisis	84
Tracheloterata	84
Trachelotomy	84
Tracheo-	84
Tracheotomy	84
Trachielcosis	84
Trachinus	84
Trachoma	84
Tracing	84
Tract or Tractus	84
Traction, Axis	84
Tractoration	84
Trades, Dangerous	84
Tragacanth	100
Tragus	101
Training	101
Trance	101
Trans-	101
Transference	101
Transfusion	101
Transillumination	103
Transitional Epithelium	103
Transition Organism	103
Transmigration	103
Transmission	103
Transplantation	103
Transport	103
Transposition	103
Transudation	103
Transvaal	103
Transversalis	103
Transverse	103
Trap	103
Trapezium	103
Trapezius	103
Trapezoid	103
Trapp's Coefficient	104
Traube-Hering's Curves	104
Traube-Rosenstein Theory	104
Traube's Corpuscles	104
Traube's Plugs	104
Traube's Space	104
Traube's Theory	104
Trauma	104
Traumaticin	104
Traumatides	104
Traumatol	104
Traumatopnœa	104
Tread	104
Treatment	104
Trefriw	104
Trematodes	104
Trembles	104
Trembling Palsy	104
Tremens	104
Tremor	104
Trendelenburg Posture	107
Trepan	107
Trephine	107
Triacid	107
Triacid Stain	107
Triakaidekaphobia	107
Triangle	107
Triangular	107
Tri-	107
Tricephalus	107
Triceps	107
Trichauxis	107
Trichiasis	107
Trichina	108
Trichiniasis	108
Tricho-	108
Trichocephalus	108
Trichoglossia	108
Trichophyton	108
Trichophytosis	108
Trichorrhexis Nodosa	108
Trichotrachelidæ	108
Trichromic Vision	108
Trichterbrust	108
Tricornis	108
Tricresol	108
Tricrotic	108
Tricuspid	108
Triencephalus	108
Triethylamine	108
Trifacial or Trigeminal	108
Trigger Finger	108
Trigone	108
Trigonocephalus	108
Trillat's Autoclave	108
Trilocular	108
Trimethylamine	108
Trinitrin	108
Triocephalus	108
Trional and Tetronal	109
Triorchidy	109
Trioxypurin	109
Tripara	109
Tripe	109
Triphalangy	109
Triphenamine	109
Triphenin	109
Tripier's Amputation	109
Triple	109
Triplets	109
Triplopia	109
Tripod Fœtus	109
Triradiate	109
Trismus	109
Tristitia	109
Triticum	109
Trituration	109
Trocar	109
Trochanter	109
Trochiscus or Troche	109
Trochlea	109
Trochlear	109
Trochocephaly	109
Trommer's Test	109
Tromomania	109
Tropacocaine	110
Trophic	110
Trophoblast	110
Trophoneurosis	110
Tropical	110
Tropics, Unclassed Fevers of the	110
Tropidonotus Natrix	117
Tropometer	117
Trough Closet	117
Trousseau's Disease	117
Trousseau's Marks	117
Trousseau's Symptom	117
True	117
Trusses	117
Trypanosoma	117
Trypanosomiasis	117
Trypsin	117
Trypsinogen	117
Tryptophane	117
Tscherning's Theory	117
Tsetse Fly	117
Tubal	117
Tube	117
Tube-Casts	117
Tuber	117
Tubercle	117
Tubercula	117
Tubercular	117
Tuberculide	117
Tuberculin	117
Tuberculocidin	118
Tuberculoderma	118
Tuberculosis	118
Tuberculotoxine	150
Tuberculum	150
Tuberiform Abscess	150
Tuberose	150

	PAGE
Tuberosity	150
Tubo-	151
Tubule	151
Tuffier's Disease	151
Tufnell's Treatment	151
Tugging, Tracheal	151
Tulpius, Valve of	151
Tumours	151
Tumours, Inoperable, Treatment of	164
Tumours of the Skin	168
Tumultus	174
Tunbridge Wells	174
Tunic or Tunica	174
Tunicata	174
Tuning Fork	174
Tunnel-Anæmia	174
Tunnel Disease	174
Tunny	175
Tupelo	175
Turacin	175
Turacoporphyrin	175
Turbinal	175
Turbinated Bones	175
Türck's Bundle	175
Türck's Column	175
Türck's Trachoma	175
Turkey	175
Turkish Bath	175
Turmeric	175
"Turn of Life"	175
Turpentine	175
Turpeth	175
Tussiculation	175
Tussis Convulsiva	175
'Tween-Brain	175
Twelfth Nerve	175
Twins	176
Twitching	176
Tyloma	176
Tylophora Leaves	176
Tylosis	176
Tympana	176
Tympanic	176
Tympanites	176
Tympanitis	176
Tympanum	176
Type, Menstrual	176
Typesetters	176
Typewriter's Cramp	176
Typhlitis	176
Typhlo-	176
Typhoid	176
Typhoid Fever	176
Typhomalaria	202
Typhomania	203
Typhopneumonia	203
Typhotoxin	203
Typhus Fever	203
Tyremesis	210
Tyro-	210
Tyrosin	210
Tyrotoxicon	210
Tyrrel's Fascia	210
Tyson's Glands	210
Uberty	210
Ucambin	210
Uchetu	210
Udransky's (Von) Test	210
Uffelmann's Test	210
Uhlenhuth's Test	210
Ulæmorrhagia	210
Ulcers and Ulceration	210
Ulcerated	218
Ulceration	218
Ulcerative	218
Ulcus	218
Ulé	218
Ulexine	219
Ulitis	219
Ulna	219
Ulo-	219
Ultra-	219
Ultra-Violet Rays	219
Ultzmann's Test	219
Ululation	219
Umbilical	219
Umbilicus, Diseases of	219
Umbo	221
Unavoidable Hæmorrhage	221
Unborn Infant	221
Unciform	221
Uncinaria Duodenalis	221
Uncinariasis	221
Uncinate	221
Unconscious Cerebration	221
Unconsciousness	221
Uncontrollable	231
Uncus	231
Underhung Jaw	231
Underpinning	231
Undue Influence	231
Undulant Fever	231
Ungual	239
Unguentum	239
Ungulates	239
Uni-	239
Uniceptor	239
Unicornis	239
Unilateral	239
Uniovular	239
Unit	240
Unna's Bandage	240
Unna's Dermatosis	240
Unna's Paste	240
Unsound Food	240
Unsound Mind	240
Unwell	240
Unzen	240
Urachus	240
Uræmia	243
Uræmic	250
Ural or Uralium	250
Uranalysis	250
Uraniscoplasty	250
Uranism	250
Uranomania	250
Uranoschisis	250
Urari	250
Urate	250
Uratic	250
Uraturia	250
Urberoago de Alzola	250
Urea	250
Urechysis	250
Uredo Fœtida	250
Uremia	250
Ureters	250
Ureteralgia	250
Ureterectomy	250
Ureteritis	250
Uretero-	250
Ureterotomy	251
Urethane	251
Urethra, Diseases of	251
Urethral	262
Urethralgia	262
Urethrameter	262
Urethritis	262
Urethro-	262
Urethrotomy	262
Urgency Certificate	262
Urginea	262
Uriage	262
Uric Acid	262
Uricacidæmia	266
Uricedin	266
Urinal	266
Urinalysis	266
Urinary	266
Urination, Disorders of	266
Urine, Pathological Changes in	269
Urine, Bacteria in	307
Urinettes	312
Uriniferous	312
Urino-	312
Urnings	313
Uro-	313
Uroanthropos	313
Urobilin	313
Urochrome	313
Urocyanogen	313
Urocyanosis	313
Uroerythrin	313
Urogenital	313
Urogosan	313
Urohæmatoporphyrin	313
Urolith	313
Urophæin	313
Uropoietic	313
Urorosein	313
Urostealiths	313
Urotoxic	313
Urotropin	313

	PAGE
Uroxanthin	313
Urtica	313
Urticaria	313
Uskow's Theory	319
Ustilago	319
Ustion	319
Uteralgia	319
Uterine	319
Utero-	319
Uterus, Diseases of	319
Malformations of the Vulva, Vagina, and Uterus	319
Displacements of the Uterus	327
Inflammations of the Uterus	340
Benign or Non-malignant Tumours of the Uterus	348
Malignant Tumours of the Uterus	363
Insanity associated with Uterine and Ovarian States	376
Utricle or Utriculus	378
Uva Ursi	378
Uvea	378
Uvula, Diseases of	378
Uvulaptosis	380
Uvulatomy or Uvulotomy	380
Uvulitis	380
Vaccination	380
Vaccine	400
Vaccinia	401
Vaccinophobia	401
Vaccinosyphilis	401
Vacuolation	401
Vagabond's Disease	401
Vagina, Disorders of the	401
Vaginal	414
Vaginectomy	414
Vaginismus	414
Vaginitis	414
Vagino-	414
Vagitus	414
Vago-	414
Vagrant's Disease	414
Vagus Nerve	414
Valée's Law	415
Valencia	415
Valenta Test	415
Valentine's Beef Juice	415
Valerian	415
Valerian, Indian	416
Valescur	416
Valetudinarianism	416
Valeur Globulaire	416
Valgus	416
Vallecula	416
Valleix's Points	416
Vallum	416
Vals	416
Valsalva's Experiment	416
Valsalva's Sinus	416
Valsuani's Disease	416
Valve	416
Valvula	416
Valvular Disease	416
"Vana"	416
Vapores	416
Vaporisation of the Uterus	416
Vaporole or Vapourole	416
Vapours	416
Varese	416
Varicella	419
Varicocele	419
Varicose	419
Variola	419
Varioloid	419
"Varium"	420
Varix	420
Varnish-Making	420
Varolii, Pons	420
Varus	420
Vas	420
Vascularisation	420
Vasectomy	420
Vaseline	420
Vasicin	420
Vasiform	420
Vaso-	420
Vasogen	420
Vasotribe	420
Vastus	420
Vater's Ampulla	420
Vater's Corpuscles	420
Veal	420
Vecordia	420
Vectis	420
Vector	420
Vegetables	420
Vegetarian Diet	420
Vegetations	420
Vehicle	420
Veins, Diseases of	420
Velamentous	427
Veldt Fever	427
Veldt Sore	427
Vella's Fistula	427
Velpeau's Hernia	427
Velum	427
Vena	427
Venereal Disease	427
Venesection	432
Venom	433
Venous	433
Venter	433
Ventilation and Warming	433
Ventnor	440
Ventose	440
Ventrad	440
Ventral	440
Ventricle	440
Ventrofixation	441
Veratrine	441
Veratrum Viride	441
Verbigeration	441
Verga's Ventricle	441
Verheyen's Stars	441
Vermicelli	441
Vermicides	441
Vermiform	441
Vermifuges	441
Vermination	441
Vermin Paste	441
Vermis	441
Vernet-les-Bains	441
Vernix Caseosa	441
Veronal	441
Verruca	441
Verrücktheit	441
Verruga Peruana	441
Version	444
Vertebra	444
Vertebral	444
Vertex	444
Vertigo	444
Verumontanum	448
Vesalius, Foramen of	448
Vesalius, Ligament of	448
Vesania	448
Vesical	448
Vesicants	448
Vesicle	448
Vesico-	448
Vesicula	448
Vesiculæ Seminales	448
Vesicular	457
Vesiculitis	457
Vesiculo-	457
Vessels	457
Vestibule	457
Vestigial	457
Vesuvin	457
Vetch	457
Veterinary Medicine	458
Veternositas	458
Via	458
Viability	458
Vibices	458
Vibration	458
Vibrio	458
Vibrio-Massage	458
Viburnum Prunifolium	458
Vicarious Menstruation	458
Vice	458
Vichy	465
Vicq d'Azyr's Bundle	465
Vicq d'Azyr's Operation	465
Vidago	465
Vidian Canal	465
Vienna Paste	465
Vierordt's Method	465
Vieussens, Annulus of	465

	PAGE
Vieussens, Valve of	465
Vigilambulism	465
Vigilium or Vigil	465
Villefranche	465
Villi	465
Vina	465
Vinadio	465
Vincent's Angina	465
Vincula Accessoria Tendinum	465
Vinegar	465
Vinum	466
Violation	466
Violet	466
Violin-Player's Cramp	466
Viperidæ	466
Virchow's Disease	466
Virginian Prune Bark	466
Virginity	466
Virility	466
Virus	466
Vis	466
Viscera	466
Visceral Pain	466
Visceroptosis	480
Viscosity	480
Viscum Album	480
Viscus	480
Vision, Field of	480
Visual	487
Visus	487
Vital Statistics	487
Vitalia	497
Vitelline	497
Vitellus	497
Viterbo	497
Vitiligo	497
Vitiligoidea	498
Vitium	498
Vitreous Humour, Diseases of	498
Vittel	502
Vitus's Dance	502
Viviparous	502
Vivisection	502
Vocal	502
Vogt's Point	502
Voice	502
Volar	503
Volatile Oils	503
Volition	503
Volkmann's Deformity	503
Volkmann's Seat	503
Volsella	503
Volt	503
Voltolini's Disease	503
Volvulus	503
Vomer	503
Vomica	503
Vomiting	503
Von Baer's Law	504
Von Baer's Triangle	504
Von Fleischl's Hæmometer	504
Von Græfe's Symptom	504
Von Hacker's Operation	504
Von Jaksh's Anæmia	504
Von Noorden's Method	504
Vowels	504
Vox	504
Vulsellum	504
Vulva, Diseases of the	504
Injuries	505
Inflammation	505
Vascular Disturbances	506
Cutaneous Diseases	507
Cysts and Tumours	508
Morbid Conditions of Clitoris and Hymen	509
Vulvar or Vulval	510
Vulvitis	510
Vulvo-	510
Wachendorf's Membrane	510
Wachsmuth's Mixture	510
Wade's Balsam or Drops	510
Wagner's Corpuscles	510
Wagner's Operation	510
Wahlberga	510
Waistcoat, Strait	510
Walcher's Position	510
Waldeyer's Epithelium	510
Walking	510
Wallerian Degeneration	510
Walther's Ducts	510
Walther's Ganglion	510
Wandering	510
Wanklyn's Process	510
Warburg's Tincture	510
Wardrop's Disease	510
Ward's Paste	510
Warmbrunn	510
Warming	510
Wartpox	510
Warts or Verrucæ	510
Warty Lupus	512
Wash	512
Washerwoman's Itch	512
Washing Out	512
Wasp	512
Wasserman, Reaction of	512
Wasting	512
Watch-Test	512
Water	512
Water-Gas	523
Water-hammer Pulse	523
Waterhouse-Forbes Steriliser	523
Water Itch	524
Waters	524
Water, Toast	524
Watery Vapour	524
Watt's Asphyxiator	524
Wave	524
Wax	524
Waxy Degeneration	524
Weaning	524
Weather	524
Webbed Fingers and Toes	524
Weber's Glands	524
Weber's Organ	524
Weber's Syndrome	524
Weber's Test	524
Webs of the Larynx	524
Webster's Process	524
"Weed"	524
Weeks' Bacillus	524
Weeping	524
Wegner's Disease	524
Weichselbaum's Bacillus	524
Weidel's Reaction	524
Weigert's Stain	524
Weighing of Infants	524
Weights	524
Weilbach	524
Weil's Disease	524
Weil's Syndrome	524
Weir Mitchell's Disease	524
Weir Mitchell's Method	525
Weismann's Theory	525
Weissenburg	525
Weiss's Reflex	525
Weiss's Sign	525
Weitbrecht's Cartilage	525
Weitbrecht's Cord	525
Welcker's Angle	525
Wells' Facies or Sign	525
Welsbach Burner	525
Wemyss Bay	525
Werlhof's Disease	525
Wernicke's Centre	525
Wernicke's Disease	525
Wernicke's Fissure	525
Wernicke's Reaction	525
West Indies	525
Weston-Super-Mare	525
Westphal's Nucleus	525
Westphal's Paradoxical Contraction	525
Westphal's Sign	525
West Virginia	525
Wet-Nursing	525
Wet Pack	525
Wetzel's Test	525
Weyl's Test	525
Weymouth	525
Wharton's Duct	525
Wharton's Jelly	525
Wheals	525
Wheelhouse's Operation	525
Wheezing	525
Whey	525
Whipworm	526
Whisky	526

	PAGE
Whispering Pectoriloquy	526
White Arsenic	526
White Bread	526
White Commissure	526
White Corpuscles	526
White Disease	526
White Flux	526
Whitehead's Gag	526
Whitehead's Operation	526
White Lead	526
White Leg	526
White Precipitate	526
White's Disease	526
White Swelling	526
Whites	526
Whitlow	526
Whooping-Cough	526
Whytt's Disease	532
Wichmann's Asthma	532
Wickersheimer's Fluid	532
Widal's Reaction	532
Wiesbaden	532
Wight, Isle of	532
Wildbad	532
Wilde's Incision	532
Wildermuth's Ear	532
Wildungen	532
Wilkinson's Disease	532
Wilk's Disease	532
Will	532
Willan's Lepra	532
Willan's Lupus	532
Williams' Tracheal Tone	532
Williamson's Test	532
Willich's Formula	532
Willis, Circle of	532
Willis' Disease	532
Willis' Glands	532
Willis' Nerve	532
Wilson's Disease	532
Winckel's Disease	532
Winds	532
Wineglass	532
Wines	533
"Winged" Scapula	533
Winking	533
Winslow, Foramen of	533
Winslow's Ligament	533
Winslow's Pouch	533
Wintergreen	533
Wintrich's Sign	533
Wiring, Operation of	533
Wirsung, Duct or Canal of	533
Witch's Milk	533
Woillez Disease	533
Wolffian Body	533
Wolffian Cyst	533
Wolfler's Operation	533
Wooden Tongue	533
Woodhall Spa	533
Wood Ticks	533
Wood Wool	533
Woolsorter's Disease	533
Woorara	533
Word-Blindness	533
Word-Deafness	533
Work	533
Wormian Bones	533
Worms	533
Worthing	533
Wounds	533
Wourali	542
Wright's Coagulometer	542
Wright's Stain	542
Wrisberg, Cartilages of	542
Wrisberg, Ganglion of	542
Wrisberg, Nerve of	542
Wrist-drop	542
Wrist-jerk	542
Wrist-Joint, Injuries of	542
Wrist-Joint, Diseases of	553
Writer's Cramp, Palsy or Spasm	558
Writing	558
Wry-Neck or Torticollis	558
Wunderlich's Law	562
Wutzer's Operation	562
Wutz's Valve	562
Xanthelasma	562
Xanthin	562
Xanthinuria	562
Xantho-	562
Xantho-creatinine	562
Xantho-cyanopia	562
Xanthodermia	562
Xanthoma	562
Xanthoproteic	563
Xanthopsia	563
Xanthosis	563
"Xaxa"	563
Xenia	564
Xeno-	564
Xenomenia	564
Xenon	564
Xero-	564
Xeroderma or Xerodermia	564
Xeroderma Pigmentosum	564
Xeroform	564
Xeroma	564
Xerophthalmia	564
Xerosis	564
Xerostomia or Xerostoma	564
Xiphi- or Xipho-	564
Xiphisternum	564
Xiphodymus or Xiphodidymus	564
Xiphopagus	565
X-Rays, Finsen Light, and High Frequency Currents	565
Xylene	584
Xylidne	584
Xylo-	584
Xyloidin	584
Xylol	584
Xylonite	584
Xylose	584
Xylotherapy	584
Y-shaped	584
Yawning	584
Yaws	585
Yeast	588
Yelk	588
Yellow Atrophy of Liver	588
Yellow Body	588
Yellow Chill	588
Yellow Colour	588
Yellow Fever	588
Yellow Jack	599
Yellow Lotion	599
"Yellows"	599
Yellow Spot	599
Yellow Vision	600
Yellow Wash	600
Yemen Ulcer	600
Yerba Santa	600
Yerbine	600
Yersin's Serum	600
Yew	600
Ymshi	600
Yohimbin	600
Yolk	600
Young-Helmholtz Theory	600
Young's Rule	600
Ypsiliform	600
Yumoto	600
Yverdon	600
Zaglas' Ligament	600
Zahnleiste	600
Zanaloin	600
Zang's Space	600
Zappert's Counting Chamber	600
Zea Mays	600
Zeismus	600
Zeiss' Glands	600
Zeller's Operation	600
Zelotypia	600
Zenker's Degeneration	600
Zenker's Paralysis	600
Zermatt	600
Zestocausis	600
Ziehl's Solution	601
Ziemssen's Points	601
Ziemssen's Test	601
Zimmermann's Corpuscles	601
Zinc	601
Zincalism	601
Zinc Perhydrol	601
Zincum	601
Zingiber	601
Zinn, Artery of	601

	PAGE
Zinn, Ligament or Ring of	601
Zinn, Zonule of	601
Zittman's Decoction	601
Zoamylin	601
Zoanthropia	601
Zollikofer's Method	601
Zomotherapy or Zoomotherapy	601
Zona	602
Zonary	602
Zone	602
Zonula	602
Zonular	602
Zonulitis	602
Zoö-	602
Zoster	602
Zosteriform	602
Zuckerkandl's Convolution	602
Zuckerkandl's Vein	602
Zygapophysis	602
Zygo-	602
Zygoma	602
Zymase	602
Zymine	602
Zymo-	602
Zymogen	602
Zymogenic	602
Zymophore	602
Zymotic	602

END OF VOL. X.

PRINTED BY NEILL AND CO., LTD., EDINBURGH.